



# GUIDELINES

*For*

# GENERAL PRACTITIONERS

2024

## Press record

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Cover Designer (Tun Zaw & Win Zaw)

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# FOREWORD

It is a great honor for me to write a foreword to [Guidelines for General Practitioners](#) by General Practitioners' society, Myanmar Medical Association (Central).

General practitioners are the primary health providers in the community looking after the majority of the people of our country. They are being trusted and depend upon by every families in the surrounding area where they practice. The first and foremost care by the General Practitioners are the most important for all the people.

Guidelines based on a critical appraisal of scientific evidence (evidence-based guidelines) clarify which interventions are of proved benefit and document the quality of the supporting data. They alert clinicians to interventions unsupported by good science, reinforce the importance and methods of critical appraisal, and call attention to ineffective, dangerous, and wasteful practices.

Clinical guidelines can improve the quality of clinical decisions. They offer explicit recommendations for clinicians who are uncertain about how to proceed, overturn the beliefs of doctors accustomed to outdated practices, improve the consistency of care, and provide authoritative recommendations that reassure practitioners about the appropriateness of their treatment policies.

The Myanmar Medical Association together with the GP society has been helping out with the CME and CPD program for the Member doctors both inhouse sessions and online courses. This guideline is one of the essential parts of this CPD for the GPs.

I would like to congratulate the GP society for their effort for producing this guideline and also, I would like to encourage them to review and updated regularly.



Professor Aye Aung  
President

Myanmar Medical Association

April, 2024



# PREFACE

We are writing this letter to express our sincerest gratitude and appreciation for the successful completion of the **second edition** of the **General Practitioners' Guidelines**. This accomplishment is the result of an exceptional collaborative effort, and we would like to extend our thanks to all those involved.

The General Practitioners' Guidelines has been an invaluable resource since its inception with the launch of the first edition in November 2017. As per the initial plan, the guidelines were intended to be updated every three years to ensure the most up-to-date information reaches Myanmar General Practitioners, enhancing their knowledge in primary healthcare and family health.

However, the unforeseen outbreak of the Covid-19 pandemic disrupted our plans and posed numerous challenges for the team. In-person meetings became impossible due to safety concerns, making it necessary for us to find alternative means of communication and collaboration. Despite the adversity faced, the team members demonstrated remarkable resilience and adaptability by utilizing online platforms and technology to continue the update process.

We would like to extend our deepest gratitude to the dedicated team members who persevered and worked tirelessly during these trying times. Their commitment, professionalism, and unwavering dedication to the project enabled us to overcome the obstacles posed by the pandemic and successfully complete the second edition of the guideline.

Furthermore, we would like to express our sincere appreciation to the specialist societies that actively contributed to the development of the guidelines. Their expertise and invaluable insights have ensured that the content remains current, accurate, and relevant, enabling our General Practitioners to provide the highest quality of care to their patients.

We would also like to extend our heartfelt thanks to the esteemed President of the Myanmar Medical Association, for their continuous support and guidance throughout this endeavor. Their leadership and unwavering commitment to advancing medical knowledge in Myanmar have been instrumental in the success of this Guidelines.

Moreover, the decision to distribute the guideline as electronic copies reflects our commitment to ensuring easy access for all Myanmar General Practitioners. By making it available in this format, we aim to facilitate the dissemination of updated knowledge, thus empowering our healthcare professionals to deliver the best possible care to the community.

In conclusion, we would like to express our deepest gratitude to all those who contributed to the development and distribution of the General Practitioners' Guidelines Second Edition. The unwavering supports and collective efforts have made a significant impact on enhancing primary healthcare and family health care in Myanmar.

Once again, thank you for your outstanding dedication, resilience, and invaluable contributions. We look forward to our continued collaboration in advancing medical knowledge and improving healthcare outcomes for all.

**Dr Khine Soe Win and Dr Win Zaw**  
**General Practitioners' Society (Central)**  
**Myanmar Medical Association**

April, 2024



# EDITORIAL

It is my privilege to inform you that our updated and revised edition of “**Guidelines for General Practitioners**” will be published very soon and it is my great pleasure to be the editor-in-chief of this guideline book. There are various reasons for revising and updating the previous edition.

This is the fact that some important topics, for example, malaria and family violence are missing in the first edition and some clinical practice guidelines like Diabetes Management have been changed during the interim period. Of course, this opportunity arises due to the emergence of COVID-19 in the world. As all you know, Medicine is an ever-changing science; we need to consider updating our guidelines at least five- yearly. Hence the time is up now!

Education is achieved by assimilating information from many resources and readers of this book can enhance their learning experience in terms of reflecting in their daily Family/General Practice. We all take immense pride in contributing good educational resource dedicated to Myanmar General Practitioners. The editors and authors anticipate that the readers will both enjoy and profit from their work in preparing this volume.

Happy studying and learning,

**Dr Win Lwin Thein**  
**Editor-in chief**  
**Vice President (GP Society)**  
**April, 2024**





# ACKNOWLEDGEMENT

We would like to thank all our talented and hard-working colleagues who have contributed to the ongoing development of the **Guidelines for General Practitioners**.

Especially, we would like to highlight the significance of the second edition which appears when the family medicine development process in Myanmar is being idle. Many factors are impeding the developing process lately, which has been accelerated previously by the commitment of the MOHS, the medical universities, and the General Practitioners' Society before the COVID-19 pandemic started.

No one can deny that the Myanmar health care system is lacking a strong and effective primary care task force. The best solution to mend this defect is retraining the thousands of general practitioners who are working individually across the country. Here comes the role of family medicine to train these GPs and primary care doctors to be able to use its principles effectively and, in turn, strengthen primary care.

Many GPs are using some family medicine principles consciously or unconsciously in varying degree of competency. Person-centered care, continuity of care, and family-oriented care became the culture of most practices for a long time. But only a few GPs can enjoy the most effective coordinated care and seamless continuity of care with secondary and tertiary care providers. The reasons behind this would be the absence of standardization in general practitioners' service quality and unawareness of the value of family medicine practitioners by other specialties and the public.

To resolve this ambiguity, primary care doctors should be involved in the retraining programs and thereafter CME/CPD and other life-long-learning programs which prescribe family medicine curricula.

We also acknowledge the effort of the contributors to make this new edition more family medicine-oriented, in addition to the Family Medicine chapter at the beginning of the book. We genuinely believe that the new edition will be a better reference for the GP/FP who wants to practice quality primary care and for future family medicine programs in Myanmar.

Finally, we would like to thank all academic writers who contributed to the General Practice Guidelines-first edition. Without their kind support, this second edition could never have happened.

Regards,

**Dr. Tin Aye and Dr. Kyaw Thu**

**General Practitioners' Society (Central), MMA**

**April, 2024**



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## SYMBOLS AND ABBREVIATIONS

<b>AAA</b> abdominal aortic aneurysm	<b>COAD</b> chronic obstructive airways disease
<b>ABC</b> airway, breathing, circulation	<b>COC</b> combined oral contraceptive
<b>ABCD</b> airway, breathing, circulation, dextrose	<b>COCP</b> combined oral contraceptive pill
<b>ABO</b> A, B and O blood groups	<b>COPD</b> chronic obstructive pulmonary disease
<b>ACE</b> angiotensin-converting enzyme	<b>COX</b> cyclooxygenase
<b>ACEI</b> angiotensin-converting enzyme inhibitor	<b>CPA</b> cardiopulmonary arrest
<b>ACTH</b> adrenocorticotrophic hormone	<b>CPAP</b> continuous positive airways pressure
<b>ADHD</b> attention deficit hyperactivity disorder	<b>CPK</b> creatine phosphokinase
<b>ADT</b> adult diphtheria vaccine	<b>CPR</b> cardiopulmonary resuscitation
<b>AFP</b> alpha-fetoprotein	<b>CR</b> controlled release
<b>AI</b> aortic incompetence	<b>CREST</b> calcinosis cutis; Raynaud's phenomenon; oesophageal involvement; sclerodactyly; telangiectasia
<b>AIDS</b> acquired immunodeficiency syndrome	<b>CRF</b> chronic renal failure
<b>AHRA</b> angiotensin II (2) reuptake antagonist	<b>CR(K)F</b> chronic renal (kidney) failure
<b>AKF</b> acute kidney failure	<b>CRP</b> C-reactive protein
<b>ALE</b> average life expectancy	<b>CSF</b> cerebrospinal fluid
<b>ALL</b> acute lymphocytic leukaemia	<b>CT</b> computerised tomography
<b>ALP</b> alkaline phosphatase	<b>CTS</b> carpal tunnel syndrome
<b>ALT</b> alanine aminotransferase	<b>CVA</b> cerebrovascular accident
<b>AMI</b> acute myocardial infarction	<b>CVS</b> cardiovascular system
<b>AML</b> acute myeloid leukaemia	<b>CXR</b> chest X-ray
<b>ANA</b> antinuclear antibody	<b>DBP</b> diastolic blood pressure
<b>ANF</b> antinuclear factor	<b>DC</b> direct current
<b>AP</b> anterior-posterior	<b>DHA</b> docosahexaenoic acid
<b>APH</b> ante-partum haemorrhage	<b>DI</b> diabetes insipidus
<b>ASD</b> atrial septal defect	<b>DIC</b> disseminated intravascular coagulation
<b>ASIS</b> anterior superior iliac spine	<b>dL</b> decilitre
<b>ASOT</b> antistreptolysin O titre	<b>DMARDs</b> disease modifying antirheumatic drugs
<b>AST</b> aspartate aminotransferase	<b>DNA</b> deoxyribose-nucleic acid
<b>AV</b> atrioventricular	<b>DRABC</b> defibrillation, resuscitation, airway, breathing, circulation
<b>AZT</b> azidothymidine	<b>drug dosage</b> bd—twice daily, tid/tds -three times daily, qid/qds -four times daily
<b>BCC</b> basal cell carcinoma	<b>ds</b> double strand
<b>BCG</b> bacille Calmette-Guérin	<b>DS</b> double strength
<b>BMD</b> bone mass density	<b>DSM</b> diagnostic and statistical manual (of mental disorders)
<b>BMI</b> body mass index	<b>DU</b> duodenal ulcer
<b>BP</b> blood pressure	<b>DUB</b> dysfunctional uterine bleeding
<b>BPH</b> benign prostatic hyperplasia	<b>DVT</b> deep venous thrombosis
<b>Ca</b> carcinoma	<b>EBM</b> Epstein-Barr mononucleosis (glandular fever)
<b>CABG</b> coronary artery bypass grafting	<b>EBV</b> Epstein-Barr virus
<b>CAD</b> coronary artery disease	<b>ECG</b> electrocardiogram
<b>CAP</b> community acquired pneumonia	<b>ECT</b> electroconvulsive therapy
<b>CBT</b> cognitive behaviour therapy	<b>EDD</b> expected due date
<b>CCF</b> congestive cardiac failure	<b>EEG</b> electroencephalogram
<b>CCU</b> coronary care unit	<b>ELISA</b> enzyme linked immunosorbent assay
<b>CD4</b> T helper cell	<b>ESRF</b> end-stage renal failure
<b>CD8</b> T suppressor cell	<b>ESR(K)F</b> end stage renal (kidney) failure
<b>CDT</b> combined diphtheria/tetanus vaccine	<b>ERCP</b> endoscopic retrograde cholangiopancreatography
<b>CEA</b> carcinoembryonic antigen	<b>esp.</b> especially
<b>CFS</b> chronic fatigue syndrome	<b>ESR</b> erythrocyte sedimentation rate
<b>CHD</b> coronary heart disease	<b>FB</b> foreign body
<b>CHF</b> chronic heart failure	<b>FBE</b> full blood count
<b>CIN</b> cervical intraepithelial neoplasia	
<b>CK</b> creatinine kinase	
<b>CKD</b> chronic kidney disease	
<b>CKF</b> chronic kidney failure	
<b>CML</b> chronic myeloid leukaemia	
<b>CMV</b> cytomegalovirus	
<b>CNS</b> central nervous system	

**FEV1** forced expiratory volume in 1 second  
**fL** femtolitre = (1e-15) litre  
**FSH** follicle stimulating hormone  
**FUO** fever of undetermined origin  
**FVC** forced vital capacity  
**g** gram  
**GA** general anaesthetic  
**GABHS** group A beta-haemolytic streptococcus  
**GBS** Guillain-Barré syndrome  
**GFR** glomerular filtration rate  
**GI** glycaemic index  
**GIT** gastrointestinal tract  
**GLP** glucagon-like peptide  
**GnRH** gonadotrophin-releasing hormone  
**GO** gastro-oesophageal  
**GORD** gastro-oesophageal refl ux  
**GP** general practitioner  
**G-6-PD** glucose-6-phosphate  
**GU** gastric ulcer  
**HAV** hepatitis A virus  
**anti-HAV** hepatitis A antibody  
**Hb** haemoglobin  
**HbA** haemoglobin A  
**anti-HBc** hepatitis B core antibody  
**HBeAg** hepatitis B e antigen  
**anti-HBs** hepatitis B surface antibody  
  
**HBsAg** hepatitis B surface antigen  
**HBV** hepatitis B virus  
**HCG** human chorionic gonadotropin  
**HCV** hepatitis C virus  
**anti-HCV** hepatitis C virus antibody  
**HDL** high-density lipoprotein  
**HEV** hepatitis E virus  
**HFM** hand, foot and mouth  
**HFV** hepatitis F virus  
**HGV** hepatitis G virus  
**HIV** human immunodeficiency virus  
**HNPCC** hereditary nonpolyposis colorectal cancer  
**HPV** human papilloma virus  
**HRT** hormone replacement therapy  
**HSV** herpes simplex viral infection  
**IBS** irritable bowel syndrome  
**ICE** ice, compression, elevation  
**ICS** inhaled corticosteroid  
**ICS** intercondylar separation  
**ICT** immunochromatographic test  
**IDDM** insulin dependent diabetes mellitus  
**IDU** injecting drug user  
**IgE** immunoglobulin E  
**IgG** immunoglobulin G  
**IgM** immunoglobulin M  
**IHD** ischaemic heart disease  
**IM, IMI** intramuscular injection  
**inc.** including  
**IPPV** intermittent positive pressure variation  
**IR** internal rotation  
**ITP** idiopathic (or immune) thrombocytopenia  
     purpura  
**IUCD** intrauterine contraceptive device  
**IUGR** intrauterine growth retardation  
  
**IV** intravenous  
**IVI** intravenous injection  
**IVP** intravenous pyelogram  
**IVU** intravenous urogram  
**JCA** juvenile chronic arthritis  
**JVP** jugular venous pulse  
**KA** keratoacanthoma  
**kg** kilogram  
**KOH** potassium hydroxide  
**LA** local anaesthetic  
**LABA** long acting beta agonist  
**LBBB** left branch bundle block  
**LBO** large bowel obstruction  
**LBP** low back pain  
**LDH/LH** lactic dehydrogenase  
**LDL** low-density lipoprotein  
**LFTs** liver function tests  
**LH** luteinising hormone  
**LHRH** luteinising hormone releasing hormone  
**LIF** left iliac fossa  
**LMN** lower motor neurone  
**LNG** levonorgestrel  
**LRTI** lower respiratory tract infection  
**LSD** lysergic acid  
**LUQ** left upper quadrant  
**LUTS** lower urinary tract symptoms  
**LV** left ventricular  
**LVH** left ventricular hypertrophy  
**mane** in morning  
**MAOI** monoamine oxidase inhibitor  
**mcg** microgram (also µg)  
**MCV** mean corpuscular volume  
**MDI** metered dose inhaler  
**MDR** multi-drug resistant TB  
**MI** myocardial infarction  
**MRCP** magnetic resonance cholangiography  
**MRI** magnetic resonance imaging  
**MS** multiple sclerosis  
**MSM** men who have sex with men  
**MSU** midstream urine  
**N** normal  
**NAD** no abnormality detected  
**NGU** non-gonococcal urethritis  
**NHL** non-Hodgkin's lymphoma  
**NIDDM** non-insulin dependent diabetes mellitus  
**nocte** at night  
**NSAIDs** non-steroidal anti-inflammatory drugs  
**NSU** non-specific urethritis  
**(o)** taken orally  
**OA** osteoarthritis  
**OCP** oral contraceptive pill  
**OGTT** oral glucose tolerance test  
**OSA** obstructive sleep apnoea  
**OTC** over the counter  
**PA** posterior–anterior  
**PAN** polyarteritis nodosa  
**Pap** Papanicolaou  
**pc** after meals  
**PCA** percutaneous continuous analgesia  
**PCB** post coital bleeding

**PCL** posterior cruciate ligament  
**PCOS** polycystic ovarian syndrome  
**PCP** pneumocystis carinii pneumonia  
**PCR** polymerase chain reaction  
**PCV** packed cell volume  
**PDA** patent ductus arteriosus  
**PEF** peak expiratory flow  
**PEFR** peak expiratory flow rate  
**PET** pre-eclamptic toxemia  
**PFT** pulmonary function test  
**PH** past history  
**PID** pelvic inflammatory disease  
**PLISSIT** permission: limited information: specific suggestion: intensive therapy  
**PMS** premenstrual syndrome  
**PMT** premenstrual tension  
**POP** plaster of Paris  
**POP** progestogen-only pill  
**PPI** proton-pump inhibitor  
**PPROM** preterm premature rupture of membranes  
**PR** per rectum  
**prn** as and when needed  
**PROM** premature rupture of membranes  
**PSA** prostate specific antigen  
**PSIS** posterior superior iliac spine  
**PSVT** paroxysmal supraventricular tachycardia  
**PT** prothrombin time  
**PTC** percutaneous transhepatic cholangiography  
**PU** peptic ulcer  
**PUO** pyrexia of undetermined origin  
**pv** per vagina  
**qds, qid** four times daily  
**RA** rheumatoid arthritis  
**RBBB** right branch bundle block  
**RBC** red blood cell  
**RCT** randomised controlled trial  
**RF** rheumatic fever  
**Rh** rhesus  
**RIB** rest in bed  
**RICE** rest, ice, compression, elevation  
**RIF** right iliac fossa  
**RPR** rapid plasma reagin  
**RR** relative risk  
**RSV** respiratory syncytial virus  
**RT** reverse transcriptase  
**rtPA** recombinant tissue plasminogen activator  
**SAH** subarachnoid haemorrhage  
**SARS** severe acute respiratory distress syndrome  
**SBE** subacute bacterial endocarditis  
**SBO** small bowel obstruction  
**SBP** systolic blood pressure  
**SC/SCI** subcutaneous/subcutaneous injection  
**SCC** squamous cell carcinoma  
**SCG** sodium cromoglycate  
**SIADH** syndrome of secretion of inappropriate antidiuretic hormone  
**SIDS** sudden infant death syndrome  
**SIJ** sacroiliac joint  
**SL** sublingual  
**SLE** systemic lupus erythematosus  
**SLR** straight leg raising  
**SND** sensorineural deafness  
**SNHL** sensorineural hearing loss  
**SNRI** serotonin noradrenaline reuptake inhibitor  
**SOB** shortness of breath  
**sp** species  
**SR** sustained release  
**SSRI** selective serotonin reuptake inhibitor  
**SSS** sick sinus syndrome  
**stat** at once  
**STI** sexually transmitted infection  
**SVC** superior vena cava  
**SVT** supraventricular tachycardia  
**T3** tri-iodothyronine  
**T4** thyroxine  
**TB** tuberculosis  
**tds, tid** three times daily  
**TENS** transcutaneous electrical nerve stimulation  
**TFTs** thyroid function tests  
**TG** triglyceride  
**TIA** transient ischaemic attack  
**TIBC** total iron binding capacity  
**TM** tympanic membrane  
**TMJ** temporomandibular joint  
**TNF** tissue necrosis factor  
**TOF** tracheo-oesophageal fistula  
**TORCH** toxoplasmosis, rubella, cytomegalovirus, herpes virus  
**TPHA** Treponema pallidum haemagglutination test  
**TSE** testicular self-examination  
**TSH** thyroid-stimulating hormone  
**TT** thrombin time  
**TV** tidal volume  
**U** units  
**UC** ulcerative colitis  
**U & E** urea and electrolytes  
**µg** microgram  
**UMN** upper motor neurone  
**URTI** upper respiratory tract infection  
**US** ultrasound  
**UTI** urinary tract infection  
**U** ultraviolet  
**VC** vital capacity  
**VDRL** Venereal Disease Reference Laboratory  
**VF** ventricular fibrillation  
**VMA** vanillyl mandelic acid  
**VSD** ventricular septal defect  
**VT** ventricular tachycardia  
**VUR** vesico-ureteric reflux  
**VWD** von Willebrand's disease  
**WBC** white blood cells  
**WCC** white cell count  
**WHO** World Health Organization  
**WPW** Wolff-Parkinson-White  
**XL** sex linked





