GUIDELINES

For

GENERAL PRACTITIONERS

2024

Press record

First Edition

Printed by SARANA PRESS (Dr. Aung Kyaw Min)

249, Theinbyu Road, Mingalartaungnyunt Township, Yangon, Myanmar

2018

Cover Designer (Tun Zaw)

Inner Designer (Tun Zaw)

Second Edition

Digital Copy Printing (TMO)

249, Theinbyu Road, Mingalartaungnyunt Township, Yangon, Myanmar.

2024 April

Cover Designer (Tun Zaw & Win Zaw)

Inner Designer (TMO)

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recommendations for clinicians who are uncertain about how to proceed, overturn the beliefs of doctors accustomed to outdated practices, improve the consistency of care, and provide authoritative recommendations that reassure practitioners about the appropriateness of their treatment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 April, 2024
General Practitioners' Society (Central)
Myanmar Medical Association

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 immerse 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**

LIST OF CONTRIBUTORS

1. *Aung Cho Myint, Prof*
2. *Aung Maw, Dr*
3. *Aye Aung, Prof*
4. *Aye Aye Than, Dr*
5. *Aye Aye Thein, Dr*
6. *Chit Soe, Prof*
7. *Hla Myat Nwe, Prof*
8. *Hla Myint Tun, Dr*
9. *Hlaing Mya Win, Prof*
10. *Hlaing Myint, Dr*
11. *Htay Win, Dr*
12. *Htin Aung Saw, Prof*
13. *Htun Lwin Nyein, Prof*
14. *Khin Hla Hla, Prof*
15. *Khin Hta Yi, Prof*
16. *Khin Mi Mi, Dr*
17. *Khin Ohnmar Khine, Prof*
18. *Khin Saw Than, Prof*
19. *Khine Khine Zaw, Prof*
20. *Khine Soe Win, Dr*
21. *Ko Ko, Prof*
22. *Kyaw Myint Naing, Prof*
23. *Kyaw Thu, Dr*
24. *Kyaw Zin Wai, Prof*
25. *Kyi Kyi Nyunt, Prof*
26. *Kyi Kyi Thinn, Prof*
27. *Kyin Htwe, Dr*
28. *Lin Htet, Dr*
29. *Lwin May Oo, Dr*
30. *Mar Mar Kyi, Prof*
31. *Maung Maung Sein, Prof*
32. *May Thandar Oo, Dr*
33. *Min Han, Prof*
34. *Min Yazar, Dr*
35. *Min Zaw Oo, Prof*
36. *Moe Naing, Dr*
37. *Moe Wint Aung, Prof*
38. *Mya Thae Han, Dr*
39. *Mya Win Hnit, Dr*
40. *Myint Thaung, Prof*
41. *Myo Khine, Dr*
42. *Myo Lwin Nyein, Prof*
43. *Myo Nyunt Aung, Dr*
44. *Myo Oo, Prof*
45. *Naing Oo, Prof*
46. *Nang Phyu Phyu Aung, Prof*
47. *Nwe Mar Tun, Prof*
48. *Nwe Nwe Aung, Dr*
49. *Nyein Moe Thaw, Dr*
50. *Phyu Phyu Khaing, Dr*
51. *Rai Mra, Prof*
52. *Samuel Kyaw Hla, Prof*
53. *Saw Win, Prof*
54. *Sein Way Lwin, Dr*
55. *Than Htike, Dr*
56. *Than Than Aung, A Prof*
57. *Than Than Aye, Prof*
58. *Thar Thar Oo, Dr*
59. *Thein Aung, Prof*
60. *Thein Myint, Prof*
61. *Thet Naing Maung, Dr*
62. *Thin Thin Nwe, Dr*
63. *Tin Aye, Dr*
64. *Tin Nyunt, Dr*
65. *Tin Tin Aye, Dr*
66. *Tin Tin Hla, Dr*
67. *Tint Tint Kyi, Prof*
68. *Vijay Kumar, Dr*
69. *Win Lwin Thein, Dr*
70. *Win Zaw, Dr*
71. *Yin Yin Soe, Prof*
72. *Yin Yin Zaw, Prof*
73. *Yu Yu Lwin, Dr*
74. *Zaw Lynn Aung, Prof*

SYMBOLS AND ABBREVIATIONS

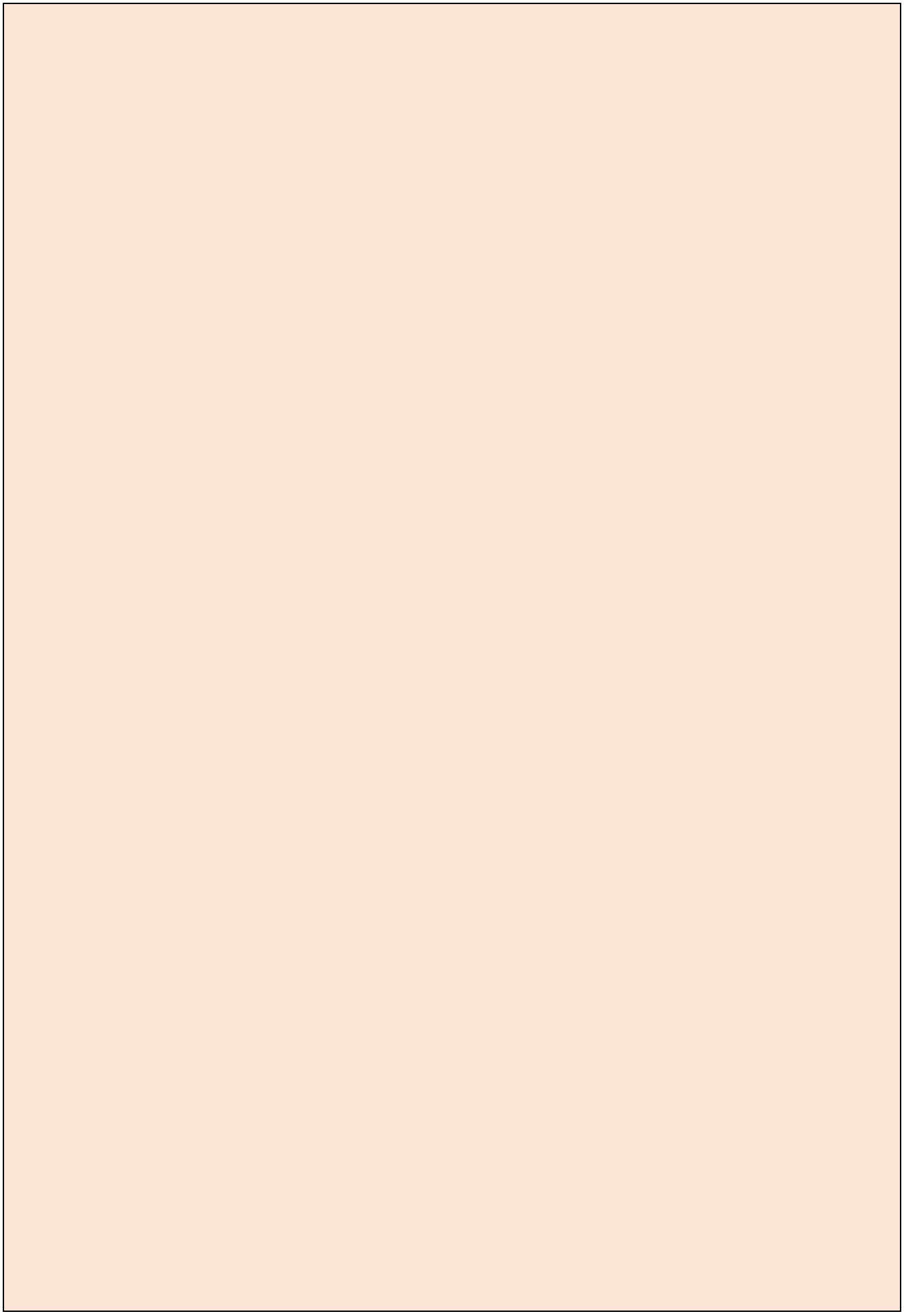
AAA abdominal aortic aneurysm
ABC airway, breathing, circulation
ABCD airway, breathing, circulation, dextrose
ABO A, B and O blood groups
ACE angiotensin-converting enzyme
ACEI angiotensin-converting enzyme inhibitor
ACTH adrenocorticotrophic hormone
ADHD attention deficit hyperactivity disorder
ADT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

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 toxaemia
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 haemoglutination 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

CHAPTER (8) RENAL PROBLEMS

1. Acute Kidney Injury/Acute Renal Failure
2. Chronic Kidney Disease (CKD)
3. Urinary Tract Infections
4. Renal stones or Urolithiasis
5. Bladder stones
6. Benign Hyperplasia of Prostate Glands (BPH)



ACUTE KIDNEY INJURY / ACUTE RENAL FAILURE

Definition

Acute kidney injury is defined as the sudden loss of kidney function over hours to days resulting in the inability to maintain electrolyte, acid-base, and water balance. Or Acute kidney injury (AKI) is defined as any of the following:

- Increase in Serum Creatinine (SCr) by ≥0.3 mg/dl (≥26.5 µmol/1) within 48 hours; or
- Increase in SCr to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
- Urine volume <0.5 ml/kg/h for 6 hours.

