GUIDELINES

For

GENERAL PRACTITIONERS

2024

Press record

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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recommendations for clinicians who are uncertain about how to proceed, overturn the beliefs of doctors accustomed to outdated practices, improve the consistency of care, and provide authoritative recommendations that reassure practitioners about the appropriateness of their treatment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 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**

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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

EYE PROBLEMS

Content

OCULAR TRAUMA

- Depending on the structures involved - Eyelid trauma, Orbital trauma, Eyeball (globe) rupture/ perforation
- Nature of injury - blunt trauma, penetrating injuries and chemical injuries

EYELID INJURY

- Lid laceration occurs in the context of both blunt and sharp injuries

Management

- Repair of eyelid laceration needs the expertise and proper suture material and suturing technique.
- It is best to repair in the hand of an ophthalmologist. (REFER)
- Never cut and throw any periocular tissue.
- As blood supply is abundant in facial area, even a necrotic looking tissue can survive later.
- Check the immune status for tetanus and systemic antibiotics is recommended.

ORBITAL INJURIES

- Resulting in soft tissue oedema and haemorrhage within the non-expansile bony orbit. It is usually associated with facial or head injuries.
- Therefore, it is important to assess any associated life threatening injury (airway, chest or neuro) associated before referring eye centre.

Management

- Prioritize where to REFER the patient.

EYEBALL INJURIES (GLOBE) RUPTURE/PERFORATION

- Injury with blunt objects can result the globe rupture and optic nerve trauma.

PENETRATING INJURY

- Following injury from sharp objects and projectiles with high mass and/or velocity.
- Penetrating injuries are three times more common in males than females, and typically occur in a younger age group (50% aged 15-34).
- The most frequent causes are assault, domestic and occupational accidents, and sport.

Management

- Patch the eye with a clean pad in eyeball injury.
- No eye drops or ointment into the eye is advisable. Then refer the patient to the eye center.
- Health education on use of protective eyewear
- Serious eye trauma from penetrating injuries could be prevented by the appropriate use of protective eyewear at work or during sports activity.

CHEMICAL INJURIES

- May occur in domestic, industrial and military settings, any corrosive agents
- common alkali, oven cleaning fluid, caustic soda and plaster
- Common acids - battery fluid, lavatory cleaning fluid, bleach and pool cleaning fluid

Management

- Can lead to blindness if timely referral and treatment is not possible.
- However, immediate irrigation with any available clean water for at least 30 min at the scene of injury is the most important determinant of outcome.
- Therefore, irrigate copiously in case of chemical injury at least an hour before referring eye center.

RED EYES

CONJUNCTIVITIS

- Bacterial
- Simple bacterial conjunctivitis
- Gonococcal keratoconjunctivitis
- Viral
- Adenoviral keratoconjunctivitis
- Herpes simplex conjunctivitis
- Chlamydial
- Trachoma
- Adult chlamydial keratoconjunctivitis
- Neonatal chlamydial conjunctivitis - Acute, profuse, purulent discharge, hyperaemia and chemosis, Corneal ulceration, perforation and endophthalmitis if severe

Presentations

- Crusted eyelids and conjunctival injection, mucopurulent discharge in Bacterial conjunctivitis
- Vision - unaffected, clear cornea and quiet anterior chamber

Treatment

- broad-spectrum topical antibiotics

Management of viral conjunctivitis

- Most of the conjunctivitis is viral in origin and takes time to resolve totally (two weeks).
- Symptomatic relief is mainly indicated - ample lubricant use and ice compression.
- Simple antibiotic eye-drop can be given to prevent secondary bacterial infection in severe cases, but not always necessary.
- Steroid eye drops should be avoided before proper ophthalmic assessment.
- Simple conjunctivitis never affects the eye sight (except transient blurring from mucous discharge)
- If the course of illness is more than few days or vision is deteriorating or affected from the beginning, please refer the patient to eye centre.

CORNEAL ULCERS

- It is a serious condition and requires ophthalmic REFERRAL
- The injudicious use of topical steroid in corneal infection predisposes to develop corneal ulcers.