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## Chapter (9)

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### **Musculoskeletal Problems**

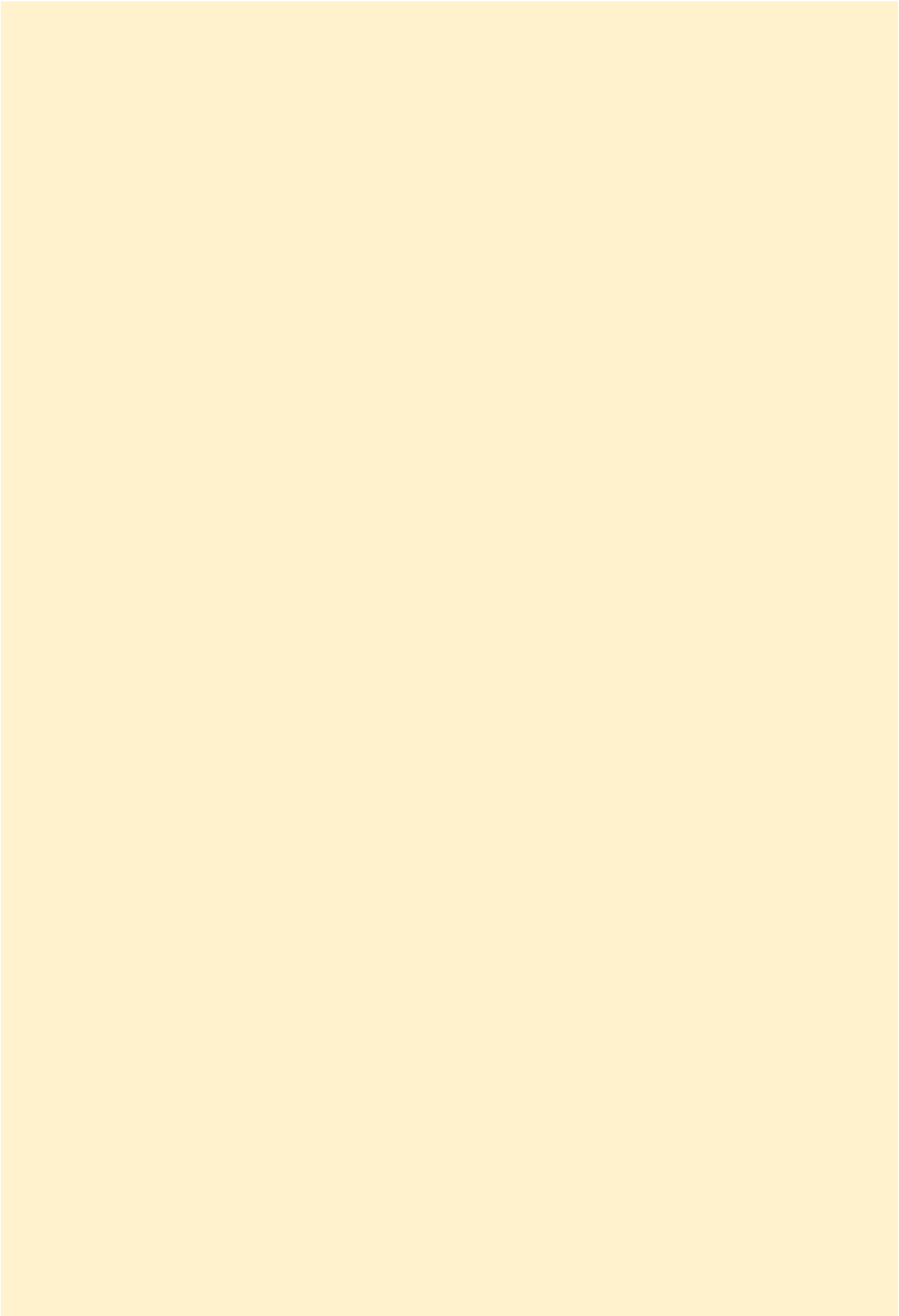
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## CHAPTER (9)

# MUSCULOSKELETAL PROBLEMS

1. Low Back Pain
2. Neck Pain
3. Shoulder Problems
4. Knee Problems
5. Elbow Problems
6. Ankle Problems
7. Foot Pain
8. Fibromyalgia Syndrome
9. Gout
10. Osteoarthritis
11. Osteoporosis
12. Rheumatoid Arthritis
13. Systemic Lupus Erythematosus



# LOW BACK PAIN

## Definition

Acute low back pain: New episode of low back pain of < 6weeks duration.

Common-lifetime prevalence 58%

Chronic low back pain: Back pain lasting >3months

## Causes of back pain

Age (Yr)	Causes		
15-30	Postural Medical Prolapse disc	Trauma Fracture Ankylosing spondylosis	Spondylolisthesis Pregnancy
30-50	Postural Prolapse disc	Discitis Spondyloarthropathies	Degenerative joint disease
>50	Postural Malignancy (Lung, Breast, Prostate, Thyroid, Kidney)	Myelanoma	Paget's disease
Other causes	Referred pain Spinal stenosis	Cauda equina tumours	Spinal infection

## History

- Circumstances of pain-history of injury; duration
- Nature/severity of pain-pain/stiffness mainly at rest/at night, easing with movement suggests inflammation, e.g. discitis, spondylarthropathy
- Associated symptoms- numbness, weakness, bowel/bladder symptoms
- PMH-past illnesses (e.g. cancer), previous back problems
- Exclude pain not coming from the back (e.g. GI or GU pain)

## Examination

- Deformity, e.g. kyphosis (typical of ankylosing spondylitis), loss of lumbar lordosis (common in acute mechanical back pain), scoliosis
- Palpate for tenderness, step deformity, and muscle spasm
- Assess flexion, extension, lateral flexion, and rotation whilst standing
- Ask to lie down-this gives a good indication of severity of symptoms
- In lower limbs look for muscle wasting and check power, sensory loss, and reflexes (knee jerk and ankle jerk) see table 1
- Assess Straight Leg Raise (SLR) - sciatica is present if SLR on one side elicits back/buttock pain (usually ipsilateral but can be either side) compared to SLR on the other side

## Straight leg raising (SLR) test

- This test is a passive test by the practitioner. The patient lies supine with both knees extended and the ankle dorsiflexed. The affected leg is raised slowly, keeping the knee extended. If sciatica with dural irritation is present, 20° to 60° of elevation causes reproduction of pain.

## 'Red flags'

- <20 or >55 years
- Non-mechanical pain
- Pain that worsens when supine
- Night-time pain
- Thoracic pain
- Past history of cancer
- HIV
- Immune suppression
- IV drug use
- Taking steroids
- Unwell
- Weight decrease
- Structural deformity
- Widespread neurology (table 1)

**Table 1: Neurology with lumbosacral nerve root entrapment**

Root	Sensory changes	Motor weakness	Reflex changes
L2	Front of thigh	Hip flexion/adduction	None
L3	Inner thigh	Knee extension	Knee
L4	Knee extension	Foot dorsiflexion	Knee
L5	Outer shin Dorsum of foot	Knee flexion Foot inversion Big toe dorsiflexion	None
SJ	Lateral side of foot/sole	Knee flexion Foot plantarflexion	Ankle

## Management of acute pain in the community

- Triage according to history and examination-see Figure 1
- FOR PATIENTS WHO DO NOT REQUIRE IMMEDIATE REFERRAL
- Prescribe analgesia, e.g. paracetamol ± NSAIDs ±amitriptyline (10-25 mg nocte) and use the Keele STarT back screening tool.

## Keele STarT Back Pain Scoring Tool

- Ask patients to consider the following statements and state whether they agree or disagree with them. Thinking about the past 2weeks:
- My back pain has spread down my leg(s) at some time in the last 2 week
- I have had pain in the shoulder or neck at some time in the last 2 weeks
- I have only walked short distances because of my back pain
- In the last 2 weeks, I have dressed more slowly than usual because of back pain
- It's not really safe for a person with a condition like mine to be physically active
- Worrying thoughts have been going through my mind a lot of the time
- I feel that my back pain is terrible and it's never going to get any better
- In general, I have not enjoyed all the things I used to enjoy.
- (If the patient agrees with a statement, score 1; if disagrees, score 0)
- Overall, how bothersome has your back pain been in the last 2 weeks?
- (Not at all, slightly, or moderately- score 0, Very much or extremely- score 1)

- If total score:  $\leq 3$ , explain likely natural history of the pain and advise to avoid bed rest and maintain normal activities as far as possible (decrease chance of chronic pain). Suggest self-help exercises
- If total score is  $\geq 4$ , check question 5-9 sub-score:
- If:  $\leq 3$  - if not resolved in 4 weeks, refer for physical therapy. Options include: back exercise classes, physiotherapy, chiropractic osteopathy, or acupuncture, if available.
- If  $\geq 4$  - if not resolved in 4 weeks, refer directly for specialist intervention, sooner if worsening or severe pain
- In all cases, challenge any 'yellow flag' factors (see Figure 1) that may inhibit recovery and delay return to normal functioning
- Do not X-ray for back pain routinely
- X rays require a high radiation dose, and clinically meaningful findings are rare. Exceptions:
  - Young ( $< 25$  years) - X-ray SI joints to exclude ankylosing spondylitis
  - Elderly-if vertebral collapse/malignancy suspected
  - History of trauma

## CAUDA EQUINA SYNDROME:

- Compression of the cauda equina below L2, e.g. by disc protrusion at L4/5 presents with:
  - Numbness of the buttocks and backs of thighs
  - Urinary/faecal incontinence
  - Lower motor neurone weakness:
    - L4 -loss of dorsiflexion of the foot (and toes-L4/5)
    - S1 - loss of ankle reflex, plantarflexion, and eversion of the foot

### **Management**

- Refer/admit as a neurological emergency.
- Rapid surgical intervention increases the chance of full motor and sphincter recovery.

## SPINAL CORD COMPRESSION

- Affects 5% of cancer patients -70% in the thoracic region
- Maintain a high level of suspicion if history of cancer and new back pain-especially if known bony metastases or tumour likely to metastasize to bone.

### **Presents with:**

- Back pain, worse on movement-often appears before neurology
- Neurological symptoms/signs-
  - can be non-specific, e.g. constipation, weak legs, urinary hesitancy.
  - Lesions above L1 (lower end of spinal cord) produce upper motor neurone signs (e.g. increased tone/reflexes) and a sensory level;
  - lesions below L1 produce lower motor neurone signs (decreased tone/reflexes) and perianal numbness (cauda equina syndrome)

### **Management**

- Prompt treatment ( $< 24$ -- $48$  h from first neurological symptoms) is needed; once paralysed,  $< 5\%$  walk again.
- Treat with oral dexamethasone 16 mg/day and refer for same-day assessment and surgery/ radiotherapy unless in final stages of disease.

## OSTEOPOROTIC VERTEBRAL COLLAPSE

- Causes pain, decreased height, and kyphosis
- Pain can take 3-6months to settle and requires strong analgesia.
- Calcitonin is useful for pain relief for 3months after vertebral fracture if other analgesics are ineffective.

## SCOLIOSIS

- Lateral curvature of the spine associated with rotation of vertebrae  $\pm$  ribs or wedging of vertebrae.
- Early treatment prevents progression and complications, e.g. cardiopulmonary disturbance.

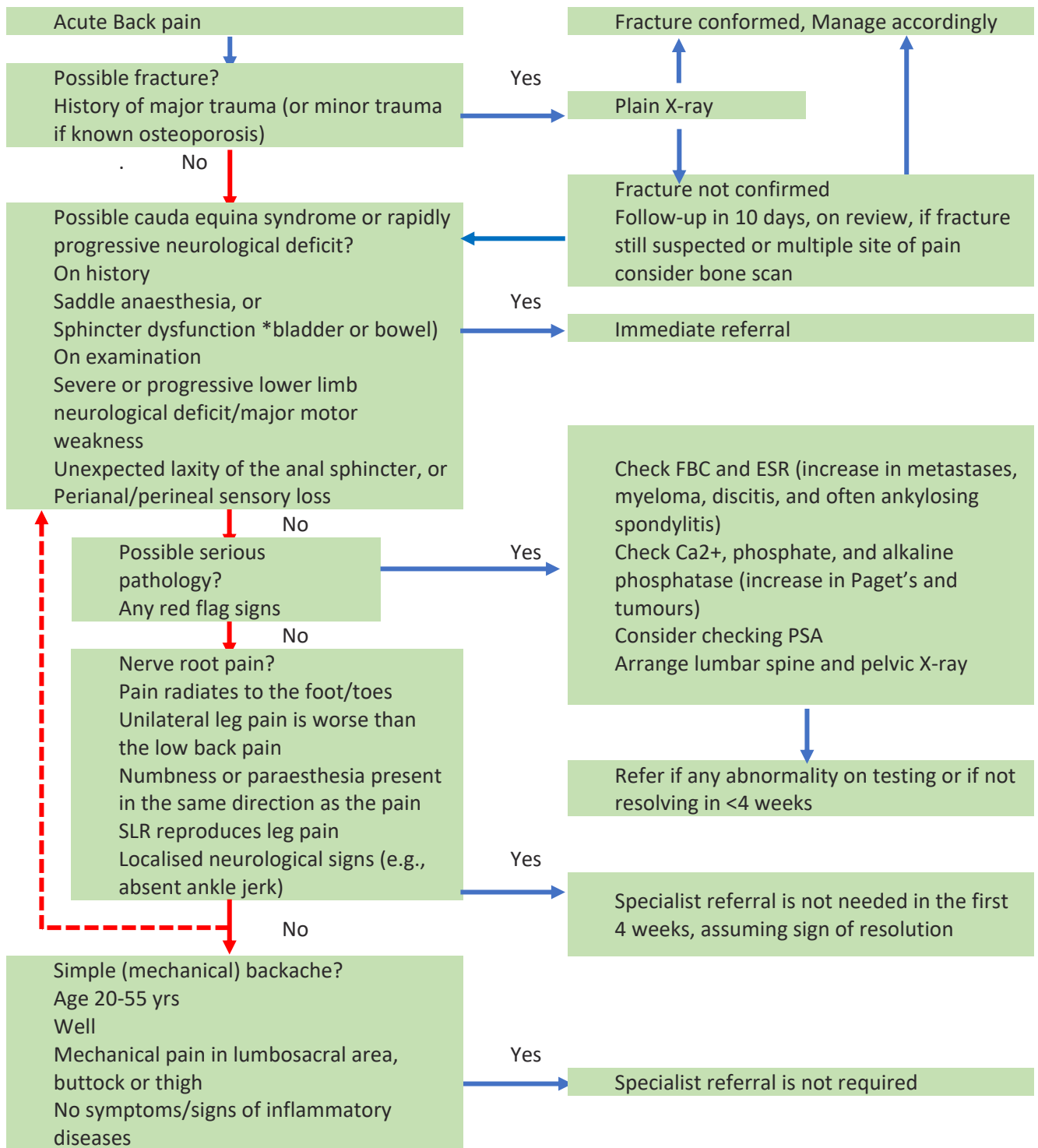
### ***Clinical features***

- Difference in shoulder height;
- Spinal curvature;
- Difference in the space between the trunk and upper limbs.
- Scoliosis which disappears on bending is postural and of no clinical significance.

### ***Management***

- In all cases where structural scoliosis is suspected, refer for an orthopaedic opinion.
- If associated with pain, especially at night, consider spinal tumour and refer urgently.





- Yellow flags – psychosocial barriers to back pain recovery
- Belief that pain and activity are harmful
- Sickness behaviours, e.g., extended rest
- Social withdrawal
- Emotional problems, e.g., low mood, depression, anxiety, or stress
- Problems and/or dissatisfaction at work
- Problems claims/compensation/time oof work
- Overprotective family, lack of support
- Inappropriate expectations of treatment

**Fig: 1 Triage of Acute Back Pain**

# LOW BACK PAIN

## **Causes**

- Mechanical low back pain,
- Malignancy - primary or secondary, infection (e. spinalTB)
- ankylosing spondylosis,
- pyelonephritis or kidney stone or
- referred pain e.g. AAA

## **Red Flags (referred to criteria within Oxford guidance)**

- Unexplained weight loss
- Previous or suspected malignancy
- Drug abuse, HIV, immunosuppression or corticosteroids use.
- Abnormal bloods (ESR >50) or fever
- Thoracic pain or night pain
- Trauma
- <20, >50 years onset (worsening or new onset)
- Disturbed gait or progressive neurological deficit
- Co-morbidity or unwell patient
- Abnormal function /controlled of bladder or bowel or saddle anesthesia
- Known osteoporosis and suspected crush fracture
- Severe morning stiffness
- Consider cauda equina syndrome if on examination there is gait disturbance, urinary retention, abnormal perianal sensation or anal tone with lower limbs weakness

## **Yellow Flags (Psychological factors)**

- A negative attitude that back pain is harmful or potential severe disabling
- Fear avoidance behaviour and reduced activity levels
- An expectation that passive, rather than active, treatment will be beneficial.
- History of depression, low mood and social withdrawal
- Social or financial problems
- <20 years – Consider HLAB27 testing for AS if morning stiffness and pain awakens the patient
- 55 years -Consider an ESR, electrophoresis (myeloma screen), urine dipstick, palpating for AAA, PSA in men and CA125 in women.
- So, should all >55 years with new onset back pain have an urgent MRI if they have no other red flags and bloods are normal. This is where clinical judgement comes in and follow local guidance.

Oxford CCG guidance	
Cauda equina and widespread neurological deficit or infection	Refer as an emergency to the spinal team
Red Flags symptoms (No mention age)	Direct referral to Urgent limited MRI
Suspected osteoporotic crush fracture only	Lateral X ray then routine MRI
No red flags and persistent pain > weeks	Review diagnosis, conservative Rx and refer to MSK assessment team
Progressive deformity(kyphosis) Intractable pain Deteriorating neurology	Review diagnosis and refer to MSK assessment team if patient will consider surgery and is a surgical candidates
Occasionally, an MRI will report that it cannot exclude malignancy in which case consider a bone scan. For simple back pain offer analgesia and exercise. Give realistic prognosis.	

•

## NECK PAIN

- Neck pain is common (lifetime incidence 50%) and contributes to 2% of GP consultations. Prevalence is highest in middle age.
- Most neck pain is acute and self-limiting (within days/weeks) but 1 in 3 have symptoms lasting >6months or recurring pain.

### **History**

- Pain-onset, site, radiation, aggravating and relieving factors, timing
- Stiffness-timing (continuous? worse in the mornings?)
- Deformity (e.g. torticollis)-onset, changes
- Neurological symptoms-numbness, paraesthesiae, weakness
- Other symptoms-weight loss, bowel/bladder dysfunction, sweats
- ! Pain is often poorly localized and neck problems commonly present with shoulder pain and/or headache (cervicogenic headache).

