Aster HOSPITALS & CLINICS
We'll Treat You Well

HealthNews DIGEST

SEPTEMBER 2024

Dr. Sherbaz Bichu

CEO & Specialist Anaesthetist
Aster Hospitals & Clinics, UAE

On behalf of Aster's leadership, I am excited to welcome you to the 25th edition of the HealthNews Digest. Two years ago, what started as a novel initiative evolved into a vital medium through which we continue to uphold our unwavering dedication to clinical superiority and knowledge dissemination.

The enthusiasm and contributions from our doctor fraternities have only increased since the launch, and the newsletter now features a diverse range of content, from innovative treatments of complex cases to articles on patient-centric care.

I sincerely appreciate all of the contributions made over the years. Your contribution has been crucial to HealthNews Digest's success, and I am confident that the exceptional teams of Aster doctors will continue to support it ardently by sharing their expertise in ensuring clinical distinction and the best possible patient care.

Dr. Ramanathan V

Medical Director
Aster Hospitals & Clinics, UAE

As the Medical Director for Aster Hospitals and Clinics, I welcome you to the 25th edition of our HealthNews Digest Newsletter. I am immensely pleased to witness this initiative built on the core idea of sharing clinical excellence, reaching new heights with each iteration and concluding two fruitful years.

I applaud the efforts of all those who have worked hard to make this newsletter a core part of the Aster ecosystem. Your hard work is appreciated and recognized.

With an eclectic mix of cases and articles, this newsletter has given our doctor fraternities and allied professionals several groundbreaking ideas to collaborate on clinical best practices. I encourage everyone to keep up the extraordinary accomplishments in the medical field and further augment contributions to the upcoming releases of HealthNews Digest.

A Rare Case of Supraglottic Fibroepithelial Polyp removed by Endoscopic Laryngeal Surgery at Aster Hospital, Muhaisnah

Dr. Akash Abdul Rasheed
ENT, Head and Neck Surgery (Specialist)

PRESENTATION

- 34 year old female
- History of Paroxysmal Nocturnal Dyspnea
- Recurrent cough, not responding to medications
- Change in voice
- No family history of medical illness

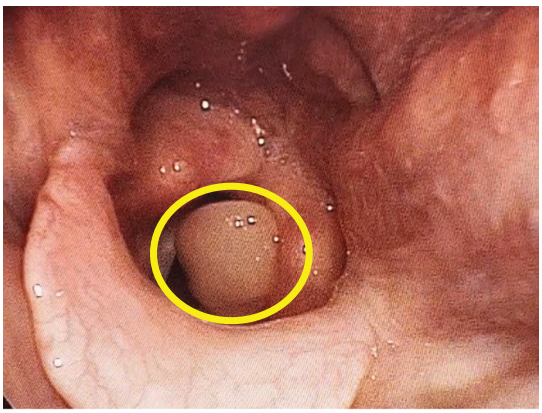
FINDINGS

During Examination:

- Conscious and oriented
- Afebrile
- Neck - Laryngeal crepitus+
- Hoarseness of voice

Laryngoscopy:

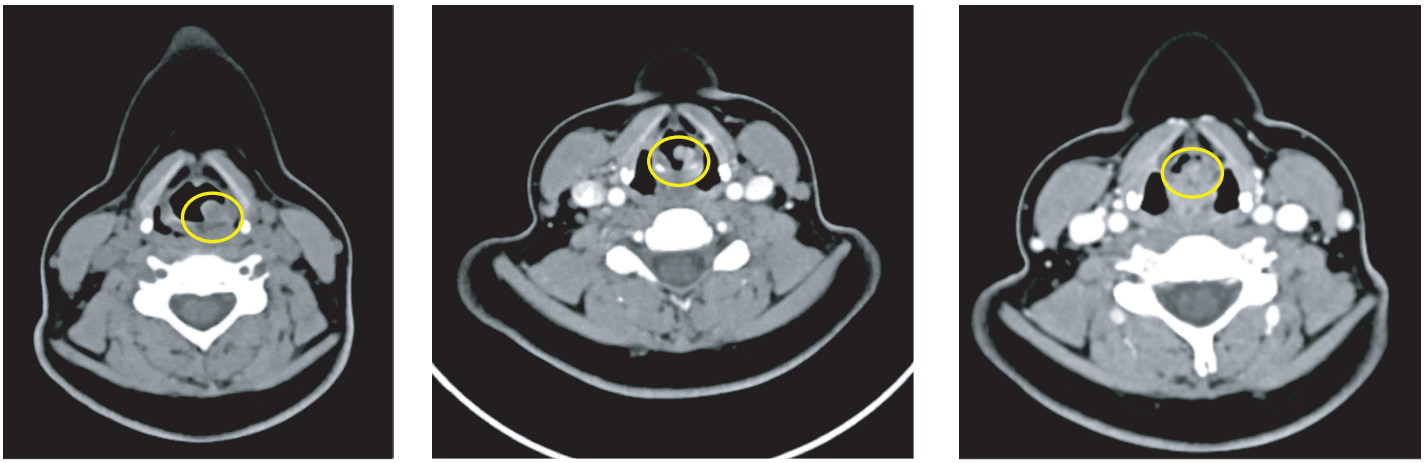
- Cyst/polyp like smooth mass was seen obscuring almost the whole laryngeal inlet (minimal gap was seen)
- Cyst arising from the left inner margin of the aryepiglottic fold
- Extending into the glottic area