Symptoms

- feeling sick or being sick
- diarrhoea
- dehydration
- peeing less than usual
- confusion
- drowsiness

Risk factors for Acute Renal Injury

Nonmodifiable	Modifiable
AIDS	Anaemia
Chronic kidney disease	Hypercholesterolemia
Chronic liver disease	Hypertension
Chronic heart failure	Hypoalbuminemia
Diabetes Mellitus	Hyponatremia
Older age (>65 years)	Mechanical ventilation
Peripheral vascular diseases	Nephrotoxic drug use
Prior kidney surgery	Rhabdomyolysis
Renal artery stenosis	Sepsis

Stages of Acute kidney injury

Stage	Urine output	Serum creatinine
1	<0.5 ml/kg/h for 6-12 hours	1.5 - 1.9 times baseline Or ≥ 0.3 mg/dl (≥ 26.5 µmol/1) increase
2	<0.5 ml/kg/h for ≥12 hours	2.0 - 2.9 times baseline
3	<0.3 ml/kg/h for ≥ 24 hours Or Anuria for ≥ 12 hours	3.0 times baseline Or Increase in serum creatinine to ≥ 4.0 mg/dl (≥ 353.6

		µmol/1) Or Initiation of renal replacement therapy Or In patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m^2

Causes

Commonest causes:

- Sepsis
- Major surgery
- Cardiogenic shock
- Other hypovolaemia
- Drugs
- Hepatorenal syndrome
- Obstruction
- It is essential to identify treatable cause to prevent further renal deterioration.

Causes of renal failure

	Pathology	**Examples**
Pre-renal	Decreased vascular volume	Haemorrhage, Diarrhoea &Vomiting, burns, pancreatitis,
	Decreased Cardiac	Cardiogenic shock, MI
	Systemic vasodilation	Sepsis, drugs
	Renal vasoconstriction	NSAIDs, ACEI, ARB, hepatorenal syndrome
Renal	Glomerular	Glomerulonephritis, Acute Tubular Necrosis (ATN) (prolonged renal hypoperfusion causing intrinsic renal damage)
	Interstitial	Drug reaction, infection, infiltration (e.g. sarcoid)
	Vessels	Vasculitis, Haemolytic uremic syndrome (HUS), Thrombotic thrombocytopenic purpura (TTP), Disseminated intravascular coagulation (DIC)
Post-renal	Within renal tract	Stone, renal tract malignancy, stricture, clot
	Extrinsic compression	Pelvic malignancy, prostatic hypertrophy, retroperitoneal fibrosis

For Early Diagnosis

Think of and investigate for acute kidney injury in patients with acute illness, if any of the following are present:

- chronic kidney disease
- history of acute kidney injury
- symptoms or history of urological obstruction, or conditions that may lead to obstruction
- symptoms or signs of nephritis (such as oedema or haematuria)
- oliguria (urine output less than 0.5 ml/kg/hour)
- those with limited access to fluids (e.g. young age, neurological impairment or disability)
- hypovolaemia
- hypotension
- sepsis
- severe diarrhoea (children and young people with bloody diarrhoea are at particular risk)
- heart failure
- liver disease
- haematological malignancy
- use of drugs with nephrotoxic potential (such as NSAIDs, aminoglycosides, ACE inhibitors, ARBs and diuretics) within the past week, especially if hypovolaemic

Consider critical care referral

RAPID ASSESSMENT AND TREATMENT OF LIFE-THREATENING EMERGENCY SITUATIONS

Physiological parameters	Score 3	2	1	0	1	2	3
Respiration rate	≤8		9-11	12-20		21-24	≥ 25
Oxygen Saturations	≤91	92-93	94-95	≥96			
Any supplemental oxygen		Yes		No			
Temperature	≤35.0		35.1-36.0	36.1-38.0	38.1-39.0	≥39.1	
Systolic BP	≤90	91-100	101-110	111-219			≥220
Heart rate	≤40		41-50	51-90	91-110	111-130	≥130
Level of consciousness				A			V.P or U

The NEWS initiative flowed from the Royal College of Physicians' NEWSDJG, and was jointly developed and funded in collaboration with the Royal College of Physicians, Royal College of Nursing, National Outreach Forum and NHS Training for Innovation.

- Early warning scores are tools to aid assessment. They do not replace clinical judgment: use yours and respect the clinical opinion of others.
- Refer to local early warning scores where available.
- Hyperkalaemia (ECG showing tall T especially with widening QRS complex)
- Fluid overload l pulmonary oedema
- Hypotension or shock
- Urinary tract obstruction, especially in patient with anuria; but always consider it although there is urine output. Urgent US is mandatory. It is surgical emergency.

Investigation

- The history and physical examination are important in determining the etiology of acute kidney injury.
- The physical examination should focus on evaluating intravascular volume status.
- Skin rashes may indicate an underlying condition (e.g., systemic lupus erythematosus, atheroembolism/vasculitis) or exposure (e.g., drug rash suggesting acute interstitial necrosis) leading to acute kidney injury.
    - Urgent:
    - Urine REME (routine examination and microscopic examination),
    - Blood urea, creatinine and creatinine clearance, electrolytes
    - ECG
    - USG abdomen

Treatment

Treatment of AKI depends on what's causing your illness and how severe it is.
The patient may need:

- to increase your intake of water and other fluids if you're dehydrated
- antibiotics if you have an infection
- to stop taking certain medicines (at least until the problem is sorted)
- a urinary catheter, a thin tube used to drain the bladder if there's a blockage
- treat acute pulmonary oedema as in other causes
- treat hyperkalemia
- treat metabolic acidosis
- nutrition
- A total energy intake of 20-30 kcal/kg/d should be given in patients with any stage of AKI.
- Protein 0.5 g/kg/day. Avoid potassium rich food and drug

may need to go to hospital for some treatments.

Prevention

- Nephrotoxic medications like aminoglycosides, NSAID including COX2 inhibitors and ACEI or ARB should not be used if possible in patients at risk for AKI or with AKI.
- To keep the patient normal hydration as a whole.
- In the absence of hemorrhagic shock, isotonic crystalloids rather than colloids (albumin or starches) should be used as initial management for expansion of intravascular volume in patients at risk for AKI or with AKI.

Referral criteria

- National early warning score for adult patient >1
- Anuria for more than12 hours
- A lack of a clear precipitating cause of AKI.
- Clinical suspicion for a diagnosis that may require specialist treatment (for example vasculitis, myeloma, glomerulonephritis, interstitial nephritis)
- Chronic kidney disease stage 4 or 5
- AKI requiring Renal Replacement Therapy

Refer urologist when one or more of the following is present:

- pyonephrosis
- an obstructed solitary kidney
- bilateral upper urinary tract obstruction with acute kidney injury.

Indications for Dialysis (Peritoneal Dialysis, Haemodialysis, Haemofiltration)

- Refractory hyperkalaemia:Potassium >7 (persistently >6) (check ECG to exclude any error during sampling)
- Uncontrolled fluid overload or pulmonary oedema
- Severe acidosis: pH <7.15, HC03 <15 mmol/1
- Uraemic complications such as pericarditis and encephalopathy

Reference

1. *Oxford handbook of Clinical Medicine, 10th Edition*
2. *https://www.google.com/search?q=acute+renal+failure+aafp&oq=acute+renal+failure+&aqs=chrome.1.69i59l3j0i512l7.8679j0j7&sourceid=chrome&ie=UTF-8*
3. *https://www.nhs.uk/conditions/acute-kidney-injury/*

CHRONIC KIDNEY DISEASE (CKD)

DEFINITION

- CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health.