### **Examination**

- Look- Posture; deformity, e.g. torticollis, asymmetry of scapulae; arms and hands-wasting, fasciculation? Leg weakness?
- Feel- Tenderness? Midline tenderness may be due to supraspinous or spinous process damage following a whiplash injury. Paraspinal tenderness± spasm radiating into the trapezius ± crepitation is common with cervical spondylosis
- Move/measure -Normal ranges: flexion/extension-130 ° total range; lateral flexion--45° in each direction from a neutral position; rotation-80 ° in each direction from a neutral position
- Neurology: Weakness in the upper limbs in a segmental distribution, with loss of dermatomal sensation and altered reflexes indicates a root lesion. If cervical cord compression is suspected, examine the lower limbs looking for up-going plantars and hyperreflexia

### **Red flags**

- A Red Flag specific for neck pain is evidence of cervical myelopathy (the equivalent of the cauda equine syndrome). Neck pain may be absent but the syndrome should be suspected when any of the following features are present:
  - sensory disturbances in upper and lower limbs;
  - weakness in upper and lower limbs;
  - clumsiness and gait disturbance;
  - spasticity of lower limbs (upper limbs may be normal, spastic or flaccid);
  - increase tendon reflexes;
- Lhermitte's sign: paraesthesiae in limbs on neck flexion indicates neck instability and warrants immediate admission.
- Refer patients with one or more Red Flags urgently for a specialist opinion.

## CERVICAL SPONDYLOSIS

### **Clinical features**

- Degenerative disease of the cervical spine can cause pain, but minor changes are normal (especially >40 yr) and usually asymptomatic.
- Pain is generally intermittent and related to activity.

## **Examination**

- Reveals decreased neck mobility. Severe degeneration can cause nerve root signs.

## **Treatment**

- With analgesia ± cervical collar.
- X-ray only if conservative measures fail, troublesome pain, nerve root signs, or the patient has psoriasis (? psoriatic arthropathy).

## **NERVE ROOT IRRITATION OR ENTRAPMENT**

- Secondary to degeneration, vertebral displacement/collapse, disc prolapse, local tumour, or abscess.

## **Clinical feature:**

- Neck stiffness, pain in arms or fingers, decreased reflexes, sensory loss, and decreased power.
- The level of entrapment can usually be determined clinically.

## **Treatment**

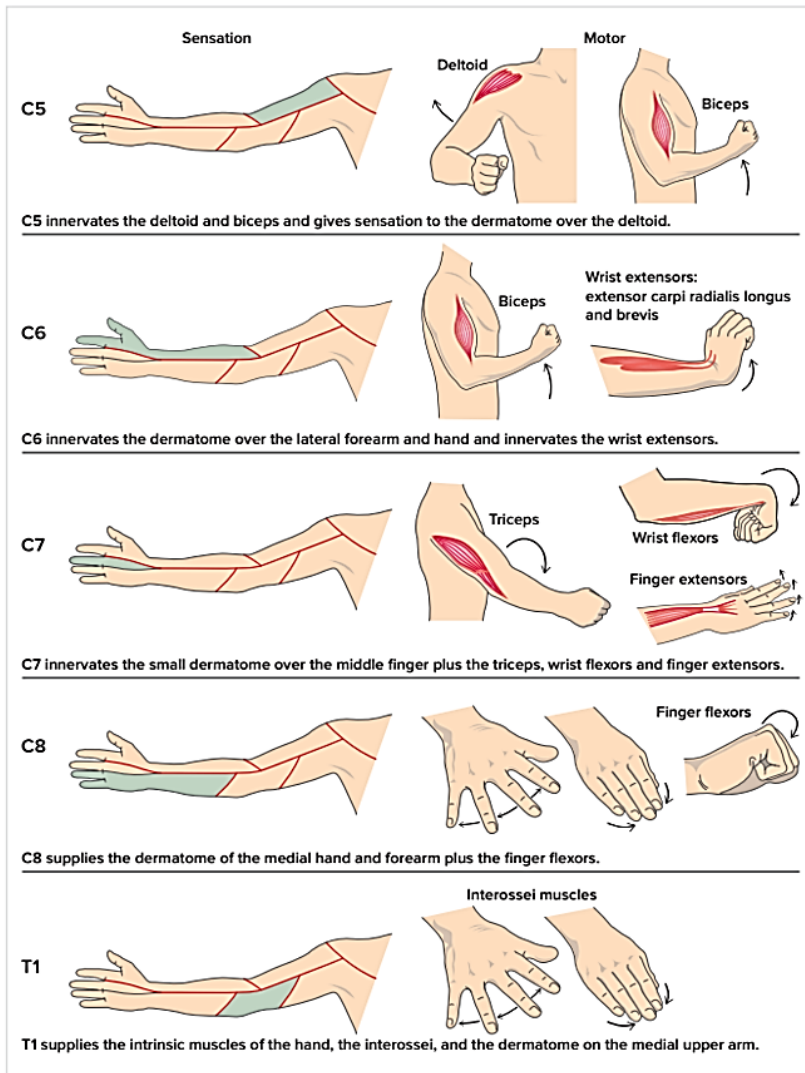
- With analgesia ± cervical collar.
- ray cervical spine -lateral or oblique views.

## **Refer**

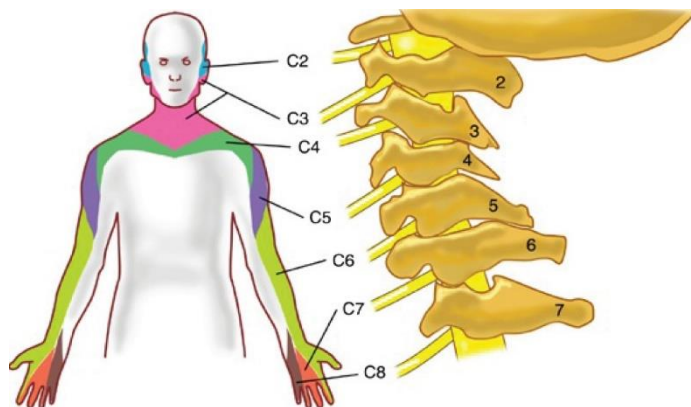
- For physiotherapy
- Refer for further investigations (e.g. MRI) if conservative management fails and there is objective evidence of a root lesion.

**Table 1. Neurology associated with cervical nerve root entrapment**

Root	Sensory changes	<b>Motor weakness</b>	Reflex changes
C5	Lateral arm	Shoulder abduction/flexion	Biceps
C6	Lateral forearm Thumb Index finger	Elbow flexion	Biceps Supinator
C7	Middle finger	Wrist extension	Triceps
C8	Medial side of lower forearm	Elbow extension Wrist flexion Finger extension Finger flexion	None
T1	Ring and little fingers Medial side of upper forearm		None



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**Fig 1. Cervical Spine contains 8 nerves, each innervating specific areas of the upper limbs**

<http://www.painnecker.com/images/cervical-nerve.jpg>

**Refer urgently if there are signs of spinal cord compression:**

- Root pain and lower motor neurone signs at the level of the lesion, *and*
- Spastic weakness, brisk reflexes, upgoing plantars, loss of coordination and sensation below the lesion

## SPASMODIC TORTICOLLIS (WRY NECK)

- Common.
- Sudden onset of painful stiff neck due to spasm of trapezius and sternocleidomastoid muscles.
- Self-limiting.
- Heat, gentle mobilization, muscle relaxants, and analgesia can speed recovery.
- A cervical collar may help in the short term but can prolong symptoms. Often caused by poor posture, e.g. computer-seating position; carrying heavy, uneven loads.

## CERVICAL RIB

- Congenital condition of C7 vertebra costal process enlargement.
- Usually asymptomatic but can cause thoracic outlet compression → hand or forearm pain, weakness or numbness, and thenar or hypothenar wasting.
- Radial pulse may be weak.
- X-ray of thoracic outlet may show cervical rib-but symptoms are sometimes due to fibrous bands that are not seen on X-ray.

***REFER to upper limb orthopaedic surgeon for further assessment.***

## WHIPLASH INJURIES

- Neck pain resulting from stretching or tearing of cervical muscles and ligaments due to sudden extension of the neck-often due to a RTA.
- Pain and decreased neck mobility typically starts several hours or days after injury.
- Pain may radiate to shoulders, arms, and head.

### ***Management***

- Examine carefully to exclude bony tenderness requiring X-ray.
- Treat with analgesia and early mobilization-collar may help initially but avoid long-term use.
- Recovery is often slow and 40% patients suffer long-lasting symptoms.
- As a general rule of thumb, the quicker the symptoms develop, the longer they will take to disappear.
- Early physiotherapy, if available, can improve recovery rate.
- Psychological problems and medicolegal issues can affect progress.
- Daily Stretching and Strengthening Exercises: Advise the patient to hold the neck for 10 seconds in each of the six positions (right and left lateral flexion and rotation, flexion and extension within the pain range. Repeat 10 times.

### ***Reference:***

1. *Oxford handbook of General Practice, 4<sup>th</sup> Edition*
2. *Alex Khat Andrew Polmear Practical General Practice, 4<sup>th</sup> Edition*

# SHOULDER PROBLEMS

## Relevance to GP

- Shoulder pain is responsible for approximately 16% of all musculoskeletal complaints and has a yearly incidence of 15 new episodes per 1,000 patients seen in the primary care setting; an estimated 20% of the population will suffer an episode of shoulder pain during their lifetime.
- Shoulder pain is second only to low back pain in patients seeking care for musculoskeletal ailments in the primary care setting.
- Peak incidence of shoulder pain occurs during the fourth through sixth decades but can affect all patients, young and old, particularly athletes

## Definition:

- Shoulder pain is defined as pain that is localized to the shoulder joint. It can have a primary (shoulder joint) or secondary (referred or systemic) etiology.

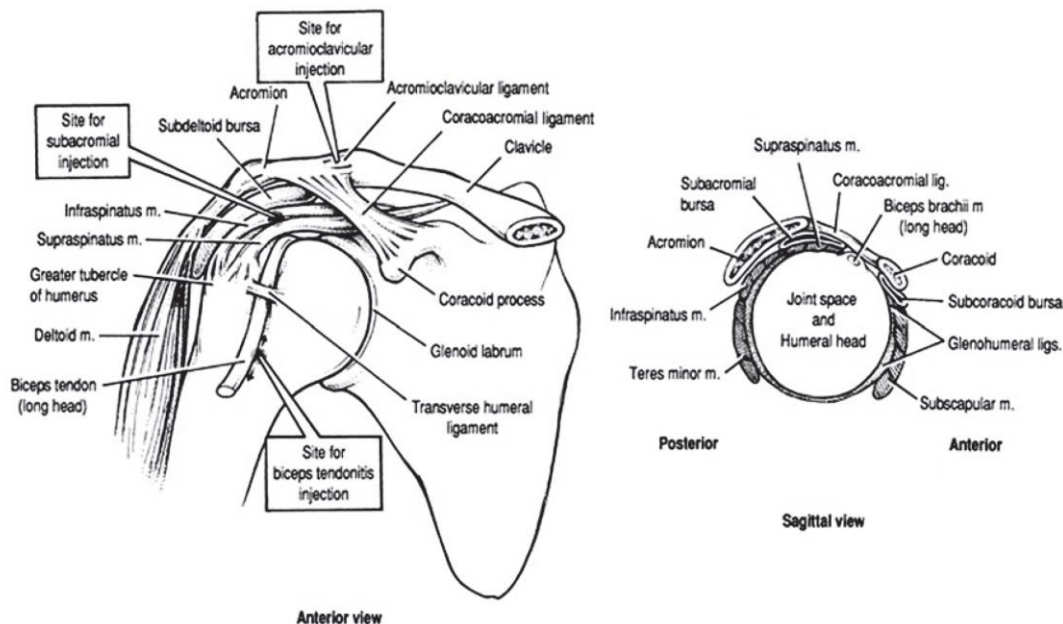


Fig. Anatomy of the shoulder joint.

## History

- Pain and stiffness joint pain is felt anteriorly and may radiate down the arm; pain on top of the shoulder suggests acromioclavicular joint problems or cervical spine disorders.
- Pain in the shoulder may be referred from the neck, heart, mediastinum, or diaphragm
- Deformity, swelling of the shoulder; prominence of the acromioclavicular (AC) joint;
- winging of the scapula
- Loss of function Difficulty reaching behind back (e.g. doing up brastrap ), brushing hair, or dressing

## Examination

- **Look:** Posture; asymmetry; muscle wasting; swelling (large effusions can be seen anteriorly); scars
- **Feel:** Tenderness; warmth; swelling; crepitus



- **Move/measure:** Compare sides. Check range of movement; complex movements (e.g. scratching opposite scapula in 3 ways, hands behind head, arm across front of chest to top of opposite shoulder); power

### Special test

- Liff off test (to diagnose rotator cuff problems)
- Drop arm test (to diagnose rotator cuff problems)
- Empty can test (supraspinatus tendon problems)
- Yergason's test (bicep tendonitis)
- **General rules:** Intra-articular disease - painful limitation of movement in all directions; tendonitis - painful limitation of movement in one plane only; tendon rupture or neurological lesions - painless weakness.

### Differential Diagnosis of the Patient with Shoulder Pain

Diagnosis	Typical Features	Frequency in Family Diagnosis Typical Features Medicine Clinic
Rotator cuff disorders (tendinopathy, partial tears, complete tears)	Often associated with repetitive overhead shoulder activities (Partial tears most common at older than age 40 years (10)) <ul style="list-style-type: none"> <li>• Complete tears most common at older than age 60 years (10)</li> <li>• Pain localized to the deltoid region</li> <li>• Pain worse with overhead activities and at night (11)</li> <li>• Pain and or weakness of the rotator cuff muscles on manual testing</li> <li>• Positive impingement tests</li> </ul>	Very common
Adhesive capsulitis	Pain with progressive loss of both active <i>and</i> passive range of motion <ul style="list-style-type: none"> <li>• Associated with diabetes, females, and middle decades of life (40–60 years)</li> </ul>	Common
Shoulder joint arthritis	Acromioclavicular joint arthritis: <ul style="list-style-type: none"> <li>• All ages with history of repetitive overhead lifting or heavy arthritis: common weight training</li> <li>• Pain localized to superior aspect of shoulder (acromioclavicular joint)</li> <li>• Pain worse at night and with cross body movements</li> <li>• Tenderness at acromioclavicular joint on exam as well as with cross body adduction testing</li> </ul>	Acromioclavicular joint : common
	Glenohumeral joint arthritis: <ul style="list-style-type: none"> <li>• Most common over age of 60 years</li> <li>• History of trauma, rheumatologic disease</li> <li>• Deep, diffuse pain localized to shoulder region</li> <li>• Loss of passive range of motion in more advanced cases, can be confused for adhesive capsulitis</li> </ul>	Glenohumeral arthritis: rare
Shoulder instability	<ul style="list-style-type: none"> <li>• Most common in young patients and athletes</li> <li>• Often prior history of acute shoulder injury or fall with frank young patients and dislocation or sensation of “pop” or “shift” in the shoulder athletes</li> <li>• Dislocation/subluxation most commonly occurs with arm in an abducted and externally rotated position</li> <li>• Positive apprehension test</li> </ul>	Common, particularly young patients and athletes

### Referred Shoulder Pain

• Hand	• Carpal tunnel
• Neck	• Cervical radiculopathy, muscle spasm
• Thoracic spine	• Myofascial pain (trapezius, rhomboid, levator scapulae)

• Chest	• Cardiac pain from MI (referred to left shoulder)
	• Pneumothorax (patient also c/o acute onset of dyspnoea)
	• Aortic dissection/aneurysm (pain between the scapulae)
• Abdomen	• Diaphragmatic irritation (gall bladder to right scapula, diffuse process to shoulder)
• Other	• Polymyalgia rheumatic, complex regional pain syndrome)

### **Red Flags for Patients with Shoulder Pain Indicating More Serious Disease**

• Red Flags	• Associated Conditions
• Fever, chills, redness of joint, intravenous drug use, immune deficiency	• Glenohumeral joint infection
• Anorexia, Weight Loss	• Malignancy (primary or metastatic)
• Persistent shoulder pain in children <8 years or in elderly without history of trauma or overuse	• Malignancy, especially sarcoma in elderly (rare)
• Profound shoulder weakness	• Massive rotator cuff tear
• Fall or trauma	• Fracture or large rotator cuff tear (particularly in elderly)
• Multiple joint involvement with effusion	• Auto-immune etiology
• Numbness, tingling or radiation of pain past elbow	• Cervical etiology
• Muscle atrophy	• Peripheral nerve injury

### **Causes of a stiff, painful shoulder joint**

- Adhesive capsulitis- primary or secondary to DM or intrathoracic pathology
- Inflammation - inflammatory arthritis (e.g. RA, psoriatic), infection
- Osteoarthritis
- Prolonged immobilization, e.g. haemiplegia, strapping after dislocation
- Polymyalgia rheumatic

## **SHOULDER OA**

- Often occurs after a history of trauma. Less common than knee or hip OA. Often associated with crystal-induced inflammation and secondary causes of OA (e.g. gout, haemochromatosis).
- Imaging for synovitis (USS/MRI) is important to rule out disease that may benefit from Steroid injection.
- Shoulder replacement may be considered in severe cases.

## **FROZEN SHOULDER (ADHESIVE CAPSULITIS)**

- Over diagnosed in primary care. Affects patients aged 40-60years.
- Painful, stiff shoulder with global limitation of movement -notably external rotation. Pain is often worse at night.
- Cause unknown, but increased in diabetics and those with intrathoracic pathology (MI, lung disease) or neck disease.

## Management

- If not known to be diabetic, check fasting blood glucose.
- NSAIDs, physiotherapy, and local steroid injection can all be helpful. May take > 1 year to recover and long-term outcome is uncertain.
- If restricted movements are slow to return consider orthopaedic referral.

## ROTATOR CUFF INJURY

- The shoulder is the most mobile joint in the body and relies on the musculo-tendinous rotator cuff to maintain stability.
- Disorders of the rotator cuff account for most shoulder pain.
- *Acute tendinitis* Often caused by excessive use/trauma in patients < 40 years.
- Presents with severe pain in the upper arm. Patients hold the arm immobile and are unable to lie on the affected side. Usually starts to resolve spontaneously after a few days. In middle age can be caused by inflammation around calcific deposits - requires steroid injection
- *Rotator cuff tears* may accompany subacromial impingement pain and is difficult to diagnose clinically unless the tear is large - suspect if impingement pain is recurrent. Refer
- Subacromial impingement. Pain occurs in a limited arc of abduction (60-120° -
- *painful arc syndrome*) or on internal rotation due to acromial or ligament pressure on a damaged rotator cuff tendon. In patients < 40 years, associated with glenohumeral instability from generalized connective tissue laxity or labral injury. In older patients, often due to chronic rotator cuff tendinitis or functional cuff weakness/tear

## Investigations:

- X-ray may show calcification of the supraspinatus tendon in acute tendinitis and irregularities/cysts at the humeral greater tuberosity if chronic cuff tendinitis.

## Treatment

- Rest followed by mobilization and physiotherapy, NSAIDs, and/or subacromial steroid injection.
- If conservative measures fail, refer for imaging, arthroscopy, and consideration for surgery.

## SHOULDER DISLOCATION

- Usually due to fall on arm or shoulder - anterior dislocation is most common. Shoulder contour is lost (flattening of deltoid) and the head of the humerus is seen as an anterior bulge.
- Axillary nerve may be damaged. It causes absent sensation on a patch below the shoulder.
- Refer to A&E for X-ray and reduction.
- In young patients, 30% have recurrent dislocations afterwards due to labral tear. Dislocation is associated with rotator cuff tear in 25% of elderly patients.