Endoscopy Image

CT Neck:

- The aryepiglottic folds towards the midline showed a low-density (HU 30) non-enhancing lesion measuring 6.8 x 6.0 mm, narrowing the airway.
- No evidence of extension into adjacent structures.

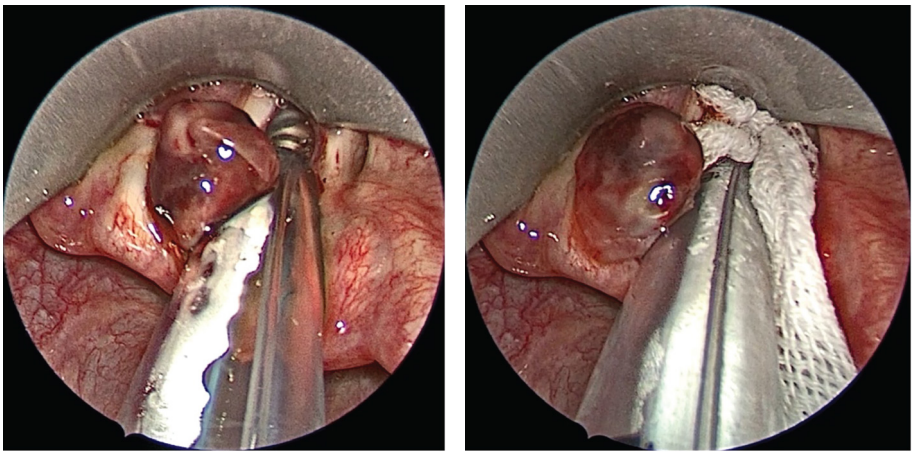


CT Images

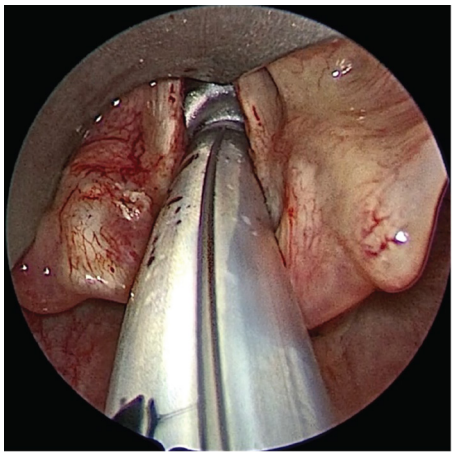
DURING PROCEDURE

The patient was admitted for Endoscopic Laryngeal Surgery under general anaesthesia:

- Patient was put in a supine position.
- Laryngoscope was introduced into the larynx and stabilised with a chest piece.
- Endoscope was introduced, and the larynx was visualised.
- Left aryepiglottic fold mass was seen and removed completely.
- Stalk area was cauterised, and haemostasis was achieved.



Endoscopic Images



Post Excision and Cauterisation

FOLLOW UP

On follow-up after 1 month, the patient was completely relieved from the symptoms. Endoscopy showed no presence of cyst/polyp.

DISCUSSION

A fibroepithelial polyp is a benign lesion of mesodermal origin and is one of the most common cutaneous lesions but is rarer in the head-and-neck region. Fibroepithelial polyps in the head-and-neck region are documented in the external auditory canal, nasal cavity, oropharynx, epiglottis, hypopharynx, trachea, and bronchus. Here, we discuss a rare case of fibroepithelial polyps of the Supraglottis obscuring laryngeal inlet (1).