CORNEAL INFECTION - KERATITIS

- Bacterial keratitis
- Fungal keratitis

- Acanthamoeba keratitis
- Herpes simplex keratitis
- Herpes zoster keratitis

Presentation

painful red eye with reduced vision in affected eye

BACTERIAL KERATITIS

- Predisposing factors - contact lens wear, defective ocular surface - trauma, post herpetic, exposure, administration of topical corticosteroids
- Staph aureus, Strep pneumoniae: oval, yellow - white, densely opaque stromal suppuration surrounding relatively clear cornea
- Pseudomonus: thick mucopurulent exudates, diffuse liquifactive necrosis and semi- opaque ground glass appearance of adjacent stroma - rapid progression
- Corneal perforation common within 48 hours

Management

- URGENT REFERRAL to ophthalmic centre since hospital admission is advisable in severe cases

ANGLE CLOSURE GLAUCOMA

(see common eye diseases)

EYELID INFECTION & INFLAMMATION

STYLE

- Acute small staphylococcal abscess of a lash follicle and its associated glands.
- Tender inflamed swelling pointing anteriorly through the skin

Treatment

- no treatment is necessary as spontaneous resolution in most cases
- Hot compression, Epilation (removal of lip hair by roots) is helpful.
- Systemic antibiotics are recommended.

CHALAZION

- Chronic lipogranulomatous inflammatory lesion caused by blockage of gland orifices and stagnation of sebaceous secretions
- Presents with painless, roundish, firm nodule

Treatment

- Surgery (incision & curettage)
- Steroid injection
- Systemic antibiotics

EYELID TUMORS

- Numerous benign and malignant cutaneous neoplasms can develop in the periocular skin.
- The malignant lesions that most frequently affect the eyelids are basal cell carcmoma, squamous cell carcinoma, sebaceous cell carcinoma, and melanoma.

Presentations

- slow, painless growth of a lesion
- ulceration, drainage, bleeding, and crusting
- pigmentary changes
- destruction of normal eyelid margin architecture.

Management

- Any benign looking tumors should be monitored with photographic document and regular follow-up.
- Any suspected cutaneous malignancies should be REFERED to the eye center for a biopsy for histologic examination.

LEUKOCORIA (WHITE PUPIL)

What is leukocoria?

Leukocoria literally means "white pupil."
It occurs when the pupil is white rather than the usual black.

How is leukocoria detected?

In obvious cases the pupil may appear white on casual observation.
Sometimes leukocoria is detected from photographs when one pupil has an abnormal or "white reflex" compared to the other eye having a normal "red reflex."

What conditions cause leukocoria?

Leukocoria can be caused by congenital or acquired eye diseases.
This is an ophthalmologic emergency particularly because of the need to promptly diagnose and treat conditions such as retinoblastoma, glaucoma, retinal detachment and infections.

The differential diagnosis

Includes many diseases. Important causes -

- Cataract
- Corneal opacity
- Glaucoma
- Persistent hyperplastic primary vitreous
- Retinal detachment
- Toxocara infection
- Endophthalmitis

RETINOBLASTOMA

Are any of these conditions serious?

All diseases which cause leukocoria represent a serious threat to vision and some pose a threat to life.
Prompt evaluation of leukocoria by an ophthalmologist is always appropriate.

STRABISMUS (SQUINTING EYES)

- What is strabismus and how common is it?
- Strabismus is any misalignment of the eyes. It is estimated that 4% of the population has strabismus.

Types

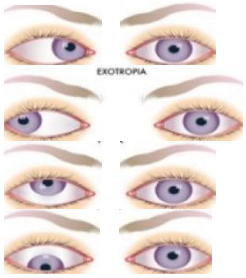
- Based on direction of the eye misalignment
- Common types of strabismus are

esotropia,

exotropia,

hypertropia,

hypotropia.



Picture credit: www.hollywoodvision.com

Based on its cause

- The 3 cranial nerves (III, IV, VI) responsible for eye movement can be weak or palsied and cause strabismus.
- Some examples of paralytic strabismus include:
- third nerve palsy
- superior oblique palsy.

Special patterns of strabismus:

- Brown syndrome,
- Duane syndrome.

HORIZONTAL STRABISMUS ESOTROPIA

- Inward turning of the eyes
- Infantile esotropia,
- Accommodative esotropia,
- Sixth nerve palsy.

EXOTROPIA

- Outward turning of the eyes

VERTICAL STRABISMUS

- Hypertropia
- Hypotropia

Hypertropia

- is an abnormal eye higher than the normal eye. Hypotropia is when the abnormal eye is lower than the normal eye.

Causes

- Most strabismus is the result of an abnormality of the poorly understood neuromuscular (including brain) control of eye movement. Less commonly, a problem with the actual eye muscle causes strabismus.