## Recurrent dislocation:

- Usually anterior and follows trauma - but 5% recurrent dislocations are in teenagers with no trauma but general joint laxity. Refer for specialist physiotherapy and consideration of surgery.

## ACROMIOCLAVICULAR JOINT PROBLEMS

- Pain on the top of the shoulder or in the suprascapular area suggests a problem with the acromioclavicular (AC) joint or neck.
- AC joint pain is usually due to trauma or OA - joint tenderness and pain are present on palpation and passive horizontal adduction.

### **Management:**

- NSAIDs ± local steroid injection.

## SHOULDER PAIN

- **The three joints of the shoulder** – Sternoclavicular, acromioclavicular and glenohumeral joint.
- The four rotator cuff muscles:
- Supraspinatus (initiation of adduction)
- Infraspinatus (external rotation)
- Subscapularis (internal rotation)
- Teres minor (external rotation)
- These joint together to form the rotator cuff tendon which travels through the sub-acromial space.
- Rotator cuff tendonitis
- It is where inflammation which causes impingement (where the tendon becomes trapped.) reduces the range of movement, hence a painful arc.
- For example, if there is a supraspinatus tendonitis there is compression of supraspinatus tendon between the humeral head and acromion

### **Causes**

- Tendonitis secondary to an injury or repetitive strain or calcium deposits.
- Subacromial space narrowing: bony spurs from wear and tear or enlargement of bursa.
- As USS may be helpful to diagnose and guide a steroid injection.
- It may also diagnose tears in the rotator cuff tendons, some of which may require surgical repair.
- Most tendonitis settles with rest. Physio and NSAID/ steroid injection. Occasionally, (e.g if chronic /bony spur) arthroscopic decompression is necessary.

## FROZEN SHOULDER (ADHESIVE CAPSULITIS)

- It is generalized inflammation within the capsule, which cause pain and limits any ROM of the glenohumeral joint.
- It usually affects the non dominant shoulder. Frozen shoulder may occur after rotator cuff injury or spontaneously and cause is unknown.
- There are typically three phases:
- **Painful freezing stage** – most painful stage with gradual loss of movement, may last up to 9 months.

- **Frozen/Adhesive stage** – reduced movement but less painful, last up to 1 year
- **Thawing/ Recovery phase** -gradual return to normal function which may take 1-3 years.
- Tell patients that symptoms may last between 1 months and 3 years, although the vast majority of patients have recovered by 2 years.
- The treatment is physiotherapy.
- A steroid injection may be helpful within the first 8 weeks.
- Patients with diabetes or thyroid conditions have an increased risk of a bilateral frozen shoulder.
- Osteoarthritis:
  - In an older patient it may be helpful to see if there is OA present.
  - Dislocations
    - usually occur after an impact injury.
  - Consider referred pain from the diaphragm (ruptured ectopic pregnancy) or heart (MI).
  - If you are ever giving a PII then describe what is on it!
  - If unable to abduct the shoulder the first 10 degrees, consider a complete tear of the supraspinatus and speak to a specialist about ranging a clinical appointment.

## References

1. Chantal Simon; et.al, *Oxford handbook of General Practice-Fourth edition,2014*
2. Philip D. Sloane MD; et.al, *Essential of Family Medicine- Sixth edition,2012*

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# ELBOW PROBLEMS

## DIAGNOSTIC WORKUP

### **History**

- Pain and stiffness: Joint pain is diffuse; pain well localized over the medial or lateral epicondyles may be due to tendinitis
- Deformity: Swelling? Nodules? Structural deformity?
- Loss of function: May be limitation of flexion, extension, pronation, and/or supination. This can affect function, e.g. causing difficulty eating (can't get hand to mouth) or with personal care.
- Neurology: Numbness and paraesthesiae distal to the elbow - particularly in the ulnar nerve distribution

### **Examination**

- Look Carrying angle (11° for male 13° for female). Effusion may be visible either side of the olecranon. A discrete swelling over the olecranon could be RA nodule, gouty tophus, olecranon bursa, or other nodule. Check for muscle wasting
- Feel: Tenderness? Swellings? Warmth? If indicated test neurology and check pulses distal to the elbow.
- Move Active and passive movements. Compare both sides. Normal range is from 0° in full extension to 145° in full flexion. Check pronation/supination. Normal range is 75° and 80° respectively.

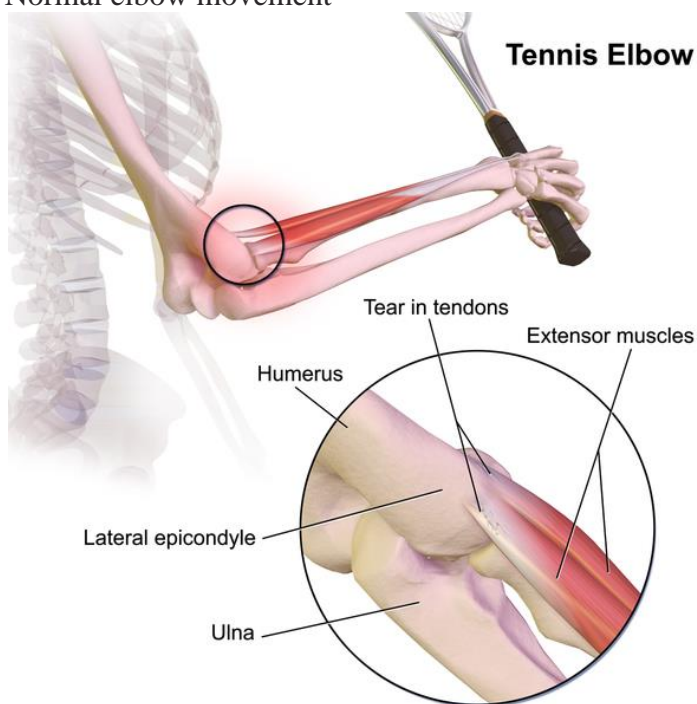
## TENNIS ELBOW AND GOLFER'S ELBOW (EPICONDYLITIS)

- Common extensor tendon inflammation at the epicondyle
- CAUSE: repeated strain.
- Tennis elbow - tenderness over the lateral epicondyle and lateral elbow pain on resisted wrist extension

### **Lateral tennis elbow: typical clinical profile**

- **Age:** 40-60 years
- **Occupation:** Carpenter, bricklayer, housewife, gardener, dentist, violinist, Sport Tennis, squash
- **Symptoms:** Pain at outer elbow, referred down back of forearm. Rest pain and night pain (severe cases). Pain in the elbow during gripping hand movements (e.g. turning on taps, turning door handles, picking up objects with grasping action, carrying buckets, pouring tea, shaking hands)
- **Signs:** No visible swelling
- Localised tenderness over lateral epicondyle, anteriorly Pain on passive stretching wrist
- Pain on resisted extension wrist and third finger

Normal elbow movement



[https://upload.wikimedia.org/wikipedia/commons/b/bb/Tennis\\_Elbow.png](https://upload.wikimedia.org/wikipedia/commons/b/bb/Tennis_Elbow.png)



<https://blog.wimi-fitness.com/wp-content/uploads/2016/06/tennis-elbow-test-2.jpg>

<https://blog.wimi-fitness.com/wp-content/uploads/2016/06/tennis-elbow-test-3.jpg>

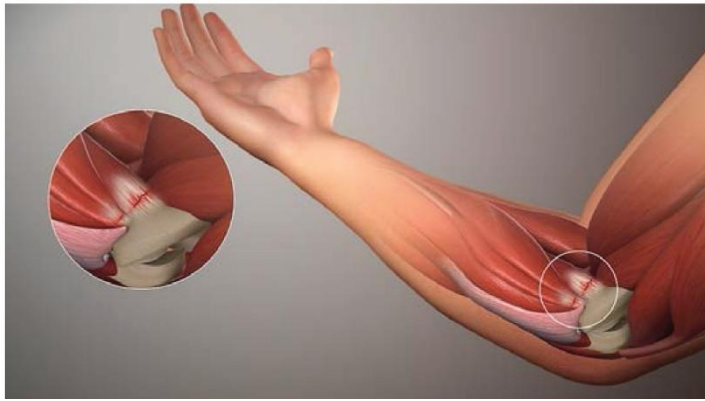
- Course: 6 to 24 months

### **Management basic:**

- rest from offending activity
- RICE and oral NSAIDs if acute (RICE: rest, ice, compression, elevation)
- exercises-stretching and strengthening
- Additional (if refractory):
- corticosteroid/LA injection (max. two)
- Manipulation
- Surgery

## **GOLFER'S ELBOW**

- Tenderness over the medial epicondyle and medial elbow pain on resisted wrist pronation.
- The pain is felt on the inner side of the elbow and does not radiate far.
- The main signs are localised tenderness to palpation and pain on resisted flexion of the wrist.



[https://upload.wikimedia.org/wikipedia/commons/3/31/Golfers-Elbow\\_SAG.jpg](https://upload.wikimedia.org/wikipedia/commons/3/31/Golfers-Elbow_SAG.jpg)

## Management

- Stop trigger movements if possible. Often settles with time  $\pm$  NSAIDs. Recovery is speeded by local steroid injection although relapse is more common after injection. Physiotherapy may help, as may an epicondylar clasp.
- Exercises: Stretching and strengthening exercises for the forearm muscles represent the best management for tennis elbow. Three options are presented:
- With the arm extended, grasp the towel with the affected side placed in neutral.
- Then exert maximum wring pressure: first flexing the wrist for 10 seconds, then extending the wrist for 10 seconds. This is an isometric 'hold' contraction. This exercise should be performed only twice a day, initially for 10 seconds in each direction. After each week increase the time by 5 seconds in each twisting direction until 60 seconds is reached (week This level is maintained indefinitely.
- Note: Despite severe initial pain, the patient must persist, using as much force as possible. Review at 6 weeks to check progress and method.
- '*Weights*' exercise. The muscles are strengthened by the use of hand-held weights or dumbbells.
- A suitable starting weight is 0.5 kg, building up gradually (increasing by 0.5 kg) to 5kg, depending on the patient.

## Method

- To perform this exercise the patient sits in a chair beside a table.
- The arm is rested on the table so that the wrist extends over the edge.
- The weight is grasped with the palm facing downwards in lateral epicondylitis and upwards in medial epicondylitis
- The weight is slowly raised and lowered by flexing and extending the wrist.
- The flexion/extension wrist movement is repeated 10 times, with a rest for 1 minute, and the program is repeated twice.
- The pronating exercise. A suitable stretching exercise is to rhythmically rotate the hand and wrist inwards with the elbow extended and the forearm pronated.

## DISLOCATED ELBOW

- Usually due to fall on outstretched hand with flexed elbow. Ulna is displaced backwards,
- elbow is swollen and held in fixed flexion. May have associated fracture. Refer to hospital for reduction.



## OLECRANON BURSTITIS

- Olecranon bursitis presents as a swelling localised to the bursa (which has a synovial membrane) over the olecranon process. The condition may be caused by trauma, arthritic conditions (rheumatoid arthritis and gout) or infection.
- Traumatic bursitis may be caused by a direct injury to the elbow or by chronic friction and pressure as occurs in miners (beat elbow), truck drivers or carpet layers.
- Acute olecranon bursitis with redness and warmth can occur in rheumatoid arthritis, gout, pseudogout, haemorrhage and infection (sepsis). Septic bursitis must be considered where the problem is acute or subacute in onset. Aspiration of the bursa contents with appropriate laboratory examination is necessary (smear, Gram stain, culture and crystal examination).
- Treatment depends on the cause.

## ULNAR NEURITIS

- Narrowing of the ulnar groove (from OA, RA, or post-fracture) causes pressure on the ulnar nerve and ulnar neuropathy.
- Clumsiness with the hand is often the first symptom, then weakness wasting of hand muscles innervated by the ulnar nerve and decreased sensation in the little finger and medial half of the ring finger. Rule out metabolic and autoimmune causes of a mononeuritis and refer for consideration of surgical decompression ± nerve conduction studies if entrapment is likely

## PULLED ELBOW

- Common in children <5years.
- Traction injury to elbow causes subluxation of radial head.
- Often occurs when the child is pulled up suddenly by the hand. Child will not use the arm. No clinical signs. M > F. Left arm > right.
- Xrays are unhelpful.

### **Management**

- Apply anterior pressure with the thumb on the radial head whilst supinating and extending the forearm. Immediate recovery is seen after reduction.

### **Reference**

1. *Oxford handbook of General Practice (4<sup>th</sup> Edition)*
2. *John Murtagh 's General Practice (6<sup>th</sup> Edition)*

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# KNEE PROBLEMS

- DIAGNOSTIC WORKUP

## **History**

- Trauma: History of injury - ask about degree and direction of force.
- Pain/stiffness: Attempt to distinguish well-localized mechanical pain and diffuse inflammatory/degenerative pain
- Deformity: Swelling? If injury, time of onset of swelling in relation to history (immediate effusion suggests haemarthrosis; post-traumatic effusions appear later). Knock-knees or bow-legs?
- Age-related causes of painful knee
- First decade (0-10 year)
- Infection
- Juvenile chronic arthritis
- Second decade (10-20 years)
- Patellofemoral syndrome
- Subluxation/dislocation of patella
- Slipped femoral epiphysis (referred)
- 'Hamstrung' knee
- Osteochondritis dissecans
- Osgood-Schlatter disorder
- Anserinus tendonopathy
- Third decade (20-30 years)
- Bursitis
- Mechanical disorders
- Fourth and fifth decades (30-50 years)
- Cleavage tear of medial meniscus
- Radial tear of lateral meniscus
- Sixth decade and older (50 years & over)
- Osteoarthritis
- Osteonecrosis
- Paget disease (femur, tibia or patella)
- Anserinus bursitis
- Chondrocalcinosis and gout
- Osteoarthritis of hip (referred pain)

## **Examination**

- Always compare the two knees.
- Look: Watch the patient walk. Look at the knees whilst standing - varus/valgus deformity?
- Ask: the patient to lie down. Note quadriceps wasting, scars, skin changes, swelling, and deformity. A space under the knee viewed laterally suggests a fixed flexion deformity. With legs extended, lift both feet off the bed to demonstrate hyperextension.
- Feel: Feel the quadriceps for wasting and palpate the knee for warmth. Check the joint line, collateral ligaments, tibial tubercle, and femoral epicondyles for tenderness.
- Palpate: the popliteal fossa for a Baker's cyst. Check for an effusion. Test for patellofemoral lesions by sliding the patella sideways across the underlying femoral condyles.

- Move: With the patient lying on his back check active and passive range of movement
- pain reproduced on movement? Crepitus? Test the medial and lateral collateral ligaments and cruciate ligaments.
- NB. Knee pain can be referred from the hip so examine the hip as well.

## OSTEOARTHRITIS OF THE KNEE

- Very common

### **Symptoms**

- Usually appear in middle life or later. It is more common in women, the obese, and in those with knee deformities (e.g. genu varum) or previous trauma, especially meniscal tears.
- The degenerative changes may involve either the lateral or the medial tibiofemoral compartment, the patellofemoral joint or any combination of these sites.

### **Diagnosis**

- Confirmed by X-ray (weight-bearing view)

### **Treatment:**

- Education
- Relative rest
- Weight loss
- Analgesics and/or judicious use of NSAIDs
- Walking aids and other supports
- Physiotherapy (e.g. hydrotherapy, quadriceps exercises, mobilisation and stretching techniques)
- Intra-articular injections of corticosteroids are generally not recommended but a single injection for severe pain can be very effective.
- Surgery is indicated for severe pain and stiffness and, especially for the medial compartment with focal arthritis and varus deformity.

## INFECTION OF THE KNEE JOINT (SEPTIC ARTHRITIS)

- Most commonly infected joint. Septic (pyogenic) arthritis should be suspected when the patient complains of intense joint pain, malaise and fever.

### **Signs:**

- Hot, red, swollen, painful knee.

### **Differential diagnosis:**

- Reiter's disease,
- gout,
- pseudo gout,
- traumatic effusion,
- RA.
- If infection is suspected refer as an emergency to rheumatology or orthopaedics.

## RHEUMATOID ARTHRITIS

- The knee is frequently affected by rheumatoid arthritis (RA) although it rarely presents as monoarticular knee pain.
- RA shows the typical features of inflammation - pain and stiffness that is worse after resting. Morning stiffness is a feature.
- *Note:* The spondyloarthropathies have a similar clinical pattern to RA. Synovectomy is a useful option with persistent boggy thickening of synovial membrane but without destruction of the articular cartilage.

## BIPARTITE PATELLA

- Detected on X-ray. Usually asymptomatic incidental finding but can cause pain due to excessive mobility of a patella fragment.
- If troublesome refer for fragment excision.

## PATELLAR DISLOCATION

- Lateral dislocation of the patella and tearing of the medial capsule/quadriceps can occur due to trauma. More common in young people and if joint hypermobility syndrome, patient is in pain and unable to flex knee.
- REFER to Accident and Emergency Department or orthopaedics for reduction.

## RECURRENT SUBLUXATION OF THE PATELLA

- Medial knee pain+ knee 'gives way' due to lateral subluxation of the patella Most common in girls with valgus knees
- ASSOCIATIONS: familial, hypermobility, high-riding patella.

### **Signs:**

- increased lateral patella movement and +ve apprehension test (pain and reflex contraction of quadriceps on lateral patella pressure).

### **Refer**

- to physiotherapy for vastus medialis exercises.
- If that is unhelpful, refer to rheumatology to exclude a hereditary connective tissue disorder and/or to orthopaedics for consideration of lateral retinacular release.

## PATELLA TENDINITIS

- Small tear in the patella tendon causes pain. Most commonly seen in athletes.

### **Differential**

- includes inferior patellar pole enthesitis (spondylarthropathies), fat-pad syndrome, anterior cartilage lesion, and bursitis.

**Diagnosis** is with USS.

### **Treatment**

- is with rest,
- NSAIDs ±steroid injection around (not into) the tendon.

## **BURSITIS**

- Prepatellar bursitis (housemaid's knee) is associated with excess kneeling.
- Vicar's knee (infrapatellar bursitis) is associated with upright kneeling.
- Educate to avoid aggravating activity, and refer orthopaedics. .

## **BAKER'S CYST**

- A popliteal cyst (Baker cyst) is a herniation of a chronic knee effusion between the heads of the gastrocnemius muscle and usually is associated with osteoarthritis (most common), rheumatoid arthritis or internal derangement of the knee.
- It presents as a mass behind the knee and may or may not be tender or painful. It tends to fluctuate in size.
- A Baker's cyst indicates intra-articular pathology and indicates a full assessment of the knee joint
- Rupture may result in pain and swelling in the calf, mimicking DVT.
- Treat underlying knee inflammation (synovitis). Surgical removal of the cyst is advisable for persistent problems.

## **COLLATERAL LIGAMENT INJURY**

- Common in contact sports. Causes knee effusion if severe ± tenderness over the injured ligament. Collateral ligaments provide lateral stability to the knee.
- Normally there is <5° of movement - if >5° the ligament may be ruptured.
- TREAT with rest, knee support, analgesia.
- REFER to orthopaedic surgeon if rupture is suspected.

## **CRUCIATE LIGAMENT INJURY**

- Cruciate ligaments provide anterior/ posterior knee stability.
- Assessment can be difficult.

## **ANTERIOR CRUCIATE TEARS**

- Result from a blow to the back of tibia ± rotation when the foot is fixed on the ground.
- Signs: effusion and positive drawer test (supine with foot fixed and knee at 90°, pull the tibia forward test is positive if the tibia moves forward on the femur)

## **POSTERIOR CRUCIATE TEARS**

- Cause: e.g. when the knee hits the dashboard in car accidents.
- Reverse drawer test is positive (supine with knee at 90°; apply pressure to push the tibia backwards-test is positive if the tibia moves backward on the femur)

## ***Management***

- Refer to orthopaedics if suspected.
- Splinting and then physiotherapy helps most (60%) but some require reconstructive surgery-consider urgent referral if keen sportsman.