The aetiology of fibroepithelial polyps is unknown. There are a few theories regarding the cause of these tumours:

- The first is a theory of development secondary to focal losses of elastic tissue; however, there is inadequate proof to support these hypotheses.
- The second theory is that a fibroepithelial polyp is a mixture of different tissue elements, which could represent a slowly enlarging hamartoma of the lamina propria or a fibroma that exhibits the features of a benign lesion (2).

Although usually not life threatening, fibroepithelial polyps of the pharynx may present as an acute medical emergency, causing upper airway obstruction. Management involves securing the airway first (3).

The present case emphasises that fibroepithelial polyp should be included as one of the possible differential diagnoses in patients with recurrent cough that does not respond to medications.

REFERENCES

1. Kaipuzha RR, Pulimoottil DT, Bakshi SS, Gopalakrishnan S. Fibroepithelial Polyps of the Head and Neck. J Dent Allied Sci 2018;7:88-90.
2. B. Janjatov, M. Erić, D. Šojić. An unusual bilateral fibroepithelial pharyngeal polyps: report of a case. European Review For Medical and Pharmacological Sciences. 2012; 16:701-703.
3. A Farboud, A Trinidade, M Harris, A Pfleiderer. Fibroepithelial polyp of the tonsil: case report of a rare, benign tonsillar lesion. The Journal of Laryngology & Otology, Volume 124, Issue 1, January 2010, pp. 111 - 112.
4. Farzal Z, Ulualp SO, Rakheja D. Fibroepithelial Polyp of the Epiglottis. Am J Case Rep. 2014; 15: 340–342. PMID: 25136930.
5. Jabbour J, Chappell JR, Busby M, et al. Glottic Obstruction from Fibroepithelial Polyp. Am J Case Rep. 2019; 20: 219–223. PMID: 30778021
6. Dabholkar JP, Chhabria S, Mishra M, et al. Benign laryngopharyngeal lesions: a case series. Indian J Otolaryngol Head Neck Surg 2008 Mar;60 (1): 7-10. PMID: 23120489.

The Essentials of Idiopathic Sudden Sensorineural Hearing Loss (ISSNHL): Diagnosis, Treatment, and Management Strategies)

Dr. Amjed Almufty
ENT Surgery (Consultant)
Aster Hospital, Sharjah

INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is generally considered as a rapid decline, unilateral deterioration of hearing occurring over 24 to 72 hours, with a reduction of at least 30 dB across three or more adjacent frequencies on a pure-tone audiogram (1). The causes of SSNHL include infections, head trauma, autoimmune diseases, blood circulation problems, multiple sclerosis, and Meniere's disease (2). However, the majority of SSNHL cases are idiopathic, with a specific cause identified in fewer than 30% of patients (1). The annual prevalence of idiopathic SSNHL (ISSNHL) is reported between 5 and 27 per 100,000 people (3). Risk factors for ISSNHL include cigarette smoking, hypertension, and hyperlipidemia (4). Diagnosing ISSNHL involves a detailed medical history, physical examination, blood tests, and radiological and audiological evaluations to rule out various possible etiologies (5). The recommended treatment for ISSNHL includes corticosteroids, such as oral prednisone or intra-tympanic (IT) steroid injections, as the standard treatment for ISSNHL (6). Most individuals with ISSNHL will experience some degree of recovery, although a small percentage may have symptoms that worsen (6). If hearing loss persists after treatment, hearing aids, implantable devices, or assistive listening technology can help in management (6).

This article briefly discusses the diagnosis, treatment, and management of ISSNHL.

DIAGNOSIS OF ISSNHL

Diagnosing ISSNHL requires a comprehensive approach to precisely identify the condition and determine the main cause (7). Diagnostic methods for ISSNHL are outlined below:

1. Clinical Evaluation:

- **Medical History of Patient**

It consists of a detailed history of the onset, duration, and progression of hearing loss, along with any associated symptoms such as tinnitus, vertigo, or ear fullness (7). It is also essential to review the patient's history, including previous hearing issues, exposure to loud noise usually and at jobs without ear protection, daily habit of smoking and alcohol consumption, and any family history of hearing loss (7).

- **Physical Examination and Laboratory Investigation of Patient**

An otoscopic examination of ear canal and eardrum is conducted to detect any external or middle ear conditions, like infections, wax accumulation, or a perforated eardrum, that may lead to hearing loss (7). Additionally, laboratory tests such as complete blood count, coagulation profile, serum electrolyte levels, and erythrocyte sedimentation rate may be conducted to help rule out underlying infections, autoimmune diseases, or metabolic abnormalities (7).