Strabismus related to poor vision

- Eye misalignment can cause amblyopia (lazy eye) in children. When the eyes are oriented in different directions, the brain receives 2 different visual images. The brain may ignore the image from the misaligned eye to avoid double vision, resulting in poor vision development of that eye. Also, an eye that sees poorly tends to be misaligned.
- The visual loss from amblyopia cannot be corrected by wearing glasses. However, it is usually treatable.
- If amblyopia is not treated before the age of about 7-8 years, the visual impairment usually remains permanent.

Who develops strabismus as a child?

- Strabismus often occurs in children who are otherwise completely normal.
- However, disorders that affect the brain such as cerebral palsy, Down syndrome, hydrocephalus and brain tumor are more likely to develop strabismus.

Trauma causing strabismus

- Trauma can cause strabismus by
- brain damage that impairs control of eye movement,
- damage of the nerves that control eye movement and/or
- damage of the eye muscles either directly or secondarily from trauma to the eye socket.

Treatment

- The goal of strabismus treatment is to improve eye alignment which allows for the eyes to better work together (binocular vision).
- Treatment may involve eye glasses, eye exercises, prism, and/ or eye muscle surgery. Problems associated with strabismus (including amblyopia, ptosis, and cataract) are usually treated prior to eye muscle surgery.

Is strabismus serious?

- All children with squinting eyes may have threat to vision and prompt evaluation by an ophthalmologist is always appropriate.

Reference

1. Provided by Eye Society, Department of Ophthalmology, Yangon., University of Medicine(]), Yangon Eye Hospital, and Yangon Eye Hospital

COMMON EYE PROBLEMS

REFRACTIVE ERRORS

- Refractive errors are a type of vision problem that makes it hard to see clearly.
- They happen when the shape of eye keeps light from focusing correctly on the retina (a light-sensitive layer of tissue in the back of the eye).
- Refractive errors are the most common type of vision problem.

Types

There are 4 common types of refractive errors:

- Nearsightedness (myopia)
- Farsightedness (hyperopia)
- Astigmatism
- Presbyopia

Symptoms

The most common symptom is blurry vision. Other symptoms include:

- Double vision
- Hazy vision
- Seeing a glare or halo around bright lights
- Squinting
- Headaches
- Eye strain (when eyes feel tired or sore)
- Trouble focusing when reading or looking at a computer
- Some people may not notice the symptoms of refractive errors.

Causes

- Refractive errors can be caused by:
- Eyeball length (when the eyeball grows too long or too short)
- Problems with the shape of the cornea (the clear outer layer of the eye)
- Aging of the lens (an inner part of the eye that is normally clear and helps the eye focus)

Treatment

- Refer to Eye Specialist.
- Eye doctors can correct refractive errors with glasses or contact lenses, or fix the refractive error with surgery.
- Glasses. Eyeglasses are the simplest and safest way to correct refractive errors.
- Eye doctor will prescribe the right eyeglass lenses to give the clearest possible vision.
- Contacts. Contact lenses sit on the surface of the eyes and correct refractive errors.
- Eye doctor will fit for the right lenses and show how to clean and wear them safely.
- Surgery. Some types of surgery, like laser eye surgery, can change the shape of cornea to fix refractive errors.

CATARACTS

- A cataract is a cloudy area in the lens of the eye.
- **Cataracts are very common when get older.**

- At first, the person may not notice that he has a cataract. But over time, cataracts can make the vision blurry, hazy, or less colorful.
- Trouble in reading or doing other everyday activities.
- Surgery can get rid of cataracts. Cataract surgery is safe and corrects vision problems caused by cataracts.

Types

- Most cataracts are **age-related**
- After an eye injury or after surgery for another eye problem (like glaucoma).
- No matter what type of cataract, the treatment is always surgery.
- By age 80, most people either have cataracts or have had cataract surgery

Symptoms

- No symptoms when cataracts are mild.
- When cataracts grow, changes in vision.
- Vision is cloudy or blurry
- Colors look faded
- Can't see well at night
- Lamps, sunlight, or headlights seem too bright
- See a halo around lights
- Double (this sometimes goes away as the cataract gets bigger)
- Change the prescription for glasses often
- Over time, cataracts can lead to vision loss.