## LOOSE BODIES IN THE KNEE

- May result in locking of the joint and/or effusion.
- CAUSES: OA, chip fractures, osteochondritis dissecans, synovial chondromatosis.
- If problematic refer for removal.

## OSTEOCHONDRITIS DISSECANS

- Necrosis of articular cartilage and underlying bone can cause loose body formation. Cause is unknown.
- Seen in young adults and pain after exercise and intermittent knee swelling  $\pm$  locking. Predisposes to arthritis.
- REFER for expert management.

## MENISCAL LESIONS

- Twisting with the knee flexed can cause medial (bucket handle) meniscal tears and adduction with internal rotation can cause lateral cartilage tears.

### ***Symptoms/signs:***

- Locking of the knee-extension is limited due to cartilage fragment lodging between the condyles
- Giving way of the knee
- Tender joint line
- Positive McMurray's test-rotation of the tibia on the femur with flexed knee followed by knee extension causes pain and a click, as the trapped cartilage fragment is released. X ray reliability of this test is debated

### ***Management***

- Refer for investigation and treatment.

## MENISCAL CYST

- Pain+ swelling over the joint line due to a meniscal tear. Lateral cysts are more common than medial. The knee may click and give way.
- Refer to orthopaedics.

## CHONDROMALACIA PATELLAE

- Common in teenage girls. Pain on walking up or down stairs or on prolonged sitting.
- SIGNS: pain on stressing the undersurface of the patella.
- TREAT with analgesia+ physiotherapy (vastus medialis strengthening decrease pain in 80%). If persistent, exclude spondylarthropathy and refer to orthopaedics.

## OSGOOD-SCHLATTER DISEASE

- Seen in athletic teenagers. Pain and tenderness  $\pm$  swelling over the tibial tubercle. X-rays not required.
- Avoid aggravating activities.



- Usually settles over a few months. If not settling refer to orthopaedics or rheumatology for further assessment.

# BOW-LEGS AND KNOCK-KNEES IN CHILDREN

## GENUVARUM (BOW-LEGS)

- Outward curving of the tibia usually associated with internal tibial torsion. Except in severe cases always resolves spontaneously.
- Severe cases raise the possibility of rickets or other rare developmental disorders-refer for orthopaedic opinion.

## GENUVALGUM (KNOCK-KNEES)

- Common amongst 2--4years olds.
- Innocent if symmetrical and independent of any other abnormality. Severe, progressive cases suggest rickets-refer for X-ray.

### ***Principles of management***

- Most painful knee conditions are not serious and, providing a firm diagnosis is made and internal knee disruption or other serious illness discounted, a simple management plan as outlined leads to steady relief For more serious injuries the primary goal is to minimise the adverse consequences of forced inactivity.
- First aid: RICE (avoid heat in first 48 hours).
- Lose weight if overweight.
- Adequate support for ligament sprains- supportive elastic tubular (Tubigrip) bandage or a firm elastic bandage over Velband.
- Simple analgesics-paracetamol (acetaminophen).
- Judicious use ofNSAIDs and corticosteroid injections.
- Physiotherapy to achieve strength and stability.
- Attend to biomechanical abnormalities, inappropriate footwear and athletic techniques.
- Orthotics and braces to suit the individual patient.
- Quadriceps exercises: these simple exercises are amazingly effective.

### ***Quadriceps exercises (examples)***

- Instruct the patient to tighten the muscles in front of the thighs (as though about to lift the leg at the hip and bend the foot back but keeping the leg straight). The patient should hold the hand over the lower quadriceps to ensure it is felt to tighten. This tightening and relaxing exercise should be performed at least 6 times every 2 hours or so until it becomes a habit.
- It can be done sitting, standing or lying.
- Sitting on a chair the patient places a weight of 2-5 kg around the ankle (e.g. a plastic bag with sand or coins in a sock) and lifts the leg to the horizontal and then gently lowers it (avoid in patellofemoral problems).

### ***When to refer***

- Early referral is required for knees 'at risk' following acute injuries where one or more of the followings are present:
  - locked knee
  - Haemarthrosis
  - Instability
  - Clinical evidence of a tom cruciate ligament, third degree tear of the collateral ligaments or tom meniscus

- Undiagnosed acute or chronic knee pain
- Recurrent subluxation or dislocation of the patella
- Suspected septic arthritis
- Presence of troublesome intra-articular loose body
- Severe pain and stiffness and, especially for the medial compartment with focal arthritis and varus deformity of OA knee.
- Knee pain
- Osteoarthritis

### **Diagnosis**

- Diagnose osteoarthritis clinically without investigations if a person:
- Is 45 or over and
- Has activity-related joint pain and
- Has either no morning joint-related stiffness or morning stiffness that lasts no longer than 30 minutes
- Atypical features include history of trauma, prolonged morning joint related stiffness, rapid worsening of symptoms or the presence of a hot swollen joint.

### **Differential diagnosis**

- Gout
- Septic arthritis
- Inflammatory arthritis
- Malignancy
- Injury from trauma
- NICE advises a holistic assessment of a person with OA:
- social situation and impact,
- health beliefs,
- mood,
- quality of sleep,
- support network,
- other MSK pain,
- attitudes to exercise,
- influence of comorbidity and
- a full pain assessment.

### **GP management options include:**

- NSAIDs
- Muscle strengthening exercise
- Advice about aerobic fitness and foot wear
- Physiotherapy
- Steroid injections
- Consider additional needs for TENS or aid.

### **When to consider surgery**

- Mechanical locking or loose bodies on X ray for arthroscopic larvae and debridement.
- After at least three core (non-surgical) treatment option
- (Basic core treatment – weight loss, appropriate exercise, suitable footwear)
- Symptoms causing a significance impact on life or ongoing functional limitation

# ANTERIOR CRUCIATE LIGAMENT INJURY

## ***Mechanism***

- Most commonly associated with a non-contact mechanism – knee in and toe out.
- The risk is greater in female.

## ***Injury triad***

- Anterior cruciate ligament + medial collateral ligament +medial meniscus
- Acute injury
- Refer acute injuries to orthopaedics for assessment, imaging and management options.
- NB: Immediate effusions following trauma / injury are often due to haemarthrosis which is associate with more significant joint injuries.
- Have a low threshold for referral of such cases (possibly same day or urgently to trauma /fracture clinic).
- If presenting as a chronic injury, you may be able to refer for direct access MRI to guide diagnosis and management.
- Check local protocols.

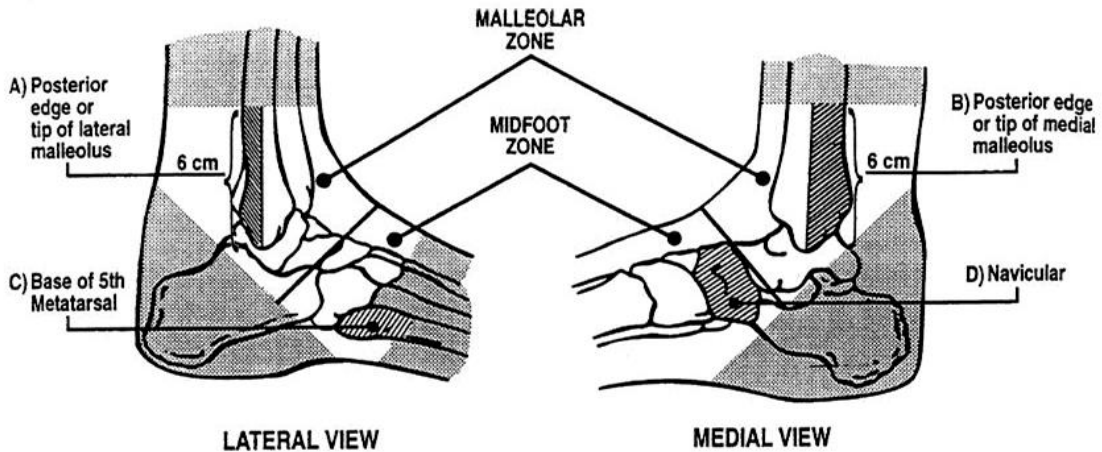
## ***Reference***

1. *Oxford handbook of General Practice, 4th Edition*
2. *John Murtagh's General Practice, 6th Edition*

# ACUTE ANKLE INJURY

- The Ottawa Ankle Rule reduces the need for X-rays following ankle injury by 30-40% (see Table). Note that the rule is very good at identifying patients who do not need an X-ray (high sensitivity). It is poor at identifying those who have a fracture (low specificity). An ankle X-ray is required if there is any pain in the malleolar zone and:
  - there is bone tenderness at the posterior edge or tip of the lateral malleolus; or
  - there is bone tenderness at the posterior edge or tip of the medial malleolus; or
  - the patient is unable to weight bear both at injury and when seen.

Figure 5. The Ottawa Ankle Rules for ankle and midfoot injuries.



- a) An ankle x-ray series is only required if:  
There is any pain in malleolar zone and any of these findings:
- bone tenderness at A **or**
  - bone tenderness at B **or**
  - inability to bear weight both immediately and in the ED
- b) A foot x-ray series is only required if:  
There is any pain in midfoot zone and any of these findings:
- bone tenderness at C **or**
  - bone tenderness at D **or**
  - inability to bear weight both immediately and in the ED

Reproduced with permission from: Michael JA, Stell IG. Ankle injuries. In: Tintinalli JE, Kelen GD, Stapczynski JS, eds. *Emergency Medicine: A Comprehensive Study Guide*. New York: McGraw-Hill; 2000:1828. Figure 268-2.

Figure 5. The Ottawa Ankle Rules for ankle and midfoot injuries.

- An ankle x-ray series is only required if:
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    - bone tenderness at A or
    - bone tenderness at B or
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  - b) A foot x-ray series is only required if:
    - There is any pain in midfoot zone and any of these finding!.:
      - bone tenderness at C or
      - bone tenderness at D or
      - inability to bear weight both immediately and in the ED
- Reproduced with permission from: Michael JA, SteiiiG. Ankle Injuries. In: Tintinalli JE, Kelen GO, Stapczynski JS, eds. *Emergency Medicine: A Comprehensive Study Guide*. New York: McGraw-Hill; 2000:1828. Figure 268-2 .
- [https://meds.queensu.ca/central/assets/modules/reproducibility !The\\_Ottawa\\_Ankle\\_RulesJar\\_ankle\\_and\\_midfo ot\\_injuries\\_Emegency\\_Medicine\\_Practice\\_1.JPG](https://meds.queensu.ca/central/assets/modules/reproducibility!The_Ottawa_Ankle_RulesJar_ankle_and_midfo ot_injuries_Emegency_Medicine_Practice_1.JPG)

Table: Reliability of the Ottawa Ankle Rules for identifying ankle and foot fractures or avulsion fractures

Patients Groups	Sensitivity (95%CI)	Specificity (Interquartile Range)	LR-
All patients	96% (94 to 99 %)	26% (19 to 34 %)	0.10
Ankle injuries	98% (96 to 99 %)	40% (30 to 48 %)	0.08
Midfoot injuries	99% (97 to 100 %)	38% (25 to 70 %)	0.08

LR - is the likelihood ratio for a negative result.

- A foot X-ray is required if there is pain in the midfoot zone and:
- there is bone tenderness at the navicular; or
- there is bone tenderness at the base of the 5th metatarsal; or
- patient is unable to weight bear both at injury and when seen.
- The immediate treatment of an ankle sprain (as for any injured joint or limb) is RICE (rest, ice, compression, elevation). Of these, compression appears to be the most important and, with elevation, must be maintained for at least 48 hours. Ice should be applied no more than 20 minutes at a time, three times a day and the skin should be separated from the ice by a wet towel. It is an approach based on experience rather than evidence.
- Analgesia, support with mobilization, immobilization and surgical repair are all used in inversion injuries of the ankle. There is no robust evidence to guide the clinician in their use although the use of support and early mobilization seems to result in faster recovery and better long-term outcome.
- Rehabilitation after ankle injuries
- Recommend active mobilization to restore proprioception. This can be achieved by regular exercises:
- imagine writing the alphabet with the foot, first capitals then small letters;
- balance on the injured leg while moving the free leg forward and backward and side- to- side; initially with eyes open then with eyes shut;
- use a wobble board.

# FOOT PAIN

## Differential diagnosis of foot pain

• Soft tissue	• Chronic heel pad inflammation	• Warm dull throbbing pain over weight bearing area of the heel → worse when first getting up
	• Acute synovitis	• Throbbing pain made worse by • Movement → rule out systemic causes
	• Acute inflammation of anterior metatarsal heads	• Common in women wearing slip-on / heeled shoes → burning and throbbing on walking
	• Planter metatarsal bursitis	• Throbbing pain under metatarsal head → persist at rest and exacerbated when the area if first loaded
• Bone	• Fracture	• Has there been impact? Use the Ottawa rules.
	• Stress fracture (march fracture)	• Palpable tender lump
	• Osteoarthritis	• Common at first MPjt, tarsus joint and mid foot
	• Rheumatoid arthritis	• Swelling and deformity, may describe walking on pebbles due to swelling/subluxation
	• Sever's disease	• e.g boys 8 -13 years and exacerbated by jumping
	• Hallux valgus	• Great toe moves towards /overlies the 2 <sup>nd</sup> toe
	• Bunion	• Inflamed and painful metatarsal head
• Red hot	• Gout / septic arthritis	• Requires urgent aspiration /culture/ Rx
• Nerve	• Morton/s neuroma	• Burning and numbness → often the 3 <sup>rd</sup> /4 <sup>th</sup> toe → Metatarsal squeeze test (mulder's click)
	• Peripheral neuropathy	• e.g tarsal tunnel or secondary to decrease B12, alcohol or DM
• Arterial	• ischaemia	• Absent pulses, signs of Peripheral Vascular Disease (PVD) and check ABPIs.

## Reference

1. Oxford handbook of General Practice, 4<sup>th</sup> Edition

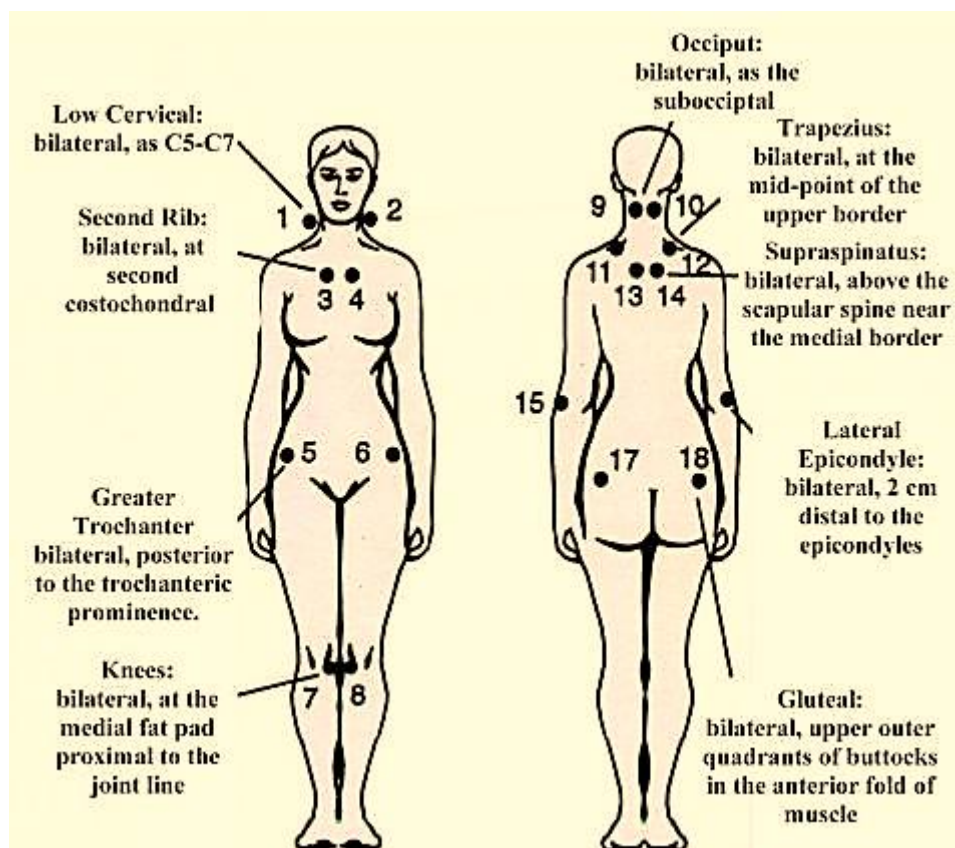
# FIBROMYALGIA SYNDROME

## Definition

- Fibromyalgia syndrome is a distinct syndrome of widespread, chronic pain. The mechanism for pain is thought to be driven by central sensitization.

## Clinical features

- The main diagnostic features are:
- history of widespread pain (neck to low back)
- pain in 11 of 18 tender points on digital palpation
- These points must be painful, not tender. Smythe and Moldofsky have recommended 14 of these points on a map as a guide for management. These are:
- *Occiput*: suboccipital muscle insertions.
- *Low cervical*: anterior aspects of the intertransverse space at C5-C7.
- *Trapezius*: midpoint of upper border.
- *Supraspinatus*: origin, above the scapula spine near medial border.
- *Second rib*: at the second costochondral junction on upper surface.
- *Lateral epicondyle*: 2 cm distal to epicondyle.
- *Gluteal*: in upper outer quadrants of buttocks in anterior fold of muscle.
- *Greater trochanter*: posterior to the trochanteric prominence,
- *Knee*: the medial fat pad proximate the joint line.



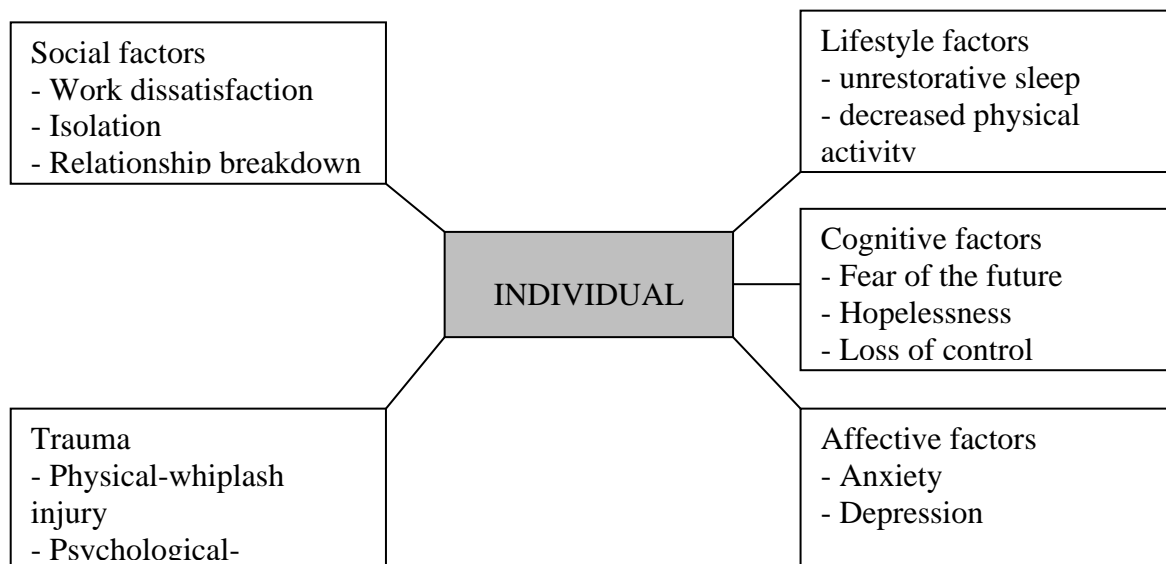
- <https://upload.wikimedia.org/wikipedia/commons/2/23/Fibromyalgia.jpg>



- symptoms have been present for over 3 months Other features
- Female to male ratio= 4:1
- Usual age onset 29-37 years: diagnosis 44-53 years
- Poor sleep pattern
- Dermatographia
- Fatigue (similar to chronic fatigue syndrome)
- Psychological disorders (e.g. anxiety, depression, tension headache, irritable digestive system)
- This disorder is very difficult to treat and is usually unresponsive in the long term to passive physical therapy or injections.
- Limited investigations to exclude other causes of widespread pain are usually undertaken at presentation (thyroid function tests, full blood count, inflammatory markers, serum calcium and alkaline phosphatase, biochemical profile, creatinine kinase, random blood glucose).
- There is no diagnostic test for fibromyalgia.