2. Imaging Techniques:

- **Magnetic Resonance Imaging (MRI)**

MRI of the internal auditory canal, brainstem, and adjacent structures is crucial for excluding tumors like acoustic neuromas and other intracranial abnormalities that might be contributing to hearing loss (7,8). MRI offers detailed images of soft tissues, making it particularly effective for identifying subtle changes that other imaging methods may overlook (7,8).

3. Audiological Tests:

- **Pure-Tone Audiometry**

Pure-tone audiometry can be classified as either screening or threshold search (9). Screening audiometry consists of delivering tones within the speech range (500 to 4,000 Hz) at the upper thresholds of normal hearing levels (15 to 20 dB for children and 25 to 30 dB for adults) (9). During the test, the patient listens to tones at various volumes and indicates when they can hear them (9). ISSNHL is typically diagnosed when there is a sudden hearing loss of >30 dB or across at least three consecutive frequencies, indicating sensorineural hearing loss (9).

- **Tympanometry**

This test evaluates middle ear function and the mobility of the tympanic membrane (2). It is frequently used in clinical settings to identify otitis media with effusion and eustachian tube dysfunction (2). Additionally, it can assess the acoustic stapedial reflex, with the acoustic reflex threshold being the lowest sound intensity that triggers this reflex (2).

- **Otoacoustic Emissions (OAEs)**

OAEs are sounds produced by the inner ear in respone to auditory stimuli (2). The presence or absence of OAEs can help distinguish between sensorineural and conductive hearing loss (2).

- **Electrophysiological Tests**

Auditory brainstem testing evaluates nervous system activity and can be influenced by cerebellopontine angle tumors compressing the cochlear nerve and neural demyelination (2). It is also used to estimate hearing thresholds in infants (2).

- **Speech Audiometry**

This test is crucial for evaluating how hearing loss affects communication (2).

Additionally, the AAOHNS guidelines (2019) on evidence-based diagnostic approaches for SSNHL for HCPs are illustrated in Figure 1 (10).

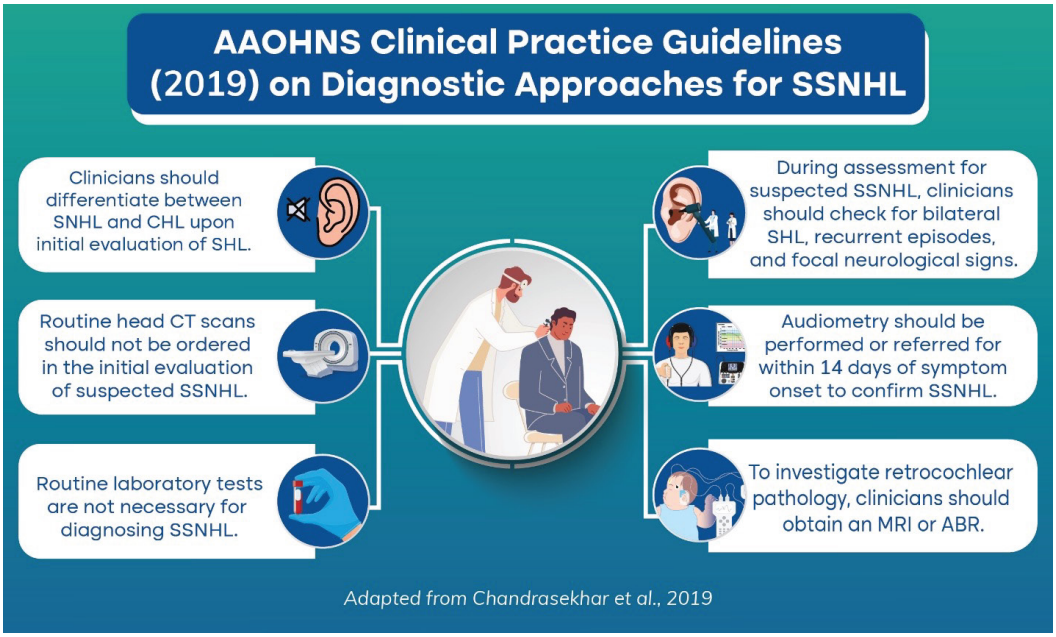


Figure 1: Different Diagnostic Approaches for SSNHL (10)

(Abbreviations: AAOHNS - American Academy of Otolaryngology Head and Neck Surgery, SNHL - Sensorineural Hearing Loss, CHL - Conductive Hearing Loss, CT - Computed Tomography, SSNHL - Sudden Sensorineural Hearing Loss, SHL - Sudden Hearing Loss, MRI - Magnetic Resonance Imaging, ABR - Auditory Brainstem Response)

TREATMENTS OPTIONS FOR ISSNHL

The various treatments for ISSNHL are outlined below.