***Criteria for CKD** (either of the following present for >3 months)*	
Markers of kidney damage (one or more)	• Albuminuria (AER ≥30 mg/24 hours; ACR ≥30 mg/g) • Urine sediment abnormalities • Electrolyte & other abnormalities due to tubular disorders • Abnormalities detected by histology • Structural abnormalities detected by imaging • History of kidney transplantation
Decreased GFR	• <GFR 60ml/min/1.73m^2 (GFR categories G3a-G5)

Abbreviations: AER- albumin excretion rate; A CR- albumin creatinine ratio; GFR- glomerular filtration rate.

STAGING OF CKD

Stage	GFR (ml/min/1.73 m^2)	Terms
1	≥ 90	Normal or high
2	60 -89	Mildly decreased
3a	45 – 59	Mildly to Moderately decreased
3b	30 – 44	Moderately to severely decreased
4	15 – 29	Severely decreased
5	<15	Kidney failure

Symptoms	*Signs*
• No symptoms in early stage • At a more advanced stage, symptoms can include: • tiredness • swollen ankles, feet or hands • shortness of breath • feeling sick • blood in urine • Weight loss, poor appetite, difficult in sleeping, headache • Nausea, anorexia, lethargy, itch, nocturia, impotence. muscle cramps • Later: oedema, dyspnoea, chest pain (from pericarditis), vomiting, confusion, fits, hiccups, neuropathy, coma	• pallor, • lemon tinge to skin, • pulmonary/peripheral oedema, • pericarditis,pleural effusion, • metabolic flap, • Hypertension, • retinopathy

Markers of kidney damage

- Proteinuria
- Albuminuria
- Presence of hematuria, cellular casts, chronic pyuria, tubular concentrating defects, insufficient renal acidification

Investigations

Blood: Hb (normochromic normocytic anemia), ESR, U&E, glucose, Ca, P04, alkaline phosphate, PTH,
Urine: Dipstick, MC&S, albumin, creatinine ration or protein : creatinine ration
Imaging: In CKD, kidneys are usually small (<9cm), but enlarged in infiltrative disorders (amyloid, myeloma), APKD and DM. If asymmetrical, consider renogram
Histology: consider renal biopsy if unclear cause or rapidly progressive or normal sized kidneys

Assessment of Patient at risk of CKD

(algorithm see next page)

Retarding progression to ESRD

Antihypertensive therapy

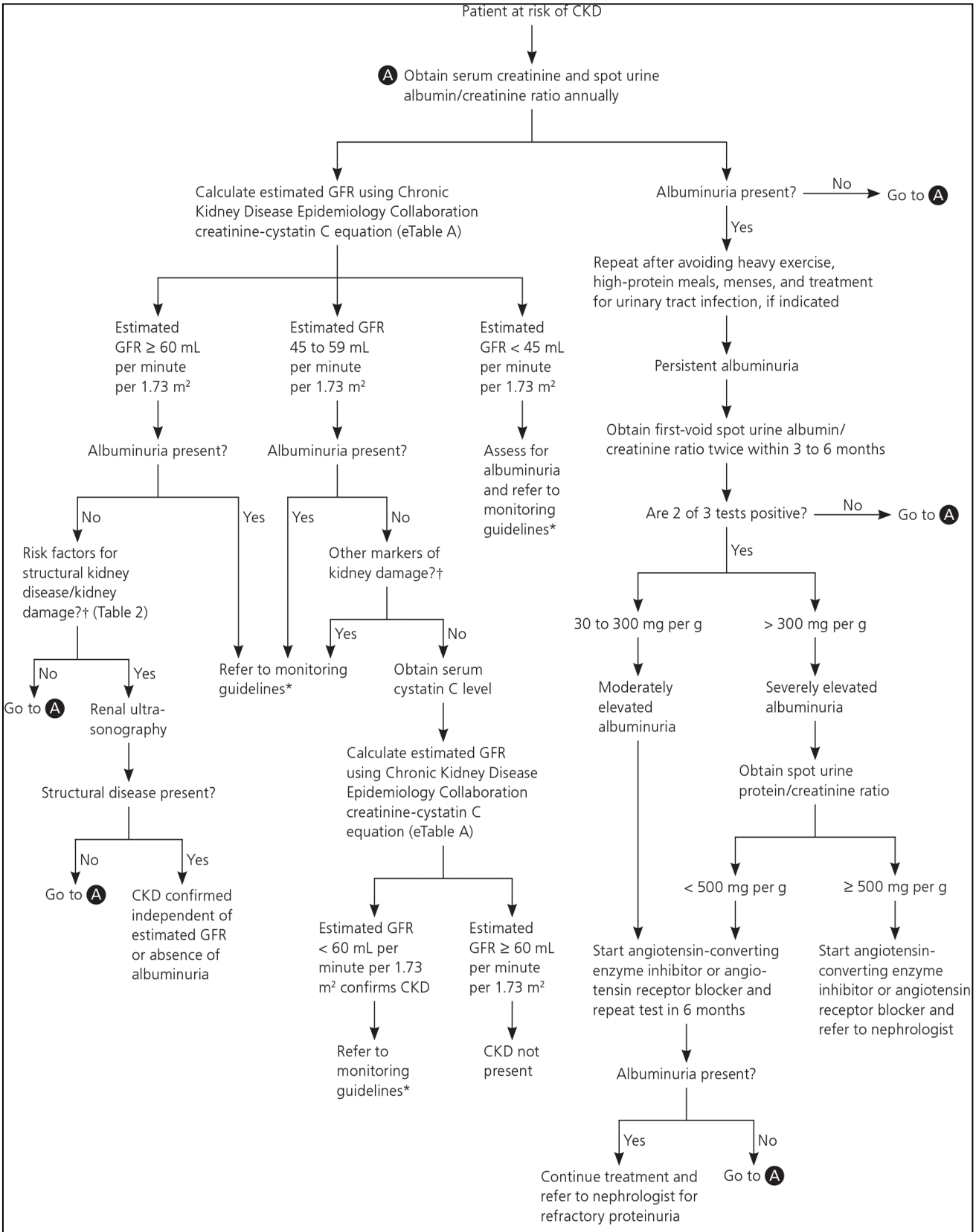
- Hypertension is an important risk factor for progression of kidney disease and is a common cause of ESRD.
- Recommended target BP is ≥ 130/80 mmHg in CKD.
- Choice of antihypertensives may depend on the presence or absence of co- morbidities:
- ARB or ACEI is recommended to be used in both diabetics and non-diabetics with CKD
- In addition, the following drugs can be used alone or in combination:
- Calcium channel blocker (preferably non dihydropyridine).
- Beta blocker (preferably vasodilating group).
- Centrally acting agent (methyl dopa).
- Alpha blocker (prazosin/ doxazosin).
- Diuretics: low dose spironolactone (be careful of hyperkalaemia), thiazides (chlorthalidone, indapamide), loop diuretics (particularly in CKD 4-5).

Control of diabetes mellitus (DM)

- Diabetic nephropathy is the most common cause of ESRD in the developed world, and its incidence is rising in Myanmar.
- Tight glycaemic control may minimize the rate of decline of renal function and a target HbAl c 7.0% is recommended.

Renoprotection with ACEI/ARB

- Use of ACEI or ARB should be reviewed or discontinued if serum creatinine >265 umol/L or a 20% rise from baseline levels or development of hyperkalemia.



*—Monitoring guidelines are available in the 2012 Kidney Disease: Improving Global Outcomes recommendations (see Guide to Frequency of Monitoring [number of times per year] by GFR and Albuminuria Category in chapter 2.1.4 at http://www.kdigo.org/clinical_practice_guidelines/pdf/CKD/KDIGO_2012_CKD_GL.pdf).

†—Markers of kidney damage include, but are not limited to: structural renal disease (i.e., atrophic kidneys, thin [< 1 cm] renal cortices, hyperechoic kidneys on ultrasonography), hematuria (microscopic or otherwise), presence of cellular casts, chronic pyuria, tubular concentrating defects, and insufficient renal acidification.

Dietary protein restriction

- It is suggested to avoid high protein intake (>1.3 g/kg/d) in adults with CKD at risk of progression.
- Protein intake of 0.8 g/kg/d is recommended in CKD stage 4-5, but a protein intake of 1.2-1.3 g/kg/d is recommended for patients on regular HD (due to risk of malnutrition).

Salt intake

- Salt intake is recommended to be <2 g/d of sodium or <5 g/d of sodium chloride in adults, unless contraindicated.