Risk for cataracts

- Diabetes mellitus
- Smoke
- Drink too much alcohol
- family history of cataracts
- an eye injury, eye surgery, or radiation treatment on upper body
- spent a lot of time in the sun
- Take steroids (medicines used to treat a variety of health problems, like arthritis and rashes)

Causes of cataracts

- Most cataracts are caused by normal changes in your eyes as you get older.
- Around age 40, the proteins in the lens of the eye start to break down and clump together.
- This clump makes a cloudy area on lens — or a cataract. Over time, the cataract gets more severe and clouds more of the lens.

Prevention

- Protect the eyes and delay cataracts.
- Wear sunglasses and a hat with a brim to block the sun.
- Quit smoking.
- Eat healthy. Eat plenty of fruits and vegetables — especially dark, leafy greens like spinach, kale, and collard greens.
- Get a dilated eye exam. If age 60 or older, get a dilated eye exam at least once every 2 years.

Treatment

- Refer to eye specialist.
- check for cataracts as part of a dilated eye exam
- Surgery is the only way to get rid of a cataract.
- Use brighter lights at home or work
- Wear anti-glare sunglasses
- Use magnifying lenses for reading and other activities
- **New glasses or contacts.** A new prescription for eyeglasses or contact lenses can help to see better with cataracts early on.
- **Surgery.** During cataract surgery, the doctor removes the clouded lens and replaces it with a new, artificial lens (also called an intraocular lens, or IOL). This surgery is very safe, and 9 out of 10 people who get it can see better afterwards.

GLAUCOMA

- Glaucoma is a group of eye diseases that can cause vision loss and blindness by damaging a nerve in the back of the eye called the optic nerve.
- The symptoms can start so slowly that may not notice them.
- There's no cure for glaucoma, but early treatment can often stop the damage and protect the vision.

Types of glaucoma

- There are many different types of glaucoma, but the most common type is called **open-angle glaucoma** .
- Acute angle closure glaucoma
- Less common type, like angle-closure glaucoma and congenital glaucoma.

Symptoms of glaucoma

- At first, glaucoma doesn't usually have any symptoms. That's why half of people with glaucoma don't even know they have it.
- Over time, it may slowly lose vision, usually starting with the side (peripheral) vision — especially the part of vision that's closest to the nose.
- Because it happens so slowly, many people can't tell that their vision is changing at first.
- But as the disease gets worse, it may start to notice that it can't see things off to the side anymore.
- Without treatment, glaucoma can eventually cause blindness.

ACUTE ANGLE CLOSURE GLAUCOMA

- It is the glaucoma from blockage of the angle, from closure of the angle.
- It is characterized by intense pain, in the globe of the eye, in neighboring regions, the forehead, and above the affected eye.
- Sometimes it is located in the tooth area or upper jaw. Other times the picture is accompanied by general with abdominal symptoms. The presence of vomiting is very frequent.

Symptoms

- >40 years, typically elderly, long-sighted worsen with early cataract.

- The patient complains of clouded vision, colored halos around lights, considerable reduction, or loss of sight.

Signs

- Examination will reveal
- Increased intraocular pressure, between 60 and 100 mmHg
- When acute glaucoma continues without treatment with very high pressures for more than 6 h, the optic nerve is definitively damaged and atrophies or becomes complicated with a retinal vascular occlusion.
- Corneal edema, generally subepithelial, which leads to iridescence, clouded vision, or loss of vision.
- Dilated pupil, reduced, and sometimes missing, reaction to light.
- Very flat chamber.

Management

- It is an ocular emergency and needs to REFER URGENTLY. Timely referral and treatment can save the vision.
- Symptomatic treatment for severe vomiting and pain is helpful.

Referral for red eye

- Reduce visual acuity
- Pain deep in the eye not surface irritation as with conjunctivitis
- Absent or sluggish pupil response
- Corneal damage on fluorescein staining
- History of trauma
- Refer the patient to be seen by a specialist the same

Risk factor

- Anyone can get glaucoma, but some people are at higher risk. .Higher risk if
- Are over age 60
- Are African American and over age 40
- Have a family history of glaucoma

Red Flag sign

- Angle-closure glaucoma can cause these sudden symptoms:
- Intense eye pain
- Upset stomach (nausea)
- Red eye
- Blurry vision

Causes of glaucoma

- many people with glaucoma have high eye pressure.

Examination for glaucoma

- Eye specialist can check for glaucoma as part of a comprehensive dilated eye exam.
- The exam includes a visual field test to check your side vision.