## Cause

- No single pathophysiological causative mechanism has been identified, and fibromyalgia is a multifactorial syndrome characterized by abnormal processing of pain, known as central sensitization.



## Treatment

- Explanation, reassurance and counselling
- Attention to sleep disorders, stress factors and physical factors
- Relaxation program
- Rehabilitation: graduated exercise program (e.g. walking, water exercises, swimming or cycling)
- Use paracetamol for first-line analgesia
- Find out the patient's concerns, worries and answer appropriately. A patient centered approach has been found to be associated with less pain and less distress 1 year later.
- Advise: gradually increasing aerobic exercise of the sort recommended for cardiovascular fitness.
- In severe unremitting cases, consider referral to a multidiscipline team.
- Consider referral for cognitive behavioral therapy.

### **Medication (often disappointing)**

- Antidepressants (of proven short-term value); start low then monthly increments as tolerated: amitriptyline 10-75 mg PO (every night) may help with sleep/pain. Pregabalin (150- 300 mg/12 hr) PO can be alternative).
- SSRI (Selective Serotonin Reuptake Inhibitor) e.g. sertraline 25-50 mg od, may help anxiety, depression, and sleep
- Or
- duloxetine 30 mg (o) (morning), increasing to 60 mg over 2 weeks Stop if no improvement after a month's trial
- Note: NSAIDs are of no proven benefit.

### **Reference:**

1. *ABC Rheumatology*
2. *John Murtagh's General practice, 4th Edition*
3. *Alex Khat Andrew Polmear Practical General Practice, 6<sup>th</sup> Edition*

# GOUT (MONOSODIUM URATE CRYSTAL DISORDERS)

## **Definition:**

- Gout is an abnormality of uric acid metabolism resulting in hyperuricaemia and urate crystal deposition. Urate crystals deposit in:
  - joints - acute gouty arthritis
  - soft tissue - tophi and tenosynovitis
  - urinary tract - urate stones
- Four typical stages of gout are recognised:
  - Stage 1 -asymptomatic hyperuricaemia
  - Stage 2 -acute gouty arthritis
  - Stage 3 -intercritical gout (intervals between attacks)
  - Stage 4 -chronic tophaceous gout and chronic gouty arthritis
- Asymptomatic hyperuricaemia:
  - 10 times more common than gout
  - Elevated serum uric acid (>0.42 mmol/L in men,> 0.36 mmol/L in women)
  - Absence of clinical manifestations
  - Usually does not warrant treatment

## **Clinical features**

- Typical clinical features of gout include:
  - mainly a disorder of men (5-8% prevalence)
  - onset earlier in men (40-50) than women (60 +)
  - acute attack: excruciating pain in great toe, early hours of morning
  - skin over joint- red, shiny, swollen and hot
  - exquisitely tender to touch
  - relief with colchicine, NSAIDs, corticosteroids
  - can subside spontaneously (3 to 10 days) without treatment

## **Causes/precipitating factors**

- Alcohol excess (e.g. binge drinking)
- Surgical operation
- Starvation
- Drugs (e.g. frusemide, thiazide diuretics)
- Chronic kidney disease
- Myeloproliferative disorders
- Lymphoproliferative disorders (e.g. leukaemia)
- Sugary soft drinks
- Cytotoxic agents (tumour lysis)
- Hypothyroidism
- Low-dose aspirin
- Others
- THE ARTHRITIS
  - Monoarthritis in 90% of attacks:
    - MTP joint great toe - 75%
    - other joints- usually lower limbs: other toes, ankles, knees
  - Polyarticular onset is more common in old men and may occur in DIP and PIP joints offingers. No synovial joint is immune.

- Other features
- Prone to recurrence
- Tophi in ears, elbows (olecranon bursa), big toes, fingers, Achilles tendon (take many years)
- Can cause patellar bursitis
- Can get cellulitis (does not respond to antibiotics)

## NODULAR GOUT

- Develops in postmenopausal women with kidney impairment taking diuretic therapy who develop pain and tophaceous deposits around osteoarthritic interphalangeal (especially DIP) joints of fingers.

### **Diagnosis**

- Blood: increased WCC; increased ESR;
- Elevated serum uric acid: (up to 30% can be within normal limits with a true acute attack)
- Synovial fluid aspirate: typical uric acid crystals using compensated polarized microscopy; this should be tried first (if possible) as it is the only real diagnostic feature
- X-ray: Not usually required, shows soft tissue swelling only, unless severe disease when an erosive pattern is seen

### **Management**

- Management of gout includes these principles:
- good advice and patient education information
- provision of rapid pain relief
- preventing further attacks
- prevention of destructive arthritis and tophi
- dealing with precipitating factors and comorbid conditions (e.g. alcohol dependence, obesity, CKD, polycythaemia vera, diabetes, hypertension)
- The acute attack
- Resolves in <2week-often after 2-7days if treated.
- Exclude infection
- Rest and elevate joint - apply ice packs
- NSAIDs are helpful - e.g. naproxen 500mg bd - caution if GI problems
- Note: Any other NSAID can be used.
- Alternatively, if NSAIDs are contraindicated, try colchicine
- Colchicine: 0.5 mg (o) stat, then 0.5 mg every 6 or 8 hours until pain relief (usually 24-28 hours) or diarrhoea develops (max. 6 mg/24 hours)
- Note:
- Must be given early
- Avoid if kidney impairment
- Avoid use with macrolide antibiotics e.g. clarithromycin especially in CKD
- Avoid long-term use
- Consider:
- corticosteroids: intra-articular following aspiration and culture (gout and sepsis can occur together); a digital anaesthetic block is advisable. An oral course can be used: start with prednisolone 40 mg/day for 4 days then decrease gradually over 10 days

- corticotrophin (ACTH) IM in difficult cases (e.g. synthetic ACTH: tetracosactrin 1 mg IM)
- Note:
- Avoid aspirin and urate pool lowering drugs (probenecid, allopurinol, sulphapyrazone)
- Monitor kidney function and electrolytes
- Long-term therapy
- When acute attack subsides preventive measures (life style modification and dietary advice) include:
- weight reduction
- a normal, well-balanced diet
- avoidance of purine-rich food, such as organ meats (liver, brain, kidneys, sweetbread), tinned fish (sardines, anchovies, herrings), shellfish and game
- reduced intake of alcohol
- reduced intake of sugary soft drinks
- good fluid intake (e.g. water-2 litres a day)
- avoidance of drugs such as diuretics (thiazides, frusemide) and salicylates/low- dose aspirin
- wearing comfortable shoes.
- increase exercise
- control of co-morbidities(eg; hypertension, hyperlipidaemia)
- Prevention (drug prophylaxis)
- 

Allopurinol (a xanthine oxidase inhibitor) is the drug of choice: dose 100-300 mg daily.

Febuxostat (80 mg/24 hr) is an alternative. Indications:

frequent acute attacks (> 1 attack in 12 months)

tophi or chronic gouty arthritis

kidney stones or uric acid nephropathy

hyperuricaemia Adverse effects:

rash (2%)

severe allergic reaction (Steven Johnson's Syndrome) Precautions:

beware of kidney insufficiency and elderly patients - use lower doses

beware of drug interactions:

azathioprine and 6 mercaptopurine - potentially lethal

amoxicillin - prone to rashes

### ***Method: treatment of intercritical and chronic gout***

- Commence 6-8 weeks after last acute attack.
- Start with 50 mg daily for the first week and increase by 50 mg weekly to maximum 300 mg.
- Check uric acid level after 4 weeks: aim for level <0.38 mmol/L.
- Add colchicine 0.5 mg bd for 6 months (to avoid precipitation of gout) or NSAIDs.
- Probenecid (uricosuric agent)
- Good for hyperexcretion of uric acid by blocking renal tubular reabsorption.
- Dose: 500 mg/day (up to 2 g)
- *Note:* Aspirin antagonises effect.
- Gout may be linked to increased risk of hypertension and coronary heart disease - screen patients

## **Refer to rheumatologist**

- Any patient with gout and kidney stones or recurrent UTI to Urology
- Recurrent attacks, tophi (urate deposits) in pinna, tendons and joints, and joint damage
- Suspected septic arthritis
- Uncertained diagnosis
- Suspicious underlying systemic illness (e.g. rheumatoid arthritis, connective tissue disorder)
- Gout occurs during pregnancy or under 25 yr of age.

## **Risk factors**

- Thiazides, ACEI, alcohol, obesity
- Do not start allopurinol during an acute attack
- Do not stop allopurinol or debuxostat during an acute attack if treatment is already established.

## **Acute medication**

- 1<sup>st</sup> line NSAID (Diclofenac, indomethacin or naproxen)
- 2<sup>nd</sup> line colchicine
- 3<sup>rd</sup> line if NSAID contraindicated – consider systemic corticosteroids
- When initiating allopurinol consider a course of NSAID or colchicine. If in 2/12 you need to increase the allopurinol give another overlap of colchicine to prevent an acute attack as the urate levels decrease again.
- Uric acid levels measure 4-6 weeks after an acute attack.
- Aim for normal levels of uric acid if the decision has been made to start allopurinol.

## **Self-management**

- Rest
- Avoid trauma
- Keep the joint cool
- Lifestyle
- Reduce alcohol
- Weight control
- Dietary changes
- Avoid dehydration

## **Reference**

1. *John Murtagh's Handbook of General Practice, 6<sup>th</sup> Edition*
2. *Oxford Handbook of General Practice, 4<sup>th</sup> Edition*

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# OSTEOARTHRITIS

## Definition

- Osteoarthritis (OA) is the most important cause of locomotor disability. It used to be considered 'wear and tear' of the bone/cartilage of synovial joints but is now recognized as a metabolically active process involving the whole joint i.e. cartilage, bone, synovium, capsule, and muscle.

## Important risk factors for osteoarthritis

Risk Factor	Notes
Genetics	Hand, knee and hip OA show strong heritability (40-60%): this probably results from combinations of multiple common polymorphisms rather than rare
Race	Knee OA is prevalent across the world, whereas hip OA is particularly prevalent in
Age	Although not an inevitable consequence of ageing, OA is strongly age-related; this may reflect the cumulative effect of insults to the joint aggravated by
Sex	Women have a higher prevalence and radiographic severity of OA at all joints sites apart from the hip. Women are also more likely to have symptoms if
Obesity	This is an important risk factor for knee OA, but a more modest risk factor for hip
Bone density	High density is a risk factor for development of knee, hip and hand OA; low density
Abnormal joint shape and alignment	Acetabular dysplasia is a recognized cause of hip OA, and distal femoral dysplasia (often overlooked) may contribute to knee OA; varus or valgus mal alignment may be a risk for development and more rapid progression
Joint trauma and usage	Major joint injury is an important factor at the knee (especially if it causes subchondral fracture, meniscal injury or ligament rupture) and can cause OA at any site; recognized occupational hazards include farming (hip OA), underground mining (knee OA), professional soccer (knee OA) and some

- Primary OA is usually symmetrical and can affect many joints.
- In primary OA all the synovial joints may be involved, but the main ones are:
  - first carpometacarpal (CMC) joint of thumb
  - first metatarsophalangeal (MTP) joint of great toe
  - distal interphalangeal (DIP) joints of hands
- Other joints that are affected significantly are the proximal interphalangeal joints, the knees, hips, acromioclavicular joints and joints of the spine, especially the facet joints of the cervical (C5-6, C6-7) and lumbar regions (L3-4, L4-5, L5-S1).

## Clinical features

- Pain: worse by the end of the day, aggravated by use, relieved by rest, worse in cold and damp
- Variable morning stiffness
- Variable disability

## **Signs**

- Hard and bony swelling
- Crepitus
- Signs of inflammation (mild)
- Restricted movements
- Joint deformity
- Note: There should be no systemic manifestations.
- Crystal arthropathy can complicate OA, especially in the fingers of people taking diuretics (e.g. nodular gout).

## **Differentiation from an inflammatory arthropathy**

- OA does not exhibit the typical inflammatory pattern. The clinical diagnosis based on:
- Gradual onset of pain after activity (worse towards the end of the day)
- The pattern of joint involvement
- The lack of soft tissue swelling
- The transient nature of the joint stiffness or gelling
- Takes <30 minutes to settle after rest while inflammatory arthritis takes at least 30 minutes

## **Diagnosis**

- The diagnosis is clinical and radiological but the degree of changes on X-ray do not always parallel levels of symptoms.

## **RAY findings**

- Joint space narrowing with sclerosis of subchondral bone
- Formation of osteophytes on the joint margins or in ligamentous attachments
- Cystic areas in the subchondral bone
- Altered shape of bone ends
- Exclude other causes of pain, e.g. check FBC and ESR if inflammatory arthritis is suspected (normal or mildly increase in OA, ESR >30mm/h suggests RA or psoriatic arthritis).
- Mnemonics of X-ray finding (LOSS)
- loss of joint space
- Osteophytes
- subarticular sclerosis
- subchondral cyst

## **Management of osteoarthritis in primary care**

- The goals of medical management of OA are to:
- provide patient education and information access;
- relieve pain;
- (c) optimize function; and
- minimize disease progression

## **Patient education and information access**

- This is a professional responsibility, but education also improves outcome and is a treatment in its own right and use of educational programme to help patients understand OA and develop self management strategies.



## **Exercise**

- Local quadriceps-strengthening exercise can reduce pain and disability and improve the physiological accompaniments of knee OA (muscle weakness, impaired proprioception and balance, tendency to fall). Aerobic activity also reduces pain and disability from OA, improves well-being and sleep-quality, and is beneficial for common co-morbidities. Both forms of exercise need to be prescribed. Increased activity and exercise can be accomplished in a variety of ways (e.g. home exercise, group classes), tailored to the patient's wishes and lifestyle.

## **Reduction of adverse biomechanical factors**

- Spreading physically hard jobs (e.g. housework, mowing the lawn) at intervals through the day, with breaks in between ("pacing") can reduce sustained mechanical loading. Weight reduction can improve function and reduce pain in obese and overweight patients and may slow progression of knee and hip OA. Appropriate footwear (thick soft sole, no raised heel, broad forefoot and deep soft uppers) can reduce impact loading in people with knee and hip OA, and wedged in soles can counteract knee varus deformity . Walking sticks and other walking aids reduce loading across OA joints.

## **Pharmacological treatment**

- Pain is the main reason patients seek help.
- Paracetamol should be the first oral analgesic (1-2 qid regularly) to try, based on its excellent safety and reasonable efficacy.
- Topical non-steroidal anti-inflammatory drugs (NSAIDs) and topical capsaicin
- are also safe and are particularly useful for hand and knee OA.
- Oral NSAIDs including highly selective COX inhibitors, and weak opioids (e.g. codeine, tramadol) may be considered for those patients who obtain insufficient relief from paracetamol and/or topical agents.
- The increased risk of gastrointestinal ulceration and bleeding from traditional NSAIDs can be decreased by concomitant prescription of a proton pump inhibitor or misoprostol.
- Oral NSAIDs and selective COX inhibitors therefore should be given at the lowest effective dose on an as-required, rather than regular, basis . Weak opioids, either alone or in combination with paracetamol, may provide good pain relief, but central nervous system side effects (e.g. constipation, headache, confusion) often limit their usefulness.
- Low-dose antidepressants, e.g. amitriptyline 10-75 mg, are a useful adjunct especially for pain causing sleep disturbance
- Intra-articular corticosteroid injection is a valuable treatment that often gives quick effective relief of pain that may last just a few weeks to a few months. It is particularly useful to tide a patient over an important event (e.g. family wedding, holiday) and to improve pain during initiation of other interventions such as an exercise programme.

## **Surgery**

- The success of prosthetic joint replacements has greatly advanced management of end-stage hip and knee OA.
- The criteria for referral for consideration of joint replacement include:
- Uncontrolled pain and
- Severe impairment of function despite conservative treatment
- Age, in itself, is not a contraindication.
- Psychological factors
- Psychological factors have a major impact on the disability from OA. Seek and treat depression and anxiety with screening tools.

## **Refer**

- To rheumatologist:
- to confirm diagnosis if coexistent psoriasis (psoriatic arthritis mimics OA and can be missed by radiologists);
- rule out secondary causes of OA (e.g. pseudo gout, haemochromatosis) if young OA or odd distribution
- To orthopaedic surgeon:
- if symptoms are severe for joint replacement
- as an emergency if you suspect joint sepsis
- To physiotherapist
- for advice on exercises especially isometric exercises for the less mobile

## **Reference**

1. *Oxford handbook of General Practice, 4<sup>th</sup> Edition*
2. *Oxford handbook of Clinical Medicine, 10<sup>th</sup> Edition*

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# OSTEOPOROSIS

## Definition

- Osteoporosis, which literally means porous bone, is **reduced bone mass** per unit volume thus predisposing the person with it to an increased risk of fracture. It also refers to the increased bone fragility that accompanies ageing and many illnesses.

## Key facts and checkpoints

- Osteoporosis is silent, common, measurable, treatable and potentially lethal (analogous to hypertension).
- Osteoporosis is commonest in postmenopausal women.
- Up to 50% of women will develop fractures in their lifetime and 30% of all women reaching 90 years of age will suffer a hip fracture.
- Osteoporosis leads to reduced bone strength and susceptibility to fracture, even with minor trauma.
- Osteoporosis usually causes pain when complicated by fracture.
- First presentation is usually a fracture (Colles, femoral neck and vertebra) or height shrinkage.
- Vertebral collapse is the hallmark of osteoporosis.
- The disorder is of low bone mass.
- For osteoporosis in a vertebra including a pathological fracture, multiple myeloma needs exclusion.
- The first step in prevention is regular exercise, an adequate dietary intake of calcium (1500 mg per day) and maintenance of adequate serum vitamin D levels.

## Classification

- PRIMARY (AGE RELATED)
- Type 1:
  - Postmenopausal (vertebral or distal forearm fractures between the ages of 51 and 75)
  - Due to increased osteoclast activity
  - 6 times more common in women than men
- Type 2:
  - Involutional or senile osteoporosis (fracture of proximal femur and other bones).
  - It affects patients over 60 years and is twice as common in women as in men.
  - Idiopathic osteoporosis: Occurs in children and young adults of both sexes with normal gonadal function.
- SECONDARY
  - Secondary to various endocrine disorders, malabsorption and malignancies. Various causes and risk factors are presented in Table 1.