1. Systemic Corticosteroids:

Corticosteroids, particularly prednisone, are the most commonly prescribed treatment for ISSNHL, as they help reduce inflammation and swelling in the cochlea, potentially aiding in hearing recovery (7). According to the AAOHNS guidelines (2019), oral corticosteroids are recommended as the primary treatment for ISSNHL (10). It is essential to start treatment promptly, as it is most beneficial when initiated within the first two weeks after symptoms begin (7).

2. Intratympanic (IT) Steroids:

IT steroids injections, performed using a 22 to 27-gauge spinal needle, are the most common and convenient method employed (7). Various drug concentrations have been utilized, including dexamethasone at 4mg/ml, 5mg/ml, 10mg/ml, 24mg/ml, and 40mg/ml; prednisolone at 62.5mg/ml; and methylprednisolone at 30mg/ml, 40mg/ml, 62.5mg/ml, and 80mg/ml (7). 0.4-0.8ml of the solution is administered intratympanically, with frequencies ranging from daily (up to eight sessions), every other day, three times a week, twice a week, to weekly injections for 3-4 sessions (7). IT steroid therapy has shown a maximum recovery rate of 91% in patients (7).

3. Hyperbaric Oxygen Therapy (HBOT):

HBOT involves inhaling pure oxygen within a room or chamber under increased pressure (7). This increases oxygen delivery to the inner ear, promoting healing and recovery (7). HBOT is usually administered as a series of sessions over several days or weeks and can be combined with steroid therapy to enhance its effectiveness (7).

4. Neuro-rehabilitation Therapy:

Neuro-rehabilitation therapy focuses on the recovery of auditory function through various exercises and auditory training (7). This might include the use of hearing aids, balance training, and auditory training exercises in cases of severe or persistent hearing loss (7).

5. Other Treatment Modalities:

Several other treatment options are available for ISSNHL (2,7). Anti-inflammatory drugs (AIDs), such as naproxen and ibuprofen, are being considered to reduce inner ear inflammation and support recovery (2,7). Antimicrobials like amoxicillin or ciprofloxacin may be prescribed if an infectious cause is suspected (2,7). Calcium antagonists, including nimodipine and verapamil, can potentially aid the recovery process by improving blood flow to the inner ear (2,7). High-dose vitamins, such as vitamin C, along with essential minerals like zinc and magnesium, are used to promote overall ear health and healing (2,7). Antioxidant supplements, like N-acetyl-cysteine and alpha-lipoic acid, may help safeguard the inner ear from oxidative damage, which is thought to contribute to ISSNHL (2,7). Additionally, vasodilators and vasoactive substances like pentoxifylline and betahistine can enhance blood circulation to the cochlea, potentially improving recovery outcomes (2,7).

The strategy for effectively managing ISSNHL is illustrated in Figure 2.