Lifestyle

- Be encouraged to undergo physical activity compatible with cardiovascular health and tolerance (30 minutes x 5 times/week), achieve a healthy weight (BMI 20-25), and stop smoking.
- Diet -potassium restriction if hyperkalemic, avoidance of high phosphate foods (milk, cheese, eggs)
- Cardiovascular modification -in CKD 1 and 2, risk from cardiovascular death is higher. Give statins to CKD with raised lipid, give aspirin

Managing complications of CKD

Anaemia

- Treatment of renal anaemia should start when Hb <10.0 g/dl, not to intentionally increase >13.0 g/dl
- Check haematinics and replace iron, B12 and folate if necessary. If still anaemic, consider recombinant human erythropoietin, (Erythropoietin stimulating agents (Inj. Erythropoietin /EPO) can be used to maintain the Hb concentration effectively, and the recommended starting dose is 50-150 IU/kg/week (4000-8000 IU/week), and SC Erythropoietin dose is 15-30% lower than iv dose.)
- If Hb falls despite this, and no infection, hemolysis or blood loss, suspect red cell aplasia. Stop at once and get help from haematology.

Hyperphosphataemia

- Dietary phosphate restnct10n of ≤ 30-40 mmol/d (avoid dairy products, nuts, chocolate & tinned fish).
- Phosphate binders taken a few minutes before a meal, and calcium containing binders are more appropriate than aluminium hydroxide. Non-calcium, non- aluminium containing binders (sevelamer) and lanthanum carbonate are safer but expensive.
- Give vitamin D supplement (e.g. Colecalciferol, ergocalciferol) if deficient.
- If increased PTH persist or is increasing, treat with an activated vitamin D analogue (e.g. l alpha calcidol or calcitriol).

Metabolic acidosis

- Acidosis is common in all forms of CKD and can worsen uraemic osteodystrophy and protein malnutrition.
- Early treatment with oral sodium bicarbonate (650-1300 mg bd/ tds) may prevent this and a serum bicarbonate level of <20 mEq/L is recommended.

Oedema

- High dose of loop diuretics may be needed (furosemide 250 mg - 2 g/24hr with or without metalazone 5-10 mg/24hr each morning) and restriction of fluid and salt intake.

Restless legs/cramps

- Check ferritin (low level worsen symptoms) clonazepam or gabapentin, pregabalin or quinine sulphate may help.

Nephrologist referral

Delayed referral to a nephrologist is associated with increased morbidity& mortality.
Consider if

- GFR <30 ml/min/1. 73m2 (CKD stage 4-5)
- develop AKI or abrupt sustained fall in GFR.
- Significant albuminuria (ACR >70 mg/mmol)
- Persistent microscopic haematuria and <50 year (to urologist if >50)
- functional consequences of CKD (anaemia -Hb <11 g/dl, bone disease
- hypertension refractory to treatment with antihypertensive agents (>140/90 on 4 agents)
- known or suspected rare or genetic causes of CKD
- Suspected renal artery stenosis

Renal replacement therapy (RRT)

Choice of RRT

End Stage Renal Disease (ESRD or CKD stage 5) reflects progression of CKD to a point where RRT may be required to maintain life (fatal & irreversible)
There are a variety of options for RRT to choose:

- Continuous ambulatory peritoneal dialysis (CAPD)
- Automated peritoneal dialysis (APD)
- Haemodialysis (HD)*
- Haemodiafiltration (HDF)
- Renal transplantation*

(* = these are the only options available in Myanmar, but CAPD may develop later)

Haemodialysis (HD)

Indications for HD include

- Acute on chronic conditions of kidneys such as
- metabolic acidosis (HCO3 <10 mmol/L)
- electrolyte imbalance unresponsive to conservative treatment.
- fluid overload not responding to diuretics.
- creatinine clearance <5-10 ml/min/1.73 m^2
- uraemic signs & symptoms not responding to conservative treatment (serum creatinine >8-10 mg%)

Renal transplantation

- Renal transplantation can be done using cadaveric, live related & live unrelated donors (only live related transplantation is possible in Myanmar).
- All patients with ESRD should be considered for renal transplantation as it offers a better life expectancy and quality of life than dialysis.

Absolute contraindications are:

1. Uncontrolled cancer (especially with metastasis).
2. Active systemic infections.
3. Any condition with a life expectancy <2 years.

Reference

1. *Oxford handbook of Clinical Medicine, 10th Edition*
2. *https://www.aafp.org/pubs/afp/issues/2017/1215/p776.html*
3. *https://www.nhs.uk/conditions/kidney-disease/diagnosis/*

URINARY TRACT INFECTIONS

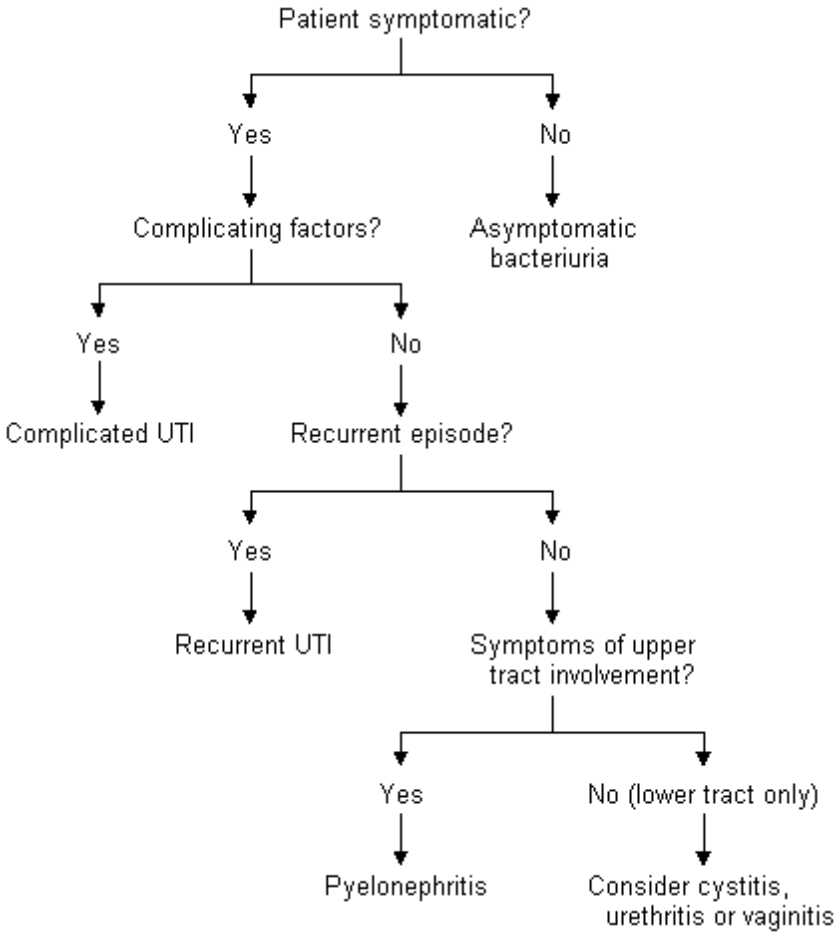
Definition

The presence of a pure growth of $>10^5$ organisms per ml of fresh MSU.
Lower UTI: urethra (urethritis), bladder (cystitis), prostate (prostatitis)
Upper UTI: renal pelvis (pyelonephritis)

Presentations of UTI

Cystitis: frequency, dysuria, urgency, strangury, low abdominal pain, incontinence of urine, acute retention of urine, cloudy or offensive urine and /or haematuria
Pyelonephritis: loin pain, fever, rigors, malaise, vomiting and/or haematuria
Organisms involved: E. coli, Proteus spp, Pseudomonas spp, Streptococci, Staphylococci, Chlamydia in young people
Risk factors: Prior infection, DM, Stones, pregnancy, dehydration, GU instrumentation, catheterization, menopause, sexual intercourse, GU malformation, Urinary stasis/ obstruction, Delayed micturition

Initial investigation



- If symptoms are present, test urine with leucocyte and nitrite dipstick, if positive, treat empirically, if negative with symptomatic, send MSU for MC&S to confirm (take MSU prior to starting antibiotic)

Causes of urinary tract infections (UTIs)

UTIs are common infections that happen when bacteria, often from the skin or rectum, enter the urethra, and infect the urinary tract.
The infections can affect several parts of the urinary tract, but the most common type is a bladder infection (cystitis). Women have a shorter urethra than men.
This means bacteria are more likely to reach the bladder or kidneys and cause an infection.
the risk of bacteria getting into the **bladder** include:

- having sex
- pregnancy
- conditions that block the urinary tract – such as kidney stones
- conditions that make it difficult to fully empty the bladder – such as an enlarged prostate in men and constipation in children
- urinary catheters (a tube in bladder used to drain urine)
- having a weakened immune system – for example, people with diabetes or people having chemotherapy
- not drinking enough fluids
- not keeping the genital area clean and dry

Reason to send MSU for MC&S

- Unresolved infection after antibiotics
- Recurrent UTI
- Uncatheterized man with UTI
- Catheterized man or woman with symptomatic UTI
- Child
- Pregnant woman
- Suspected pyelonephritis
- Haematuria

A pure growth of $>\mathbf{10^5}$ organisms/ml is diagnostic.
If $<10^5$ organisms/ml and pyuria (>20 WBC/mm3), treat if symptomatic.