Treatment for glaucoma

- **Medicines.** Eye drops are the most common treatment. They lower the pressure in the eye and prevent damage to optic nerve.
- Prostaglandins, like Xalatan (latanoprost), Travatan Z (travoprost), Zioptan (tafluprost), and Lumigan (bimatoprost)
- Rho kinase inhibitor, like Rhopressa (netarsudil)
- Nitric oxides, like Vyzulta (latanoprostene bunod)
- Miotic or cholinergic agents, like Isopto Carpine (pilocarpine)

side effects

- Stinging, itching, burning, and redness in your eye
- Blurry vision
- Changes in your eye color or the skin around your eye
- Headaches
- Dry mouth
- Changes in your energy level, heartbeat, or breathing
- **Laser treatment.** To lower the eye pressure, doctors can use lasers to help the fluid drain out of the eye. It's a simple procedure that the doctor can do in the office.
- **Surgery.** If medicines and laser treatment don't work, your doctor might suggest surgery.

MACULAR DEGENERATION

- Macular degeneration (also called age-related macular degeneration or AMD) is an eye disease that affects the central vision.
- It damages the macula, which is the center area of retina that allows to see fine details. It's the leading cause of vision loss in people over the age of 60.
- Macular degeneration can either be wet or dry.
- Wet AMD happens when abnormal blood vessels grow under the macula and leak blood and fluid. This damages the macula and leads to loss of central vision.
- Dry AMD results in the thinning of the macula, which blurs your central vision over time. Dry AMD is more common than the wet form, accounting for 70% to 90% of cases.
- Symptoms of AMD, which usually aren't noticed until the disease has progressed, include:
- Blurred central vision.
- Black or dark spots in the center part of field of vision.
- Wavy or curved appearance to straight lines.
- Although there is no cure, treatment can slow the progress of disease or prevent severe vision loss.
- Recent advances have been made in the treatment of wet AMD using intraocular injections of anti-VEGF medications.

FLOATERS

- These are tiny spots or specks that float across your field of vision. Most people notice them in well-lit rooms or outdoors on a bright day.
- Floaters are usually normal, but they sometimes can be a sign of a more serious eye problem, like retinal detachment. That's when the retina at the back of the eye separates from the layer

underneath. When this happens, it might also see light flashes along with the floaters or a dark shadow come across the edge of the sight.

- If you notice a sudden change in the type or number of spots or flashes, see or a new dark “curtain" in peripheral vision, go to eye doctor as soon as possible.

DRY EYES

- This happens when eyes can't make enough good-quality tears. It might feel like something is in the eye or like it’s burning. Rarely, in severe cases, extreme dryness can lead to some loss of vision.
- Some treatments include:
    - Using a humidifier in home
    - Special eye drops that work like real tears
    - Plugs in tear ducts to lessen drainage
- Lipiflow, a procedure that uses heat and pressure to treat dry eyes
- Testosterone eyelid cream
- Nutritional supplements with fish oil and omega-3
- If your dry eye problem is chronic, it may have dry eye disease. Eye specialist could prescribe medicated drops like cyclosporine (Cequa, Restasis) or lifitegrast (Xiidra) to stimulate tear production.

DIABETES AND THE EYE

Magnitude of the problem

- Ten percent or more Diabetic patient - visual impairment within 15 years of diagnosis
- Prevalence of Diabetic Retinopathy increases with duration of diabetes and patient age
- Major cause of visual loss in adults (20-64 years - 8% of legally blind) in most of the world is due to Diabetic Retinopathy (DR)

Effects of diabetes mellitus on the eye

- Prolong hyperglycemia can affect the eye
- Anterior segment - cornea epithelial erosion/ impaired healing (diabetic neuropathy), chronic open-angle glaucoma, neovascular glaucoma,transient variations in refractive error - changing glasses too often
- Increased incidence in development of senile cataract
- Posterior segment - Diabetic Retinopathy*
- Orbital / intraocular infection
- Neuro-ophthalmic presentations - ischaemic optic neuropathy, 3rd (pupil sparing), 4th,6th cranial nerves palsies.

DIABETIC RETINOPATHY (DR)

- It is an eye condition that can cause vision loss and blindness in people who have diabetes. It affects blood vessels in the retina (the light-sensitive layer of tissue in the back of your eye).