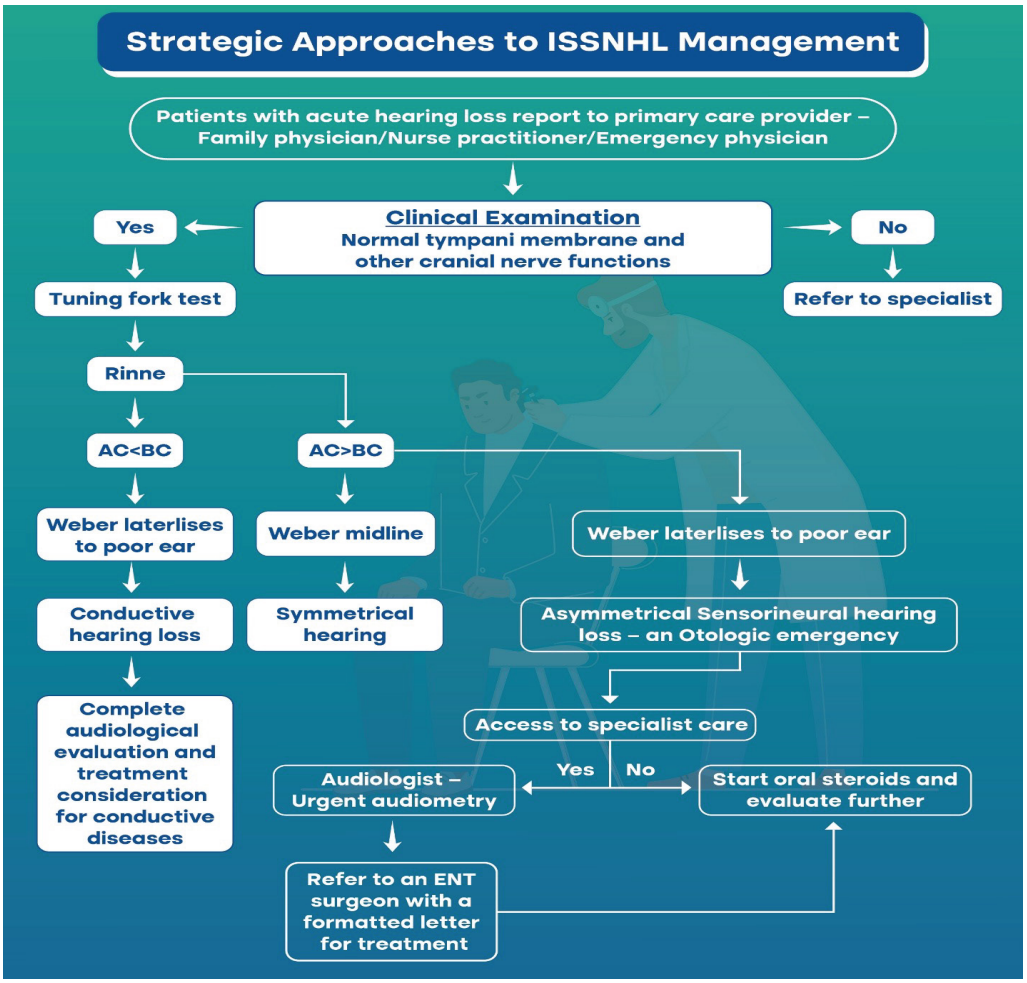


Figure 2: Management of ISSNHL (11)

(Abbreviations: ISSNHL - Idiopathic Sudden sensorineural hearing loss, AC - Air Conduction, BC - Bone Conduction, ENT - Ear Nose Throat)

Key Highlights

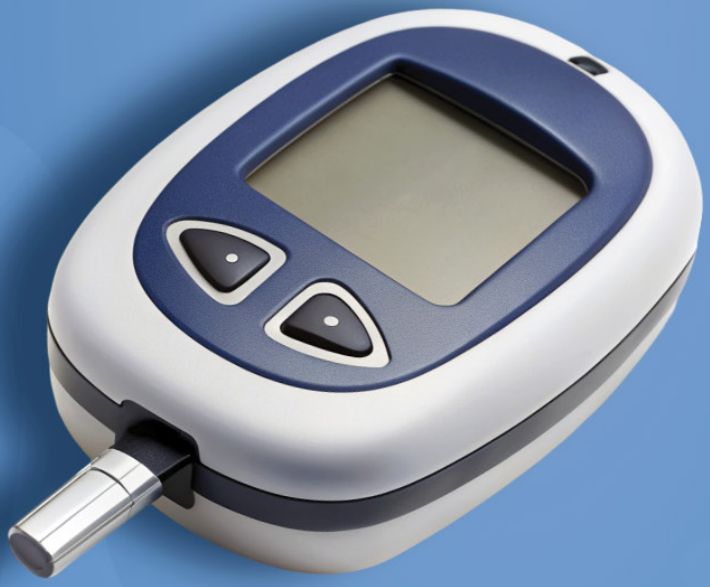
- ISSNHL is a rapid onset, unilateral hearing loss occurring over 24 to 72 hours, characterized by a decrease of 30 dB or more across at least three contiguous frequencies (1).
- Diagnosis of ISSNHL involves complete clinical history, physical examination, routine laboratory tests, imaging (such as MRI), and various audiological tests (7).
- The American Academy of Audiology and AAOHNS recommends corticosteroids, such as oral prednisone or intra-tympanic (IT) steroid injections, as the most common treatment (6,10).