Further investigation

- FBC, CRP, and blood culture if systematic unwell,FBS,
- Blood test (U&E, Cr, eGFR, and/or PSA if >40 years old and male) and/or radiology (renal tract USS, KUB) if
- UTI in man, child,
- Recurrent UTI in women

PYELONEPHRITIS

- Unclear diagnosis (persisting symptoms but negative MSU)
- Unusual infecting organism
- Sterile pyuria

Management

SYMPTOM MANAGEMENT

- Increase fluid intake (>3L/24hr)
- Alkalinize urine (potassium citrate solution)

Table – Urinary Tract Infection in adults

Category	Diagnostic criteria	Principal pathogens	First-line therapy	Comments
Acute uncomplicated cystitis	Urinalysis for pyuria and hematuria (culture not required)	*Escherichia coli* *Staphylococcus saprophyticus* *Proteus mirabilis* *Klebsiella pneumoniae*	TMP-SMX DS (Bactrim, Septra) Trimethoprim (Proloprim) Ciprofloxacin (Cipro) Ofloxacin (Floxin)	Three-day course is best Quinolones may be used in areas of TMP-SMX resistance or in patients who cannot tolerate TMP-SMX
Recurrent cystitis in young women	Symptoms and a urine culture with a bacterial count of more than100 CFU per mL of urine	Same as for acute uncomplicated cystitis	If the patient has more than three cystitis episodes per year, treat prophylactically with postcoital, patient-directed* or continuous daily therapy (see text)	Repeat therapy for seven to10 days based on culture results and then use prophylactic therapy
Acute cystitis in young men	Urine culture with a bacterial count of 1,000 to 10,000 CFU per mL of urine	Same as for acute uncomplicated cystitis	Same as for acute uncomplicated cystitis	Treat for seven to 10 days
Acute uncomplicated pyelonephritis	Urine culture with a bacterial count of100,000 CFU per mL of urine	Same as for acute uncomplicated cystitis	If gram-negative organism, oral fluoroquinolone If gram-positive organism, amoxicillin If parenteral administration is required, ceftriaxone (Rocephin) or a fluoroquinolone If Enterococcus species, add oral or IV amoxicillin	Switch from IV to oral administration when the patient is able to take medication by mouth; complete a 14-day course
Complicated urinary tract infection	Urine culture with a bacterial count of more than 10,000 CFU per mL of urine	*E. coli* *K. pneumoniae* *P. mirabilis* *Enterococcus species* *Pseudomonas aeruginosa*	If gram-negative organism, oral fluoroquinolone If Enterococcus species, ampicillin or amoxicillin with or without gentamicin (Garamycin)	Treat for 10 to 14 days

Asymptomatic bacteriuria in pregnancy	Urine culture with a bacterial count of more than 10,000 CFU per mL of urine	Same as for acute uncomplicated cystitis	Amoxicillin Nitrofurantoin (Macrodantin) Cephalexin (Keflex)	Avoid tetracyclines and fluoroquinolones Treat for three to seven days
Catheter-associated urinary tract	Symptoms and a urine culture with a bacterial count of more than100 CFU per mL of urine	Depends on duration of catheterization	If gram-negative organism, a fluoroquinolone	Remove catheter if possible, and treat for seven to 10 days
			If gram-positive organism, ampicillin or amoxicillin plus gentamicin	For patients with long-term catheters and symptoms, treat for five to seven days

Source*: https://www.aafp.org/pubs/afp/issues/1999/0301/p1225.html*

Disease management

For non pregnant women with lower UTI

- Trimethoprim 200mg bd PO or nitrofurantoin 50 mg/6 hourly PO for 3- 6 days, amoxicillin 500 mg/8 hourly PO,
- Alternative: cefalexin l g bd for 7 days
- Second line: co-amoxiclav PO for 7 days

For non pregnant women with upper UTI:

- co-amoxiclav IV 1. 2g/8hourly then PO for 7 days

For pregnant women:

- cephalexin 250 mg tds and get expert Opinium.

For men:

- 7 days course of trimethoprim or quinolone (ciprofloxacin 500 mg bd)

Asymptomatic bacteriuria in elderly men (>65 years):

- antibiotic therapy is not recommended.

All men with upper UTI, recurrent UTI or fail to respond antibiotic therapy:

- should be referred to urologist.
- Intermittent self catheterization (to insert catheter into his or her bladder four to five times per day to drain urine) in catheterized patient.

For children:

- trimethoprim bd for 3 days for lower UTI and 7 days for upper UTI

Recurrent Cystitis in Young Women

Up to 20 percent of young women with acute cystitis develop recurrent UTIs.

During these recurrent episodes, the causative organism should be identified by urine culture and then documented to help differentiate between relapse (infection with the same organism) and recurrence (infection with different organisms).

Multiple infections caused by the same organism require longer courses of antibiotics and possibly further diagnostic tests.

The most recurrent UTIs in young women are uncomplicated infections caused by different organisms.

These infections are generally not associated with underlying anatomic abnormalities and do not require further work-up of the genitourinary tract.

Women who have more than three UTI recurrences within one year can be managed using one of three preventive strategies

Acute self-treatment with a three-day course of standard therapy.

Postcoital prophylaxis with one-half of a trimethoprim-sulfamethoxazole double-strength tablet (40/200 mg) if the UTIs have been clearly related to intercourse.

Continuous daily prophylaxis with one of these regimens for a period of six months: trimethoprim-sulfamethoxazole, one-half tablet per day (40/200 mg); nitrofurantoin, 50 to 100 mg per day; norfloxacin,

200 mg per day; cephalexin (Keflex), 250 mg per day; or trimethoprim, 100 mg per day.

Complicated UTI

A complicated UTI is one that occurs because of **anatomic, functional or pharmacologic factors** that predispose the patient to persistent infection, recurrent infection or treatment failure.
These **factors** include conditions often encountered in elderly men, such as **enlargement of the prostate gland, blockages and other problems** necessitating the placement of indwelling urinary devices, and the presence of bacteria that are resistant to multiple antibiotics.

Treatment

- most often includes a **fluoroquinolone**, administered orally if possible.

In patients who are unable to tolerate oral medication or who require hospitalization for concomitant medical problems, appropriate initial therapy may be parenteral administration of one of the following:

- a third-generation cephalosporin with antipseudomonal activity such as ceftazidime (Fortaz) or
- cefoperazone (Cefobid), cefepime (Maxipime), aztreonam (Azactam), imipenemcilastatin (Primaxin) or
- the combination of an antipseudomonal penicillin (ticarcillin [Ticar], mezlocillin [Mezlin], piperacillin [Pipracil]) with an aminoglycoside.

UTI in Men

- Urinary tract infections most commonly occur in older men with prostatic disease, outlet obstruction or urinary tract instrumentation.
- These infections occur in young men
  - who participate in anal sex (exposure to *E. coli* in the rectum),
  - who are not circumcised (increased *E. coli* colonization of the glans and prepuce) or
  - whose sexual partner is colonized with uropathogens.
- In men (unlike in women), a urine culture growing **more than 1,000 CFU of a pathogen per mL of urine is the best sign of a urinary tract infection**, with a sensitivity and specificity of 97 percent.
- Men with urinary tract infections should receive **a minimum of seven days of antibiotic therapy** (either trimethoprim-sulfamethoxazole or a fluoroquinolone).
- More extensive courses may be required in, men with associated urinary tract infection and prostatitis.
- Among young men with acute cystitis who respond to seven days of treatment, diagnostic work-ups beyond cultures are generally unrewarding.
- Urologic evaluation should be performed routinely in adolescents and men with pyelonephritis or recurrent infections.
- When bacterial prostatitis is the source of a urinary tract infection, eradication usually requires antibiotic therapy for six to 12 weeks and in rare instances even longer

Refer to urology

If any abnormalities detected on further investigation or unable to resolve symptoms