### Table 1. Osteoporosis: risk factors and/or causes

- Constitutional and non-modifiable
- Female sex
- Ageing
- Thin build; low BMI <18; short stature
- Race: Asian, Caucasian
- Family history (e.g. maternal hip fracture <75 yrs)
- Premenopausal oestrogen deficiency (e.g. amenorrhoea)
- Late menarche

- Early menopause <45 years (natural or surgical)
- Modifiable lifestyle factors
- Cigarette smoking
- High alcohol intake >2 standard drinks per day
- Low calcium intake
- Lack of vitamin D
- Physical inactivity
- Medical causes
- Eating disorders (e.g. anorexia nervosa)
- Malabsorption syndrome (e.g. coeliac disease)

- Endocrine disorders:
  - Cushing syndrome
  - diabetes mellitus
  - hyperparathyroidism
  - thyrotoxicosis
  - amenorrhoea in elite athletes
  - hypogonadism/sex hormone deficiency
  - acromegaly
- Connective tissue disorders (e.g. RA)
- Chronic organ failure (kidney, liver, heart, lungs)
- Drugs causing bone loss:
  - corticosteroids
  - anti-epileptic drugs, especially hepatic enzyme inducers
  - thiazolidinediones for diabetes
  - long-term heparin
  - excessive thyroid hormone
  - prostate cancer hormone therapy
  - breast cancer hormone therapy
- Prolonged immobilization
- Plasma calcium, phosphate and alkaline phosphatase (usually normal).
- Thyroid stimulating hormone.

- Consider tests for multiple myeloma in an osteoporotic area.
- Densitometry can predict an increased risk of osteoporosis and fracture, the best current modality being dual energy X-ray absorptiometry (DEXA scan) in a facility with high-standard quality control. The spine and femoral neck are targeted: the femoral neck is the most useful index.

### ***Osteoporosis risk factors (SHATTERED)***

- **S** = Steroid use of more than 5 mg per day of prednisolone
- **H** = Hyperthyroidism, hyperparathyroidism, hypercalciuria, **A**= Alcohol and tobacco use increased
- **T** =Thin
- BMI <18.5
- **T** = reduced Testosterone, anti-androgen in carcinoma prostate treatment
- **E** = Early menopause
- **R** =Renal or liver failure
- **E** = Erosive inflammatory bone disease, e.g. myeloma, rheumatoid arthritis
- **D** = Dietary reduced calcium, malabsorption, diabetes mellitus type I,

### ***Investigations***

- Plain radiography is of limited value (low sensitivity, low specificity) Osteoporosis is not detectable until 40-50% of bone is lost.

- 25-hydroxy vitamin D (most useful test): normal range 75-250 nmol/L

## **Dexa, T scores and Z scores**

- Dual energy X-ray absorptiometry (DEXA) is the current gold standard for the diagnosis of osteoporosis. It assesses both whole-body and regional bone mass (lumbar spine and proximal femur). Bone mass is measured as bone mineral density (BMD) in g/cm<sup>2</sup> and the lower the BMD, the higher the risk of fracture. There are actually different normal ranges of BMD for each bone and for each type of DEXA measuring machine.
- The BMD 'T score' is the number of standard deviations (SD) away from the mean BMD of a 30-year-old adult (Table 2). Osteopenia (low bone density) is -1-2.5 SDs below the young adult standard mean. Osteoporosis is > -2.5 SD below this mean. This is a strong indicator of bone fragility. Consider treatment if T score is < -2.5.

- Table 2. Interpretation of T scores (WHO criteria)
- T score Interpretation

≥ (-)1	Normal
(-)1 – (-)2.5	Osteopenia
≤ (-)2.5	Osteoporosis
< (-)2.5	With fracture severe osteoporosis

- The BMD 'Z score' is the number of SDs away from the **age- and sex-matched mean BMD**. The Z score is used to express bone density in patients <50 years, premenopausal women, younger men and children. If low (<-2) it indicates prompt investigation for underlying causes of a bone deficit.
- BMD is recommended for healthy women aged over 50 with all the risk factors for osteoporosis of:
  - postmenopause
  - fracture after age 40 with minimal trauma
  - family history of osteoporosis, smoking habit or low BMI (< 18)

## **Treatment**

- The goal of treatment is to prevent osteoporosis or reduce further loss. Eliminate risk factors where possible and focus on optimal lifestyle measures as a baseline for management. No treatment has been shown to replace lost bone effectively. Anabolic agents such as nandrolone decanoate may reduce further loss but the side effects are problematic.

## **Medications of value in decreasing further loss**

- The following medications may be valuable in preventing further bone loss, possibly reversing the osteoporosis process and preventing further fractures.
- **1. bisphosphonates** (decrease bone absorption) can be used alone or combined with other agents (take care with potential adverse effects of oesophagitis and osteonecrosis of jaw):
  - alendronate 10 mg (o) daily or 70 mg (o) once weekly (take care with potential side effect of oesophagitis)
  - If intolerant,
  - etidronate 400 mg (o) for 14 days then calcium carbonate 1250 mg (o) for 76 days
  - risedronate 5 mg (o) daily or 150 mg (o) once monthly or 35 mg (o) once weekly or in combination therapy with calcium carbonate ± vitamin D zoledronic acid, single annual IV injection

- decrease bone loss and fracture rate. Mainstay of treatment for osteoporosis. Avoid if severe CKD or woman of child bearing age (possible teratogenic effects).
- Instructions for use:
- Take on an empty stomach first thing in the morning, 2:30 min before food/other medication; take in an upright position washed down with plenty of water; sit upright for 30min after taking.
- Atypical femoral fracture
- Prolonged bisphosphonate treatment >5years cause over suppression of bone turnover and increase bone fragility. Acute sub-trochanteric or mid-shaft femoral fractures are most common. To prevent this, a 'drug holiday' of 1-5years has been proposed for low-risk patients after 5years use-follow local guidance.
- **2. Strontium ranelate** has been shown to both increase osteoblastic bone formation and reduce osteoclastic bone resorption. Given at 2 grams daily orally, it may be used as first line therapy in high risk patient or in those intolerant of bisphosphonates. It is indicated in post-menopausal osteoporosis for reduction of fracture risk in hip and vertebrae but increased risk of cardiac problems.
- **3. HRT** (long-term use is not recommended but weigh potential benefits versus harms with the patient)
- **4. Raloxifene, selective estrogen receptor modulators (SERM)** is recommended as 2nd line of medical treatment. It is effective for prevention and treatment of vertebral fracture in post-menopausal women. Prescribed dosage is 60 mg once daily.

### ***Recommendations for prevention***

- Adequate dietary intake of calcium: 1200-1300 mg per day in both men and women.
- Dairy food is the main source of dietary calcium. Calcium-rich foods include low-fat calcium-enriched milk (500 mL contains 1000 mg), other low-fat dairy products (e.g. yoghurt or cheese), fish (including tinned fish such as salmon with the bone), citrus fruits, sesame and sunflower seeds, almonds, brazil nuts and hazel nuts. Oral calcium supplements will be necessary in postmenopausal women or where a person's diet does not meet their daily calcium requirements. Calcium citrate is better absorbed than carbonate. Recommend: 3 calcium citrate 2.38 g (= 500 mg elemental calcium) daily *or* calcium carbonate 1.5 g (= 600 mg elemental calcium) daily with food
- Vitamin D deficiency and sunlight: there is evidence we need significant exposure to sunlight of the face, arms and hands to produce natural vitamin D (e.g. 15-30 minutes a day in all climates, up to 50 minutes a day in winter in temperate climates). 6 Refer to regional recommendations. Measure serum 25-hydroxy vitamin D and maintain it at 75-111 nmol/L. If supplementation is required use colecalciferol 25-50 mcg (1000- 2000 IU) oral daily until target 25-OH vitamin D level 75-111 nmol/L.
- Exercise: moderate exercise against gravity- walking (brisk walking for 30 minutes four times a week), jogging or tennis-may make a small contribution to retarding bone loss.
- Lifestyle factors: stop smoking and limit alcohol and caffeine intake.
- Adequate nutrition: keep BMI >18.
- Attention to falls prevention, including avoiding sedative medication.
- Provide 'hip protectors' especially to osteoporotic patients at increasing risk of fractures after falling, but adherence is poor.

## Monitoring osteoporosis treatment

- Recommendations are to measure BMD at the lumbar spine and hip:
- 2 years after therapy begins
- 1-2 years after therapy changes significantly
- more frequently in patients at higher risk of bone loss

## OSTEOPOROSIS IN CHILDREN

- The main problem in children is secondary osteoporosis, which is usually related to chronic inflammatory disorders and their treatment with corticosteroids and also to reduced mobility. Other medical causes are malignancy, malabsorption syndromes, poor nutrition, anorexia nervosa and hypogonadism. Use DEXA to assess and monitor BMD and Z scores. Refer for treatment, which may be based on bisphosphonates.

## OSTEOPOROSIS IN MEN

- Currently only bisphosphonates and teriparatide (recombinant PTH, increased risk of renal malignancy) are recommended for treatment of osteoporosis in men.

## WHEN TO REFER

- Refer postmenopausal women and older men to a specialist according to individual needs
- Osteoporosis appears to be secondary to an underlying illness
- Advice is required about the management of a patient with pathological osteoporotic fractures or loss of height
- Fragility fracture on treatment.

## Osteoporosis (NICE Guideline)

Risk factors (low)	Female, BMI <18.5, smoking, Alcohol (units>14/week for female and >21/week for male. Previous fracture or history of falls, immobility, FH osteoporosis or hip fracture, medical mobility.
Risk factors (medium)	Corticosteroids, premature menopause, previous osteoporotic fracture
Risk factors (high)	>7.5 mg prednisolone daily for 3 months (current or recent) Previous major osteoporotic fracture Multiple fragility fracture

## Who do we screen

- Female, >65 years or <65 years with any risk factor
- Male >75 years or <75 years with any risk factor
- Anyone <50 years with a medium or high-risk factors or <40 years with a high-risk factor

## How do we screen?

- History – is the patient risk?
- Calculate the FRAX score if age 40 – 90 years or Q fracture if age 30 – 84 years
- Once the data is inserted into the tool, use the link to NOGG to guide management – lifestyle advise (low risk)
- DEXA scan (intermediate risk) – recalculate with BMD(Bone Mineral Density).
- Start treatment (High risk).
- Above the upper limits defined by the tools, consider people to be a high risk.
- >80 years predicted 10 year fracture risk may underestimate their short-term fracture risk.
- Assessment tools underestimate risk in: multiple fractures or vertebral fracture, alcohol use, steroid treatment or comorbidities.

T score	
≥-1.0	Normal
Between -1 and -2.5	Osteopenia
< -2.5	Osteoporosis
≤-2.5	With feacture, severe osteoporosis

- **For reversible causes** - consider testing of:
- TSH for hyperthyroidism
- PTH for hyperparathyroidism
- Ca<sup>2+</sup> for Osteomalacia
- vitamin D,
- Testosterone – hypogonadism
- LH and FSH

## Who should have calcium and vitamin D replacement?

- Due to new studies demonstrating risks of calcium treatment, consider vitamin D replacement alone if dietary calcium intake is < 700 mg/day.
- If calcium intake is inadequate: Prescribe 10 micrograms (400 IU) of vitamin D with at least 1000 mg calcium daily.
- Prescribe 20 microgram (800IU) of vitamin D with at least 1000 mg of calcium daily for elderly people who are housebound or living in a nursing home.

## Who should have bisphosphonates?

- NOGG guidance has indicated treatment is required or following a confirmed osteoporosis diagnosis.
- Only alendronate (OD tablets) and risedronate (once weekly tablets) are licensed for use in men.

## Management of Osteopenia

- Lifestyle
- Smoking
- Exercise and weight bearing exercise
- Test bone profile
- Blood test – Albumin, U&E, LFT, FBC, ESR, TSH
- Rescan 2-3 years
- To allow calcium intake calculation consultation



## Reference

1. *John Murtagh's General Practice, 6<sup>th</sup> Edition*
2. *Oxford handbook of General Practice, 4<sup>th</sup> Edition*
3. *Oxford handbook of Clinical Medicine, 10<sup>th</sup> Edition*
4. *Therapeutic manual (Internal Medicine), 1<sup>st</sup> Edition (2016)*

# RHEUMATOID ARTHRITIS

## **Definition**

- Rheumatoid arthritis (RA) is the most common disorder of connective tissue. It is an immunological disease, triggered by environmental factors, in patients with genetic predisposition. Disease course is variable with exacerbations and remissions.

## **Clinical features**

- Insidious onset but can begin acutely (explosive RA)
- Age 10-75 years, peak 30-50 years but bimodal 25-50 (peak age) and 65-75
- Female to male ratio= 3:1
- Joint Pain: Worse on walking, nocturnal pain, disturbed sleep: relieved with activity
- Morning stiffness can last hours.
- Rest stiffness - (e.g. after sitting)
- General: malaise, weakness, weight loss, fatigues
- Disability according to involvement

## **Signs**

- Soft swelling (effusion and synovial swelling) especially of wrist, MCP and PIP joints
- Warmth
- Tenderness on pressure or movement
- Limitation of movement
- Muscle wasting
- Later stage: deformity, subluxation, instability or ankylosing.
- Look for swan necking, boutonniere and Z deformities, ulnar deviation.
- Check for a number of everyday functions
- Power grip (lifting a jug of water)
- Precision grip (using a key or pen), undoing buttons
- Hook grip

## **Extra-articular manifestation**

- Anaemia, inflammatory to eye, sjogren's syndrome (dry eyes, dry mouth),
- Lymphadenopathy
- Pulmonary (pleural effusion, fibrosing alveolitis, nodules - caplan syndrome, vasculitis (pupura) nail fold infections and skin ulcer, cervical spine (atlantoaxial subluxation).
- Cardiac (pericarditis, myocarditis), splenomegaly (felty syndrome), subcutaneous nodules, bursitis, tenosynovitis, capal tumel syndrome, Raynaud's phenomenon, Baker cyst (popliteal fossa) peripheral sensory neuropathy, mononeuritis multiplex.

## **Diagnosis**

- According to ACR I EULAR (2010) RA criteria
- A score of >6/ 10 is needed to diagnose definite RA.

No.	Symptom	Score
A	Joint involvement (0-5)	
	1 medium-large joint	0
	2-10 medium-large joint	1
	1-3 small joints (with or without involvement of large joint)	2
	4-10 small joints (with or without involvement of large joint)	3
	> 10 joints (at least one small joint)	5
B	Serology (0-3)	
	Negative RF and negative anti-citrullinated protein antibodies	0
	Low positive RF or low positive anti-citrullinated protein antibodies	2
	High positive RF or high positive anti-citrullinated protein antibodies	3
c	Acute phase reactants	
	Normal CRP and normal ESR	0
	Abnormal CRP or abnormal ESR	1
D	Duration of symptoms	
	< 6 week	0
	>= 6 week	1

## Severity Assessment

- Disease Activity: Modified Disease Activity Score (DAS 28)

$$DAS\ 28 = 0.56 \times \sqrt{TJC28} + 0.28 \times \sqrt{SJC28} + 0.7 \times \log_{nat}(ESR) + 0.014 \times GH$$

*TJC = Tendon Joint Count, SJC = Swollen Joint Count, GH = Global Health, ESR = Erythrocyte Sedimentation Rate.*

- Can use DAS28 on-line
- <2.6 =Remission, 2.6-3.2 =Low Disease Activity, 3.2-5.1 =Moderate Activity, >5.1 =High Activity
- Poor prognostic factor
- Functional limitation (Functional Health Status III or IV)
- Extra articular manifestation
- Positive rheumatoid factor or anti-cyclic citrullinated peptide antibodies
- Bony erosions by radiograph
- Function Health Status (FHS)
- No handicap (Can perform activity of daily life (ADL), Vocational & A vocational works)
- Can perform ADL and vocational works.
- Can do ADL (self-care without assistant, may need minimal help sometimes)
- Need assistant for self-care, chair bound.
- Duration of symptoms
- <6 months or >6 months

## Management

- Early referral from primary care for suspected RA for early diagnosis
- Early intervention with Disease Modifying Anti-Rheumatic Drug (DMARD) in the window of opportunity before joint erosion occur

## **Patient assessment**

- Assess the impact on the patient's mental health, sleep, fatigue, acts of daily living and social function.
- Screen for and treat cardiovascular risk factors
- Check for extra-articular manifestations including constitutional upset

## **Pharmacological treatment**

- Pain Control
- **NSAIDs and simple analgesics:** (e.g. regular paracetamol) provide symptomatic relief but do not alter the course of disease. NSAIDs is started with the least gastric-toxic, e.g. ibuprofen 200-400 mg tds and alter as necessary, e.g. to naproxen 500mg bd. If the patient has a history of indigestion/gastric problems consider adding gastric protection, e.g. PPI, or, if there is no history of CVD, using a COX2 inhibitor, e.g. celecoxib 100 mg bd.
- It is customary to try a patient on one NSAID or coxib for a period of several weeks and then to switch to an alternatives if it is ineffective. If NSAIDs or coxibs are needed for the long term, prescribe the lowest effective dose
- **Steroids:** Daily low-dose oral steroids relieve symptoms and there is some evidence that they can modify disease progression, but concerns about adverse side effects have limited use. Consider using systemic steroid in the following situation
- 1) bridging disease control between different DMARD therapies
- to achieve rapid control of symptoms but only once the diagnosis has been established.
- Prednisolone 2.5- 10 mg per day
- \* Recommend local steroid injection for localized flare ups.

## **DMARD**

- They include antimetabolites, antimalarials and biologic agents (e.g. infliximab, rituximab, initiated by specialist). The choice of DMARD will be made by the patient and consultant. Methotrexate has emerged as the most commonly prescribed DMARD closely followed by sulfasalazine.
- Preparations before starting DMARD
- Clinical background
- Alcohol consumption Smoking
- Diabetes Hypertension
- Coronary Heart disease Menstrual history Contraception

## **Investigations**

- CRP or ESR, Cholesterol, Blood sugar, FBC, LFT, ALT, Urea and Creatinine
- HBs Ag, Anti-HCV
- Tuberculin test in those with history of close contact to open case ECG
- Chest X-ray (PA)

## **Vaccinations**

- Influenza vaccine Pneumococcal vaccine Hepatitis B vaccine
- In immunosuppressed patients not immune to measles or varicella consider using immunoglobulins after significant contact exposure. Avoid using live vaccines in patients taking immunosuppressive drugs. Withhold DMARDs/biologics if necessary and contact secondary care team.

- Methotrexate: 7.5- 15mg weekly+ folic acid 5mg weekly Leflunomide
- Body wt < 160 lb -7 60 mg od x 3 DS
- > 160 lb -7 100 mg od x 3DS Followed by 20 mg od
- CQ: 150 mg od
- Adjunctive Rx
- Omega 3 Fatty acid Lipid - Lowering Agent
- Prophylactic Rx for osteoporosis (see in osteoporosis guideline)

### ***THE GP's role***

- Support patients during the initiation of therapy. There may be no benefit for 2-6 months, counsel patient to report potential adverse effect promptly.
- **Pre-pregnancy Counselling:** Ensure patients know about the danger to conception. Several DMARDs are contraindicated in pregnancy. Both male and female patient may need to delay conception until a period has elapsed after stopping cytotoxics. The period depends on the DMARD e.g. 3 months for MTX, 2 years for women on leflunomide.
- Follow local protocols for monitoring the advance effect of these drugs, as protocols vary between institutions.