REFERENCES

1. Alexander TH, Harris JP. Incidence of sudden sensorineural hearing loss. Otol Neurotol [Internet]. 2013 Dec;34(9):1586–9. Available from: https://pubmed.ncbi.nlm.nih.gov/24232060/
2. NIDCD. Sudden Sensorineural Hearing Loss (SSHL) [Internet]. 2018. Available from: https://www.nidcd.nih.gov/health/sudden-deafness
3. Leung MA, Flaherty A, Zhang JA, Hara J, Barber W, Burgess L. Sudden Sensorineural Hearing Loss: Primary Care Update. Hawaii J Med Public Health [Internet]. 2016 Jun;75(6):172–4. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4928516/
4. Ciorba A, Hatzopoulos S, Bianchini C, Iannini V, Rosignoli M, Skarzynski H, et al. Idiopathic sudden sensorineural hearing loss: cardiovascular risk factors do not influence hearing threshold recovery. Acta Otorhinolaryngol Ital [Internet]. 2015 Apr;35(2):103–9. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4443566/
5. Binnetoglu A, Yumusakhuylu AC, Demir B, Baglam T, Derinsu U, Sari M. Association between Family History and Idiopathic Sudden Sensorineural Hearing Loss. Int Adv Otol [Internet]. 2015 Jul 7;11(1):30–2. Available from: https://advancedotology.org//en/association-between-family-history-and-idiopathic-sudden-sensorineural-hearing-loss-13815
6. AAA. Sensorineural Hearing Loss [Internet]. Available from: https://www.audiology.org/consumers-and-patients/hearing-and-balance/sensorineural-hearing-loss/#:~:text=Sudden%20sensorineural%20hearing%20loss%20is%20a%20serious%20condition%20and%20should,and%20Throat%20physician%20is%20necessary.
7. Singh A, Kumar Irugu DV. Sudden sensorineural hearing loss - A contemporary review of management issues. J Otol [Internet]. 2020 Jun;15(2):67–73. Available from: https://pubmed.ncbi.nlm.nih.gov/32440269/
8. Clyde JW, Patel VA, Kanekar S, Isildak H. Magnetic resonance imaging findings in idiopathic sudden sensorineural hearing loss. Acta Radiol [Internet]. 2019 Sep;60(9):1167–74. Available from: https://pubmed.ncbi.nlm.nih.gov/31392900/
9. Walker JJ, Cleveland LM, Davis JL, Seales JS. Audiometry screening and interpretation. Am Fam Physician [Internet]. 2013 Jan 1;87(1):41–7. Available from: https://pubmed.ncbi.nlm.nih.gov/23317024/
10. Chandrasekhar SS, Tsai Do BS, Schwartz SR, Bontempo LJ, Faucett EA, Finestone SA, et al. Clinical Practice Guideline: Sudden Hearing Loss (Update). Otolaryngol Head Neck Surg [Internet]. 2019 Aug;161(1_suppl):S1–45. Available from: https://pubmed.ncbi.nlm.nih.gov/31369359/
11. Ganesan P. Challenges in Diagnosing Sudden Sensorineural Hearing Loss. The Hearing Journal [Internet]. 2022 Jun;75(6):6–8. Available from: https://journals.lww.com/10.1097/01.HJ.0000833456.10864.09

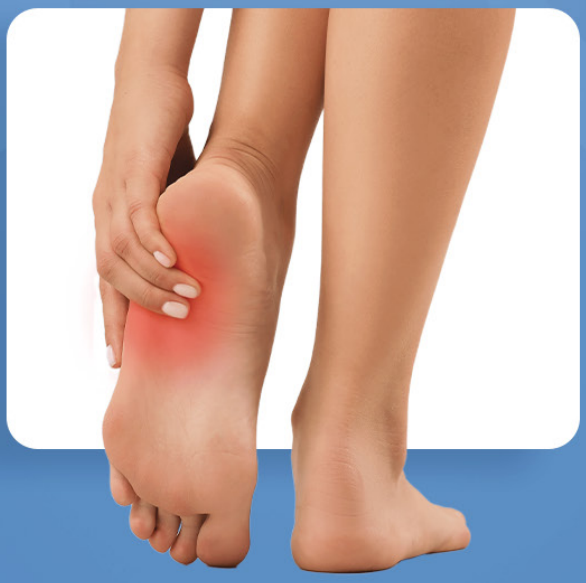
Aster
HOSPITAL
We'll Treat You Well

Diabetes isn't sweet for your feet



So, if you are sensing the symptoms, visit our

DIABETIC FOOT CLINIC



Wondering what's
diabetic foot?