Prevention of recurrent cystitis

- **General advice**: advise patient to urinate frequently, double void
- wipe from front to back when go to the toilet
- keep the genital area clean and dry
- drink plenty of fluids, particularly water – so that regularly urinate during the day and do not feel thirsty
- wash the skin around the vagina with water before and after sex
- urinate as soon as possible after sex

- promptly change nappies or incontinence pads if they're soiled
- Prophylactic antibiotic: either post-coitally (e.g. nitrofurantoin 50mg stat) or continuously (trimethoprim 1 mg hs or nitrofurantoin 50mg hs)
- Men with BPH: finasteride or dutasteride and/or doxazosin
- HRT: topical oestrogens

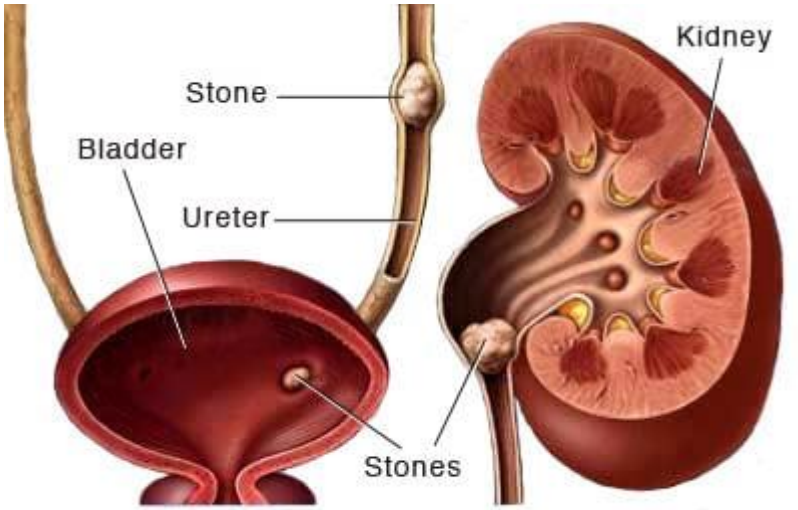
Reference

1. Oxford handbook of Clinical Medicine, 10th Edition
2. *Oxford hand book of General Practice 4th Edition*
3. *https://www.nhs.uk/conditions/urinary-tract-infections-utis/*
4. *https://www.aafp.org/pubs/afp/issues/1999/0301/p1225.html*

RENALSTONES

INCIDENCE

- About **12%** of Male and **3%** of Female will develop a renal stone at some point; peak age 20- 50 years. Symptoms are not dependent on size of the stone.



Risk factors

- Family history: increased risk 3 times. *Specific conditions:* X-linked nephrolithiasis, cystinuria, hyperoxaluria
- Anatomically abnormal kidneys, e.g. horseshoe kidney, medullary sponge kidney
- Metabolic disease, e.g. gout, hypercalcaemia / hypercalciuria, cystinuria, renal tubular acidosis or other acidosis (ileostomy, adenomatous polyp), oxaluria, aminoaciduria
- Dehydration
- Immobilization
- Chronic UTI

Composition of kidney stones in developed country

Stone type	Children (%)	Adult (%)
Calcium	50 - 90	64 - 92
Calcium oxalate	60 - 90	32 - 46
Calcium phosphate	10 - 20	3 - 5
Both calcium oxalate & phosphate	-	29 - 40
Cystine	1 - 5	1
Struvite (Magnesium ammonium phosphate)	1 -18	2 - 15
Uric acid	*1- 0*	3 -16
Other	4	1

Causes

- Kidney stones often have no definite, single cause, although several factors may increase your risk.
- Kidney stones form when your urine contains more crystal-forming substances — such as calcium, oxalate and uric acid — than the fluid in your urine can dilute.
- At the same time, your urine may lack substances that prevent crystals from sticking together, creating an ideal environment for kidney stones to form

Drugs predisposing patients to stone formation

- Acetazolamide, allopurinol, aspirin, steroids, indinavir, nelfinavir, loop diuretics, probenecid, quinolones, sulfonamides, theophylline, thiazides, triamterene, antacids, calcium/vitamin D supplements,high-dose vitamin C.

Types of kidney stones

Calcium stones

- Most kidney stones are calcium stones, usually in the form of calcium oxalate. Oxalate is a substance made daily by your liver or absorbed from your diet. Certain fruits and vegetables, as well as nuts and chocolate, have high oxalate content.
- Dietary factors, high doses of vitamin D, intestinal bypass surgery and several metabolic disorders can increase the concentration of calcium or oxalate in urine.
- **Struvite stones.** Struvite stones form in response to a urinary tract infection. These stones can grow quickly and become quite large, sometimes with few symptoms or little warning.
- **Uric acid stones.** Uric acid stones can form in people who lose too much fluid because of chronic diarrhea or malabsorption, those who eat a high-protein diet, and those with diabetes or metabolic syndrome.
- **Cystine stones.** These stones form in people with a hereditary disorder called cystinuria that causes the kidneys to excrete too much of a specific amino acid.

Risk factors

- **Family or personal history.**
- **Dehydration.** Not drinking enough water each day can increase your risk of kidney stones.
- **Certain diets.** Eating a diet that's high in protein, sodium (salt) and sugar may increase your risk of some types of kidney stones.
- **Obesity.** High body mass index (BMI), large waist size and weight gain have been linked to an increased risk of kidney stones.
- **Digestive diseases and surgery.** Gastric bypass surgery, inflammatory bowel disease or chronic diarrhea can cause changes in the digestive process that affect your absorption of calcium and water, increasing the amounts of stone-forming substances in your urine.
- **Other medical conditions** such as renal tubular acidosis, cystinuria, hyperparathyroidism and repeated urinary tract infections also can increase your risk of kidney stones.
- **Certain supplements and medications,** such as vitamin C, dietary supplements, laxatives (when used excessively), calcium-based antacids, and certain medications used to treat migraines or depression, can increase risk of kidney stones.

Presentation

- A kidney stone usually will not cause symptoms until it moves around within the kidney or passes into one of the ureters.
- If a kidney stone becomes lodged in the ureters, it may block the flow of urine and cause the kidney to swell and the ureter to spasm, which can be very painful.

- Usually presents with pain ± nausea/vomiting. Location and type of pain gives clues about the site of the stone:
    - Loin pain - kidney stone
    - Renal colic - ureteric stone
    - Renal colic (Sign and symptoms)
- Severe sharp pain with waves of increased severity. Usually starts abruptly as flank pain, below the ribs which then radiates around the abdomen to the groin as stone progresses down the ureter.
- Pain that comes in waves and fluctuates in intensity
- May be referred to testis/ tip of penis in men or labia majora in women.
- Pain or burning sensation while urinating
- Patient is obviously in pain - usually unable to sit still and keeps shifting position to try to get comfortable (in contrast to peritonitis where patients tend to keep still).
- May be pale and sweaty.
- May be mild tenderness on deep abdominal palpation or loin tenderness, though often minimal signs. If fever suspect infection.
- Pink, red or brown urine
- Cloudy or foul-smelling urine
- A persistent need to urinate, urinating more often than usual or urinating in small amounts
- Nausea and vomiting
- Fever and chills if an infection is present

Other presentations

- UTI, haematuria, retention, renal failure (rare).
- Pain caused by a kidney stone may change — shifting to a different location or increasing in intensity — as the stone moves through your urinary tract.

DIFFERENTIAL DIAGNOSIS

- Pyelonephritis
- ruptured Aneurysm of Abdominal Aorta
- Cholecystitis
- Pancreatitis
- Appendicitis
- Diverticulitis
- intestinal obstruction
- strangulated hernia
- testicular torsion
- pethidine addiction

Immediate investigation

- Dipstick urine if possible. Absence of RBCs does not exclude renal colic but consider alternative diagnosis.

Immediate management

- Stones usually pass spontaneously. Give pain relief (diclofenac 75mg IM/ l00mg PR)±antiemetic.

When to refer

- Any of the following:
- stone >5 mm in diameter
- high-grade obstruction
- gross hydronephrosis

- fever/UTI
- unremitting pain
- stone fails to progress
- Pregnant
- Strangury - bladder stone
- Interruption of flow - urethral stone

Investigation

- Blood U&E, creatinine, eGFR, Ca2+, P043-, alkaline phosphatase, uric acid, albumin
- Urine M, C&S; RBCs., Ca2+, P04 3-, uric acid, and sodium excretion
- Radiology X-ray of kidneys, ureters, and bladder - 90% of renal stones are radio- opaque only urate and xanthine stones are radio-translucent;
- High-speed or dual energy computerized tomography (CT) may reveal even tiny stones.
- Renal tract USS
- Analysis of passed stones

Management

Treatment for kidney stones varies, depending on the type of stone and the cause.