Symptoms of diabetic retinopathy

- The early stages of diabetic retinopathy usually don't have any symptoms.
- Some people notice changes in their vision, like trouble reading or seeing faraway objects.
- These changes may come and go.
- In later stages of the disease, blood vessels in the retina start to bleed into the vitreous.
- If this happens, it may see dark, floating spots or streaks that look like cobwebs. Sometimes, the spots clear up on their own — but it's important to get treatment right away.
- Without treatment, scars can form in the back of the eye. Blood vessels may also start to bleed again, or the bleeding may get worse.

Other problems associate diabetic retinopathy

- **Diabetic macular edema (DME).** Over time, about 1 in 15 people with diabetes will develop DME. DME happens when blood vessels in the retina leak fluid into the macula . This causes blurry vision.
- **Neovascular glaucoma.** Diabetic retinopathy can cause abnormal blood vessels to grow out of the retina and block fluid from draining out of the eye. This causes a type of glaucoma.
- **Retinal detachment.** Diabetic retinopathy can cause scars to form in the back of the eye. When the scars pull your retina away from the back of the eye, it's called tractional retinal detachment.

Risk for diabetic retinopathy

- Anyone with any kind of diabetes can get diabetic retinopathy — including people with type 1,

type 2, and gestational diabetes, - commoner in type 1 (40%) than in type 2 (20%)

- Duration of diabetes - most important risk factor - incidence of DR after 10 years is 50% and after 30 years 90% in type 1 DM, 5% in type 2 DM at presentation
- Risk increases the longer it has diabetes. Over time, more than half of people with diabetes will develop diabetic retinopathy.
- Women with diabetes who become pregnant — or women who develop gestational diabetes — are at high risk for getting diabetic retinopathy.
- Poor metabolic control and other risk factors - obesity, hyperlipidaemia and anaemia, Pregnancy, Hypertension, Nephropathy
- No prophylactic treatment apart from good metabolic control

Causes diabetic retinopathy

- It is caused by high blood sugar due to diabetes. Over time, having too much sugar in the blood can damage retina
- The damage to the eyes starts when sugar blocks the tiny blood vessels that go to the retina, causing them to leak fluid or bleed.
- To make up for these blocked blood vessels, the eyes then grow new blood vessels that don't work well. These new blood vessels can leak or bleed easily.

Check for diabetic retinopathy

- Eye doctors can check for diabetic retinopathy as part of a dilated eye exam.
- Regular eye exams of diabetes patient is important.
- If develop diabetic retinopathy, early treatment can stop the damage and prevent blindness.
- A fluorescein angiogram test is also important.

Clinical staging

- Non proliferative, proliferative and advanced DR.
- The progression and changes occur in a predictable stepwise fashion.

Clinical importance

- To maintain the good glycemic control
- To identify the stage of diabetic retinopathy when the active intervention is required to prevent further visual loss

DR SCREENING PROGRAM

- Awareness of importance of Screening and regular follow-up
- TIMELY REFERRAL is utmost important since DR is the preventable blindness if treated early
- For treating ophthalmologists, periodic assessment and identification of people at risk and giving in-time treatment
- Beginning 5 years after the onset of diabetes - type I diabetes mellitus
- Patients with type II diabetes mellitus - shortly after the diagnosis of diabetes
- Initial detailed retinal examinations - good peripheral retinal view
- Follow-up visits based on the clinical staging of DR

Management

- Always refer EVERY diabetic patient to eye center and
- check the patient if he or she attends without fail.
- Encourage the patient to attend the eye clinic on next follow-up appointment.

- Keep the blood glucose level as in normal range as possible.

Prevention

- Managing good control of blood sugar levels in a normal range. (A1C goal must be normal)
- Having high blood pressure or high cholesterol along with diabetes increases your risk for diabetic retinopathy.
- So controlling your blood pressure and cholesterol can also help lower your risk for vision loss.

Treatment for diabetic retinopathy and DME

- Good control your diabetes, blood pressure, and cholesterol.
- **Injections.** Medicines of anti-VEGF drugs can slow down or reverse diabetic retinopathy. Other medicines, called corticosteroids, can also help.
- **Laser treatment.** To reduce swelling in retina, lasers can be used to make the blood vessels shrink and stop leaking.
- **Eye surgery.** If retina is bleeding a lot or have a lot of scars in eye ,a vitrectomy surgery may needed.

Reference

1. *Provided by Eye Society, Department of Ophthalmology, Yangon., University of Medicine (1), Yangon Eye Hospital, and Yangon Eye Hospital*
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