### ***Antidepressants***

- Ask about sleep disturbance and fatigue. Prescribe an antidepressant to aid sleep and reduce pain as well as to treat depression if present

### ***Non-drug management***

- Education, physiotherapy and related intervention remain at the centre of care.
- Education
- Education improve knowledge, symptom control, adherence and self management.
- Consider every consultation an opportunity to educate the patient.
- Provide information leaflets.
- Exercise
- Advise patient to keep active and exercise. This improves mood and encourage self-sufficiency. Advise the patient to pace activities to a realistic level. Explain the benefits of the different forms of exercise.
- range of movement or stretching exercise relieve stiffness and maintain flexibility
- strengthening exercise maintain muscle strength, need for function and joint support
- aerobic exercise improve cardiovascular risk, aids weight control and overall function
- Osteoporosis: Patients with RA are at an increased risk of osteoporosis. Calculate the patient's fracture risk.
- Management outline: see algorithm

### ***Follow up***

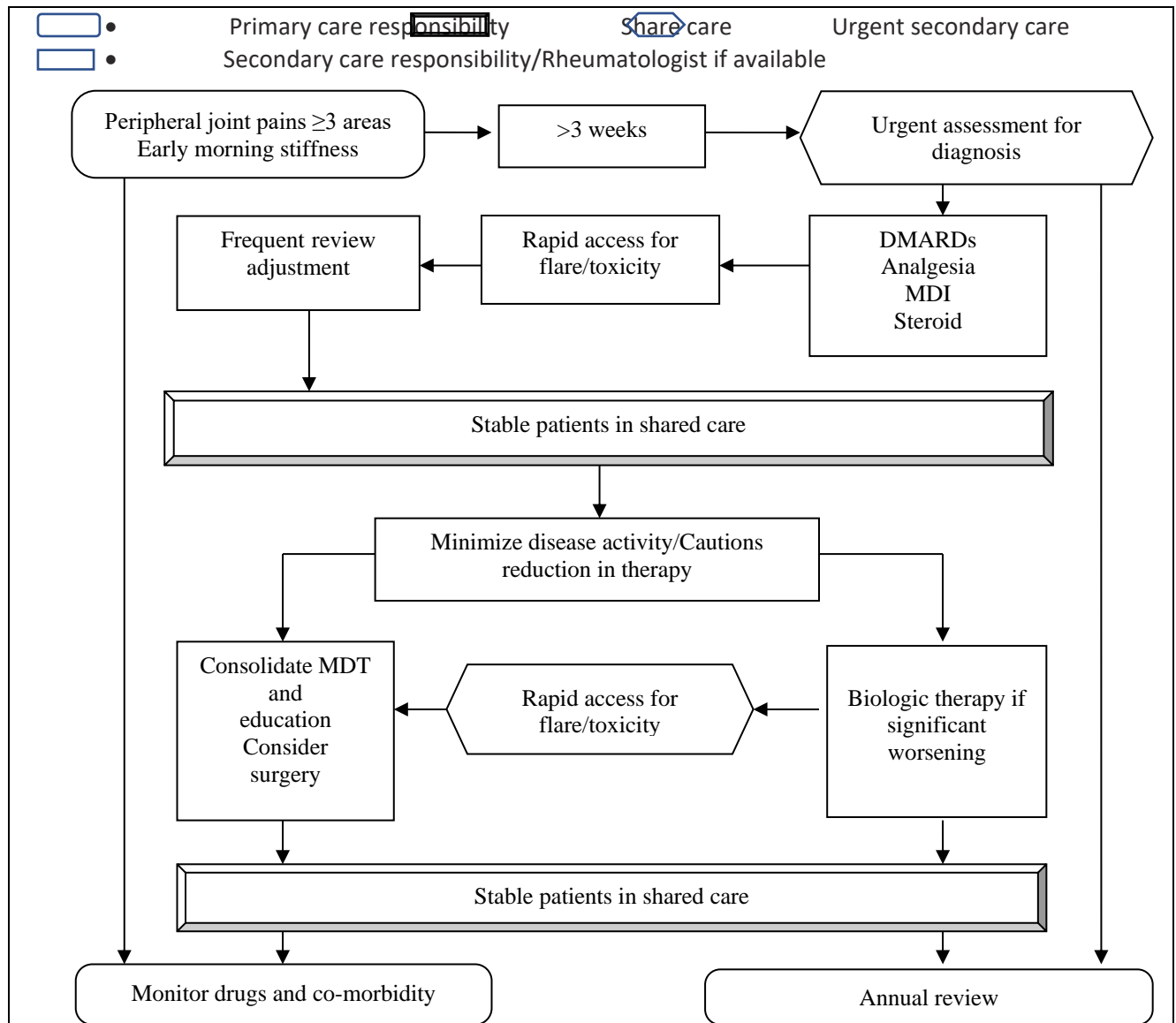
- Timing
- At week 4, week 12, week 24, week 36, week 52, then 24 weekly (6 monthly later)
- Monitor - Disease activity
- Functional Health Status
- Blood Test
- At week 4 At week 12
- At week 24 ATweek 36 At week 52
- FBC, ALT

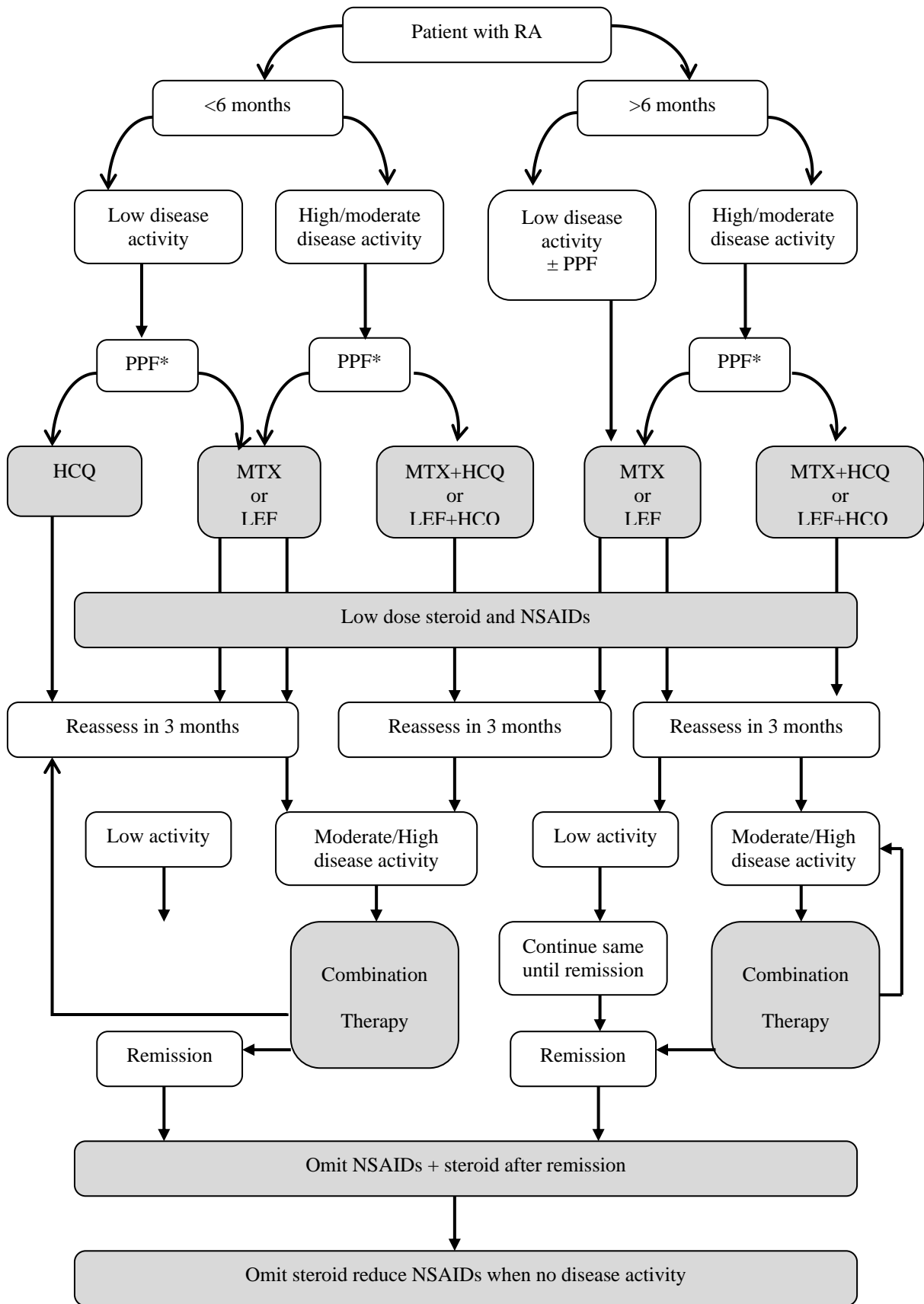
- FBC, LFT, ALT, CRP
- FBC, ALT, CRP, CXR, (PA) FBC, ALT
- FBC, LFT, ALT, CRP, Urea, Creatinine, Cholesterol, Sugar, CXR (PA) Bone mineral density (DEXA scan)

## Surgery

- Surgery can be highly effective in selected cases and covers tendon transfers, arthroplasties (including upper limb and small joints) and arthrodeses.

### MULTISECTORAL PLAN FOR MANAGEMENT OF RHEUMATOID ARTHRITIS





PPF\* = poor prognostic factors: Anti-CCP positive, RA (+), FHS III or IV  
 MTX=Methotrexate, HCQ=chloroquine, LEF=Leflunomide

• **Fig. Rheumatoid arthritis management algorithm**

## **Criteria for referral**

- All suspected cases of rheumatoid arthritis to rheumatology for early treatment with disease- modifying drugs which can significantly alter disease progression.
- Refer urgently if:
  - Small joints of the hands/feet are affected
  - > 1 joint is affected
- There has been a delay of 2:3 months between onset of symptoms and seeking medical advice:
  - Pregnancy
  - Fibrotic lung Disease
  - Liver disease
  - Vasculitis
- Moderate or High disease activity at 3 months follow up after combination of 2 DMARDs

## **References**

1. Alex Khat, Andrew Polmear: *Practical General Practice*
2. *Oxford handbook of General Practice, 4<sup>th</sup> Edition*
3. *Therapeutic Manual, Internal Medicine Society, MMA, 1<sup>st</sup> Edition, 2016*

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# SYSTEMIC LUPUS ERYTHEMATOSUS

## Definition

- SLE (lupus), which is the commonest of the connective tissue disorders
- It is a multisystem autoimmune disorder with a wide variety of clinical features that are due to vasculitis. Arthritis is the commonest feature of SLE (90% of cases). Milder manifestations outnumber more severe forms.

## Clinical features

- Mainly affects women in 'high oestrogen' period (90% of cases)
- Peak onset between 15 and 40 years
- Fever, malaise, tiredness common
- Multiple drug allergies e.g. sulfonamides

System	% of patients	Presenting complaints	
Joints	95	<ul style="list-style-type: none"> <li>• Arthritis</li> <li>• Arthralgia</li> </ul>	<ul style="list-style-type: none"> <li>• Myalgia</li> <li>• Tenosynovitis</li> </ul>
Skin	80	<ul style="list-style-type: none"> <li>• Photosensitivity</li> <li>• Facial 'butterfly' rash</li> <li>• Vasculitic rash</li> </ul>	<ul style="list-style-type: none"> <li>• Hair loss</li> <li>• Urticaria</li> <li>• Discoid lesions</li> </ul>
Lungs	50	<ul style="list-style-type: none"> <li>• Pleurisy</li> <li>• Pneumonitis</li> </ul>	<ul style="list-style-type: none"> <li>• Pleural effusion</li> <li>• Fibrosing alveolitis</li> </ul>
Kidney	50	<ul style="list-style-type: none"> <li>• Proteinuria</li> <li>• increased BP</li> </ul>	<ul style="list-style-type: none"> <li>• Glomerulonephritis</li> <li>• Renal failure</li> </ul>
Heart	40	<ul style="list-style-type: none"> <li>• Pericarditis</li> </ul>	<ul style="list-style-type: none"> <li>• Endocarditis</li> </ul>
CNS	15	<ul style="list-style-type: none"> <li>• Depression</li> <li>• Psychosis</li> <li>• Infarction</li> </ul>	<ul style="list-style-type: none"> <li>• Fits</li> <li>• Cranial nerve lesions</li> </ul>
Blood	95	<ul style="list-style-type: none"> <li>• Anaemia (very common)</li> <li>• Thrombocytopenia</li> </ul>	<ul style="list-style-type: none"> <li>• Splenomegaly</li> </ul>
Fatigue	95		

## Diagnostic criteria

- Polyarthritis + fatigue + skin lesions -7 SLE

## Classification criteria

- Systemic Lupus International Collaborating Clinics Classification (SLICC)
- The favourite differential diagnosis, SLE mimics other illnesses, with wide variety in symptoms that may come and go unpredictably. Diagnose SLE in an appropriate clinical setting if 4 criteria (at least 1 clinical and 1 laboratory) or biopsy-proven lupus nephritis with positive ANA or anti-DNA.

## Clinical criteria

- *Acute cutaneous lupus*: Malar rash/butterfly. Fixed erythema, flat or raised over the malar eminences, tending to spare the nasolabial folds. Occurs in up to 50%. Bullous lupus, toxic epidermal necrolysis variant of SLE, maculopapular lupus rash,

photosensitive lupus rash, or subacute cutaneous lupus (non-indurated psoriasiform and/or annular polycyclic lesions that resolve without scarring).

- *Chronic cutaneous lupus*: Discoid rash, erythematous raised patches with adherent keratotic scales and follicular plugging ± atrophic scarring. Think of it as a three-stage rash affecting ears, cheeks, scalp, forehead, and chest: erythema -7 pigmented hyperkeratotic oedematous papules -7 atrophic depressed lesions.
- *Non scarring alopecia*: (In the absence of other causes.)
- *Oral/nasal ulcer*: (In the absence of other causes)
- *Synovitis*: (Involving two or more joints or two or more tender joints with >30 minute of morning stiffness.)
- *Serositis*: a) Lung (pleurisy for > 1 day, or pleural effusions, or pleural rub; b) pericardial pain for > 1 day, or pericardial effusion, or pericardial rub, or pericarditis on ECG.
- *Urinalysis*: Presence of proteinuria (>0.5 g/d) or red cell casts.
- *Neurological features*: Seizures; psychosis; mononeuritis multiplex; myelitis; peripheral or cranial neuropathy; cerebritis/acute confusional state in absence of other causes.
- 9. Haemolytic anaemia.
- *Leucopenia*: (WBC <4.) At least once or lymphopenia (lymphocytes < 1) at least once.
- *Thrombocytopenia*: (Platelets < 100.) At least once.
- Laboratory criteria
- positive ANA (positive in >95%)
- Anti-double stranded DNA (Anti-dsDNA)
- Anti-Smith antibodies present
- Antiphospholipid antibodies present
- Low complement (C3, C4 or C50)
- positive Direct Coombs test
- Adapted from 'Derivation and validation of the Systemic Lupus International Collaboration Clinics classification criteria for systemic lupus erythematosus'. Petri M et al., *Arthritis and Rheumatism*, vol. 64, Issue 8 (2012) 2677-2686.

## Diagnosis

- ESR/CRP - elevated in proportion to disease activity
- ANA test- positive in 95% (perform first) (key test)
- dsDNA antibodies- 90% specific for SLE but present in only 60% (key test)
- ENA antibodies, especially Sm - highly specific
- Rheumatoid factor- positive in 50%
- LE cell test - inefficient and not used
- The diagnosis cannot be made on blood tests alone. Supportive clinical evidence is necessary. For suspected SLE, the recommended approach is to perform an ANA test. If positive, order dsDNA and ENA antibodies.



- [https:// i1.yt.com/ www.mvli\(eria .com/ wp-content / uploads/20 1 7/ 02/ tcara-merawat-sle-anda-tanda- sle.jpg ?resize=300 %2C215](https://i1.yt.com/www.mvli(eria.com/wp-content/uploads/2017/02/tcara-merawat-sle-anda-tanda-sle.jpg?resize=300%2C215)

## **Management**

- Refer to rheumatologist.
- Appropriate explanation, support and reassurance, use of sunscreens

## **Treatment for sunburn**

- Hydrocortisone 1% ointment or cream for severe sunburn on face, early.
- Use hydrocortisone 1% or 0.02% betamethasone valerate for other areas.
- Repeat in 2-3 hours and then the next day.
- Hydrocortisone is not useful after 24 hours and should be used for unblistered erythematous skin, not on broken skin.
- Oral aspirin eases pain. Oil in water baths or bicarbonate of soda paste may help and wet applications such as oily calamine lotions or simply cool compresses may give relief

## **Prevention of sunburn**

- Avoid direct exposure to summer sunlight during peak UV periods (10 am to 3 pm).
- Use natural shade- beware of reflected light from sand or water and light cloud. Use a sunscreen with a minimum of SPF 30.
- Wear broad-brimmed hats and protective clothing.
- Refer to consultant rheumatologist for shared care in a multidisciplinary team

## **Treatment**

- Based on severity and organ involved Mild (Facial rash + Arthritis)
- PO Hydroxychloroquine (HCQ) 200 mg odor Chloroquine (CQ) 150 mg od
- PO Prednisolone 5 mg od
- Moderate (Truncal rash)
- PO Hydroxychloroquine 200 mg odor Chloroquine 150 mg or liz tablet od
- PO leflunamide/methotrexate

- PO Prednisolone 5-10 mg or methyl prednisolone 4-8 mg em Treatment of organ threatening (Severe) SLE
- REFER to hospital Vaccination
- Pneumococcal vaccination stat. and 5 yrly
- Influenza vaccination stat. and yearly
- Hepatitis B vaccine stat and followed by at 1 & 6 month Family Planning
- Ask for patient's family planning and advice rightfully
- 3 month depo injection to all reproductive age patient who have potential to become pregnant
- cvs
- Check cardiovascular risk factor and correct accordingly Follow up
- Check blood for CP, ESR, ALT, Potassium, Creatinine, RBS, Urine RE monthly
- Check lipid profile 6 monthly
- Check Bone Mineral Density(BMD) after 6 month of steroid therapy
- Yearly visual field examination to detect CQ and HCQ related changes SLE and Pregnancy
- Check Anti-Phospholipid Antibodies 12 weeks apart
- Choose HCQ or Azathioprine ifDMARD is needed.
- Steroid and aspirin are safe
- Use aspirin and low molecular weight heparin (LMWH) for anti-phospholipid antibodies positive.

## References

1. *John Mutagh 's General Practice, 6th Edition*
2. *Oxford handbook of General Practice 4th Edition*
3. *Therapeutic Manual (Internal Medicine) 1<sup>st</sup> Edition*

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## ACR mnemonic of SLE diagnostic criteria

- The following are the ACR diagnostic criteria in SLE, presented in the "**SOAP BRAIN MD**" mnemonic:
- **S**erositis - Pleurisy, pericarditis on examination or diagnostic electrocardiogram (ECG) or imaging
- **O**ral ulcers - Oral or nasopharyngeal, usually painless; palate is most specific
- **A**rthritis - Nonerosive, 2 or more peripheral joints with tenderness or swelling
- **P**hotosensitivity - Unusual skin reaction to light exposure
- **B**lood disorders- Leukopenia( $< 4 \times 10^3$  cells/11L on  $>1$  occasion), lymphopenia( $< 1500$  cells/ 11L on  $>1$  occasion), thrombocytopenia( $< 100 \times 10^3$  cells/11L in the absence of offending medications), hemolytic anemia
- **R**enal involvement- Based on presence of proteinuria ( $>0.5$  g/day or 3+ positive on dipstick testing) or cellular casts (including red blood cells [RBCs], hemoglobin, granular, tubular, or mixed) or based on the opinion of a rheumatologist or nephrologist
- **A**ntinuclear antibodies (ANAs)- Higher titers generally more specific ( $> 1:160$ ); must be in the absence of medications associated with drug-induced lupus
- **I**mmunologic phenomena- dsDNA; anti-Smith (Sm) antibodies; antiphospholipid antibodies (anticardiolipin immunoglobulin G [IgG] or immunoglobulin M [IgM] or lupus anticoagulant); biologic false-positive serologic test results for syphilis, lupus erythematosus (LE) cells (omitted in 1997 revised criteria)
- **N**eurologic disorder - Seizures or psychosis in the absence of other causes
- **M**alar rash - Fixed erythema over the cheeks and nasal bridge, flat or raised
- **D**isoid rash- Erythematous raised-rimmed lesions with keratotic scaling and follicular plugging, often scarring
- <https://emedicine.medscape.com/article/332244-workup>
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