Small stones with minimal symptoms

- Most small kidney stones won't require invasive treatment. You may be able to pass a small stone by:
- **Drinking water.** Encourage drinking as much as 2 to 3 quarts (1.8 to 3.6 liters) a day will keep your urine dilute and may prevent stones from forming. Sieve urine for stones.
- **Pain relievers.** Passing a small stone can cause some discomfort. To relieve mild pain, your doctor may recommend pain relievers such as ibuprofen (Advil, Motrin IB, others) or naproxen sodium (Aleve).
- Monitor/review pain relief and for complications.
- Diclofenac 75 mg IM injection then 50 mg (oral) tds for 1 week.
- Several clinical trials have shown that NSAIDs by IM injection, including ketorolac (10-30 mg IM), are effective and at least as efficacious as opioids
- **Medical therapy.** a medication to help pass your kidney stone.
- This type of medication, known as an alpha blocker, relaxes the muscles in your ureter, helping you pass the kidney stone more quickly and with less pain. Alpha blockers include tamsulosin (Flomax) and the drug combination dutasteride and tamsulosin (Jalyn)

Large stones and those that cause symptoms

- Kidney stones that are too large to pass on their own or cause bleeding, kidney damage or ongoing urinary tract infections may require more-extensive treatment.
- **Using sound waves to break up stones.** For certain kidney stones — depending on size and location — may recommend a procedure called extracorporeal shock wave lithotripsy (ESWL).
- ESWL uses sound waves to create strong vibrations (shock waves) that break the stones into tiny pieces that can be passed in your urine. The procedure lasts about 45 to 60 minutes and can cause moderate pain, so it may be under sedation or light anesthesia to make you comfortable.
- **Surgery to remove very large stones in the kidney.** A procedure called percutaneous nephrolithotomy,
- involves surgically removing a kidney stone using small telescopes and instruments inserted through a small incision in back.
- **Using a scope to remove stones.** To remove a smaller stone in your ureter or kidney, doctor may pass a thin lighted tube (ureteroscope) equipped with a camera through your urethra and bladder to your ureter.

Urine alkalinization:

- The mainstay for medical management of uric acid stones is alkalinization (increasing the pH) of the urine.
- Uric acid stones are among the few types amenable to dissolution therapy, referred to as chemolysis. Chemolysis is usually achieved through the use of oral medications
- Diuretics:
- One of the recognized medical therapies for prevention of stones is the thiazide and thiazide-like diuretics, such as chlorthalidone or indapamide.
- These drugs inhibit the formation of calcium-containing stones by reducing urinary calcium excretion.
- Allopurinol: For people with hyperuricosuria and calcium stones, allopurinol is one of the few treatments that have been shown to reduce kidney stone recurrences.

Medical expulsive therapy

- The use of medications to speed the spontaneous passage of stones in the ureter is referred to as medical expulsive therapy. alpha adrenergic blockers (such as tamsulosin) and calcium channel blockers (such as nifedipine), have been found to be effective.

- *Antispasmodics to facilitate stone passage**

Alpha blockers	
Doxazosin (Cardura)	4 mg orally per day
Tamsulosin (Flomax)	0.4 mg orally per day
Calcium channel blocker	
Nifedipine (Procardia, SR)	30 mg orally per day

- 50% recur in 5-7years. Give general advice on prevention of stones (see Table 2).
- If investigations show any loss of renal function, renal obstruction, or remaining stones - refer to urology.
- Dependent on composition of stones, give dietary advice/refer to dietician (see Table 14.6).

HYPEROXALURIA

- May be 1° (autosomal recessive condition) or s to gut resection/malabsorption or dietary excess of spinach or vitamin C.
- Take specialist advice on management. There are two types of p hyperoxaluria:
- Type 1 hyperoxaluria -Calcium oxalate stones are widely distributed throughout the body. Presents as renal stones and nephrocalcinosis in children. 80% have chronic renal failure in <20years.
- Type 2 hyperoxaluria- More benign but less common - nephrocalcinosis but no chronic renal failure.

CYSTINURIA

- Most common aminoaciduria. Usually presents with stones at age 10 -30years. If the *Urine - increased the* cystine, omithine, arginine and lysine. Take specialist advice on management.

HYPERCALCAEMIA

- Hypercalciuria may occur without hypercalcaemia and is found in 80% of patients with calcium oxalate stones

Table 1:Prevention of renal stones

Type of stone	Preventative measures
All types	increase fluid intake (>2-2.5L/24h), especially in hot weather decrease weight if obese; decrease animal protein and increase fruit/ vegetables in diet; decrease salt intake
Calcium oxalate	Urinary alkalinization with potassium citrate; avoid chocolate, tea, rhubarb and spinach, nuts, beans, beetroot; decrease citrus fruits; bendroflumethiazide 2.5mg od may help if hypercalciuria; hyperoxaluria is treated with pyridoxine
Calcium phosphate	Low Ca2+ diet; avoid vitamin D supplements. Bendroflumethiazide 2.5mg od may help if hypercalciuria
Staghorn/ triple phosphate (calcium, magnesium, and ammonium	Associated with UTI due to *Proteus* species and urinary stasis, e.g. due to anatomical abnormality. Treat UTI with antibiotics
Urate	Avoid beer as has uricosuric effect; allopurinol; urinary alkalinization with potassium citrate (pH >6.5)
Cystine	Urinary alkalinization with potassium citrate

THE DIETARY ADVICE FOR RECURRENT URINARY CALCULI INCLUDES:

- Drink at least 2L of water every day, or more if there is increased fluid loss: this is the most important step.
- Minimise consumption of foods that contain oxalate or uric acid. Foods that contain oxalate include:
- chocolate
- coffee
- cola and similar 'soft' drinks
- rhubarb
- tea
- Foods that contain uric acid include:
- beer
- red wine
- red meat
- organ meats
- Avoid milk in tea - calcium precipitates oxalate.
- Avoid processed meats, organ meats (e.g. brain, kidney, liver and sweetbread), yeast spreads and other high-salt foods. Restrict salt intake.
- Reduce animal protein consumption: restriction to one major meat meal a day (includes chicken and fish).
- Add citrate-containing fruit juices to the diet, including grapefruit, apple and orange Juice.
- Eat a healthy diet of vegetables and fruit with a high fibre content.
- Drink at least 2L of water every day, or more if there is increased fluid loss: this is the most important step.

Minimise consumption of foods that contain oxalate or uric acid. Foods that contain oxalate include:

- Chocolate
- Coffee
- cola and similar 'soft' drinks
- rhubarb
- tea
- Foods that contain uric acid include:
- beer
- red wine
- red meat

- organ meats
- Avoid milk in tea - calcium precipitates oxalate.
- Avoid processed meats, organ meats (e.g. brain, kidney, liver and sweetbread), yeast spreads and other high-salt foods. Restrict salt intake.
- Reduce animal protein consumption: restriction to one major meat meal a day (includes chicken and fish).
- Add citrate-containing fruit juices to the diet, including grapefruit, apple and orange juice.
- Eat a healthy diet of vegetables and fruit with a high fibers content.

REFERENCES

1. John Murtagh 'S General Practice-5th Edition
2. *Oxford Handbook of General Practice _4th Edition*
3. *LYNDA FRASSETTO, MD, et.al, University of California School of Medicine, San Francisco, California,, American Family Physician, Volume 84, Number I]December 1, 201*
4. https://www.urologyhealth.org/urology-a-z/k/kidney-stones
5. *https://www.mayoclinic.org/diseases-conditions/kidney-stones/symptoms-causes/syc-20355755*

BLADDER STONES

Bladder stones are hard lumps of minerals that can form inside the bladder when it's not completely empty of urine.
They may not cause any symptoms if they're small enough to be passed out of the bladder when urination
But most people with bladder stones do experience symptoms because the stones either irritate the wall of the bladder or block the flow of urine.

Typical symptoms

lower abdominal pain, which can often be severe (men may also have pain in or around their penis)
pain or difficulty when peeing
peeing more frequently (particularly at night)
cloudy or dark-coloured urine
blood in the urine
Most cases of bladder stones affect men aged 50 or older because of the link with prostate enlargement.
But both men and women can get bladder stones. It's rare for bladder stones to affect children.
In children, they can lead to bedwetting, and some boys may experience priapism, a persistent and often painful erection that can last for hours.

Causes bladder stones

Bladder stones usually form when you can't completely empty your bladder of urine.
A common reason for this in men is having an enlarged prostate gland that blocks the flow of urine.
If urine sits in the bladder for a long time, chemicals in the urine form crystals, which harden into bladder stones.

Treatment

Surgery is usually needed to remove the stones from the bladder.
The most common procedure is a cystolitholapaxy, where a thin tube (cystoscope) with a camera at the end is used to find the bladder stones.
The cystoscope will then use stone-crushing devices, lasers or ultrasound to break up the stones before they're removed.
Where possible, it's important to treat the underlying causes of bladder stones to prevent new stones developing in the future.

Preventing bladder stones

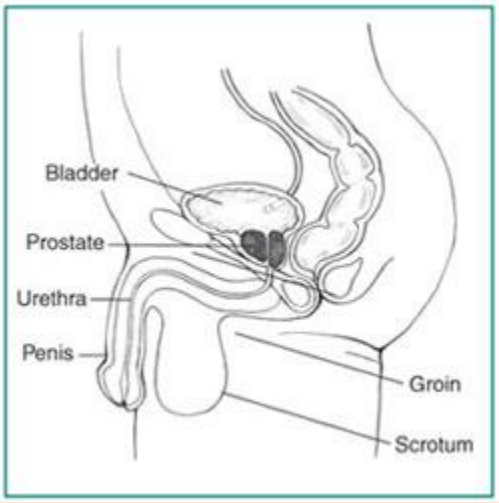
If you have had bladder stones, they can come back.
increase your daily fluid intake to 2 to 3 litres to lower the concentration of your urine
regularly empty your bladder without delaying
urinate again 10 to 20 seconds after your first attempt (if you're unable to empty your bladder completely first time); this is called double voiding and helps empty the bladder more efficiently
avoid constipation (regular laxatives may be recommended)

Reference

1. *https://www.nhs.uk/conditions/bladder-stones/*
2. *https://www.urologyhealth.org/urology-a-z/k/kidney-stones*

BENIGN PROSTATIC HYPERPLASIA (BPH)

Benign prostatic hyperplasia—also called BPH—is a condition in men in which the prostate gland is enlarged and not cancerous. Benign prostatic hyperplasia is also called benign prostatic hypertrophy or benign prostatic obstruction.



Causes

The cause of benign prostatic hyperplasia is not well understood.
Occurs mainly in older men.
Benign prostatic hyperplasia does not develop in men whose testicles were removed before puberty.
Throughout their lives, men produce testosterone, a male hormone, and small amounts of estrogen, a female hormone.
As men age, the amount of active testosterone in their blood decreases, which leaves a higher proportion of estrogen.
Benign prostatic hyperplasia may occur because the higher proportion of estrogen within the prostate increases the activity of substances that promote prostate cell growth.
Another theory focuses on dihydrotestosterone (DHT), a male hormone that plays a role in prostate development and growth. It was indicated that even with a drop in blood testosterone levels, older men continue to produce and accumulate high levels of DHT in the prostate.
This accumulation of DHT may encourage prostate cells to continue to grow.
BPH is the most common prostate problem for men older than age 50. Benign prostatic hyperplasia affects about 50 percent of men between the ages of 51 and 60 and up to 90 percent of men older than 80.

Risk factor

Men with the following factors are more likely to develop benign prostatic hyperplasia:

- age 40 years and older
- family history of benign prostatic hyperplasia
- medical conditions such as obesity, heart and circulatory disease, and type 2 diabetes
- lack of physical exercise
- erectile dysfunction

Symptoms

Lower urinary tract symptoms suggestive of benign prostatic hyperplasia may include

- urinary frequency—urination eight or more times a day
- urinary urgency—the inability to delay urination
- trouble starting a urine stream
- a weak or an interrupted urine stream
- dribbling at the end of urination
- nocturia—frequent urination during periods of sleep

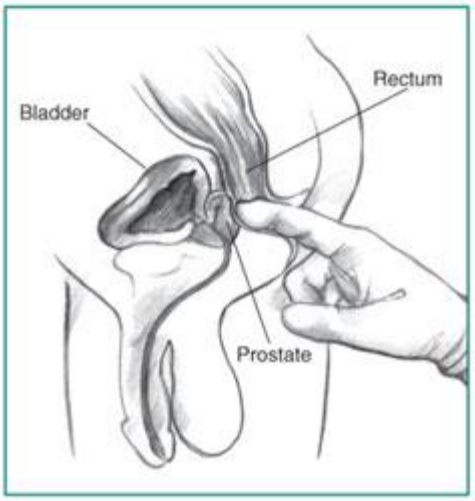
- urinary retention
- urinary incontinence—the accidental loss of urine
- pain after ejaculation or during urination
- urine that has an unusual color or smell
- Symptoms of benign prostatic hyperplasia most often come from
- a blocked urethra
- a bladder that is overworked from trying to pass urine through the blockage

Complications

acute urinary retention
chronic, or long lasting, urinary retention
blood in the urine
urinary tract infections (UTIs)
bladder damage
kidney damage
bladder stones
Seeking to Medical Care
Men with the following symptoms should seek immediate medical care:
complete inability to urinate
painful, frequent, and urgent need to urinate, with fever and chills
blood in the urine
great discomfort or pain in the lower abdomen and urinary tract

Diagnosis

A health care provider diagnoses benign prostatic hyperplasia based on
a personal and family medical history
a physical exam - performs a digital rectal exam
medical tests



Digital rectal exam

Medical Tests

Urinalysis
a prostate-specific antigen (PSA) blood test - men who have prostate cancer may have a higher amount of PSA in their blood. However, a high PSA level does not necessarily indicate prostate cancer.
urodynamic tests
Cystoscopy
transrectal ultrasound
Biopsy

Treatment

lifestyle changes
Medications
minimally invasive procedures
Surgery

Lifestyle Changes

Lifestyle changes can include
reducing intake of liquids, particularly before going out in public or before periods of sleep
avoiding or reducing intake of caffeinated beverages and alcohol
avoiding or monitoring the use of medications such as decongestants, antihistamines, antidepressants, and diuretics
training the bladder to hold more urine for longer periods
exercising pelvic floor muscles
preventing or treating constipation

Medications

alpha blockers
phosphodiesterase-5 inhibitors
5-alpha reductase inhibitors
combination medications

Alpha blockers.
These medications relax the smooth muscles of the prostate and bladder neck to improve urine flow and reduce bladder blockage:
terazosin (Hytrin)
doxazosin (Cardura)
tamsulosin (Flomax)
alfuzosin (Uroxatral)
silodosin (Rapaflo)

Phosphodiesterase-5 inhibitors.
Urologists prescribe these medications mainly for erectile dysfunction

5-alpha reductase inhibitors.
These medications block the production of DHT, which accumulates in the prostate and may cause prostate growth:
finasteride (Proscar)
dutasteride (Avodart)
These medications can prevent progression of prostate growth or actually shrink the prostate in some men. Finasteride and dutasteride act more slowly than alpha blockers and are useful for only moderately enlarged prostates.

Combination medications
The combinations include
finasteride and doxazosin
dutasteride and tamsulosin (Jalyn), (a combination of both medications that is available in a single tablet)
alpha blockers and antimuscarinics

Minimally Invasive Procedures

These procedures include:
transurethral needle ablation
transurethral microwave thermotherapy
high-intensity focused ultrasound
transurethral electrovaporization
water-induced thermotherapy
prostatic stent insertion
Minimally invasive procedures can destroy enlarged prostate tissue or widen the urethra, which can help relieve blockage and urinary retention caused by benign prostatic hyperplasia.

Surgery

For long-term treatment of benign prostatic hyperplasia, a urologist may recommend removing enlarged prostate tissue or making cuts in the prostate to widen the urethra. Urologists recommend surgery when
medications and minimally invasive procedures are ineffective
symptoms are particularly bothersome or severe
complications arise
Although removing troublesome prostate tissue relieves many benign prostatic hyperplasia symptoms, tissue removal does not cure benign prostatic hyperplasia.
TURP. Transurethral resection of the prostate
Laser surgery
Open prostatectomy

Reference

1. *https://www.mayoclinic.org/diseases-conditions/benign-prostatic-hyperplasia/symptoms-causes/syc-20370087*
2. *https://www.nhs.uk/conditions/prostate-enlargement/*
3. *https://my.clevelandclinic.org/health/diseases/9100-benign-prostatic-hyperplasia*
4. *https://www.urologyhealth.org/urology-a-z/b/benign-prostatic-hyperplasia-(bph)*