It's a complication where high blood sugar levels cause **nerve damage and poor blood circulation in the feet**

Watch out for these Symptoms

- **Numbness or tingling in the feet**
- **Foot pain or cramping**
- **Open sores or ulcers that heal slowly**

Experiencing any of these symptoms?

Visit us at Aster Hospital Mankhool

Diabetic Foot Disease: A Silent Killer Among Diabetics!

Complex cases of Diabetic Foot Ulcer managed successfully at Aster Hospital, Mankhool

Dr. Roshan Rodney Sengodan
Vascular, Endovascular and
Diabetic Foot Surgery (Specialist)

PRESENTATION

Case 1

- 65 year old male
- Medical history of:
  - Diabetes Mellitus for 10 years, on oral antidiabetics but not on regular follow-up
  - Non-healing callus ulcer in the right foot for a year
  - Decreased sensation in both feet for many years
- Surgical history of Left Hip Surgery a long time back because of a fall, implant in situ
- No significant family history of similar medical illness
- Admitted with:
  - Complaints of worsening swelling, discolouration, and pain in the right foot for the last 5 to 6 days

Case 2

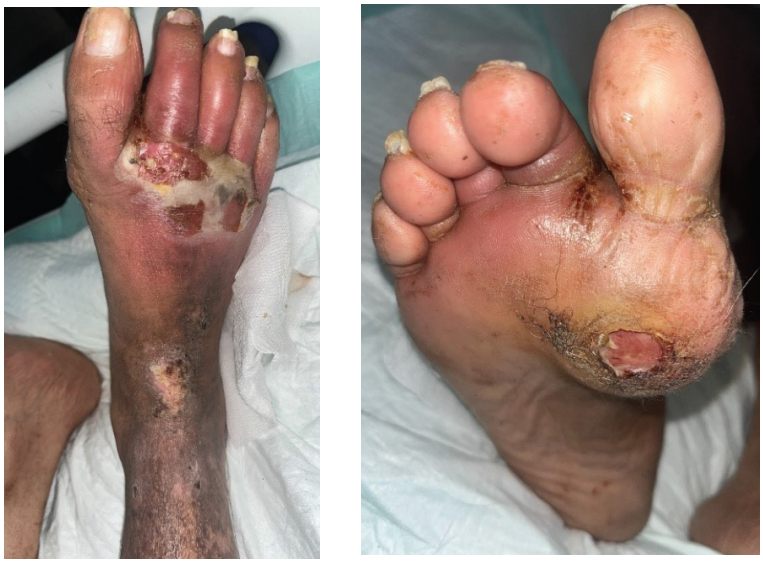
- 49 year old male
- Medical history of:
  - Diabetes Mellitus
  - Ischaemic Heart Disease (IHD) - status post Coronary Artery Bypass Graft (CABG) procedure
- No significant family history of similar medical illness
- Admitted with:
  - Complaints of non-healing ulcer in the left foot for the past 3 months

FINDINGS

During Examination:

Case 1

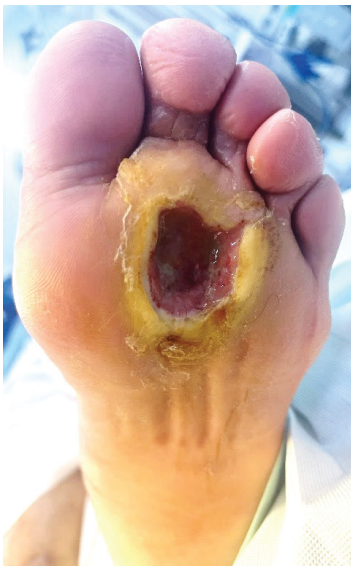
- Swollen right forefoot and red up to midfoot both in plantar and dorsal aspects.
- Right forefoot dorsum and second toe were necrotic and wet gangrenous with a necrotic base of other lesser toes in the dorsal aspect.
- Plantar callus ulcer corresponding to the presence of the first metatarsal head.
- Distal pulses palpable in both lower limbs.
- No ulcers in the left lower limb.



Pre-operative Images

Case 2

- An ulcer of the size of around 3x3 cm in the left forefoot plantar region corresponding to the second and third metatarsal heads.
- Superficial slough present.
- No active pus discharge.
- Palpable Dorsalis Pedis Artery (DPA) and Posterior Tibial Artery (PTA).



Pre-operative Image

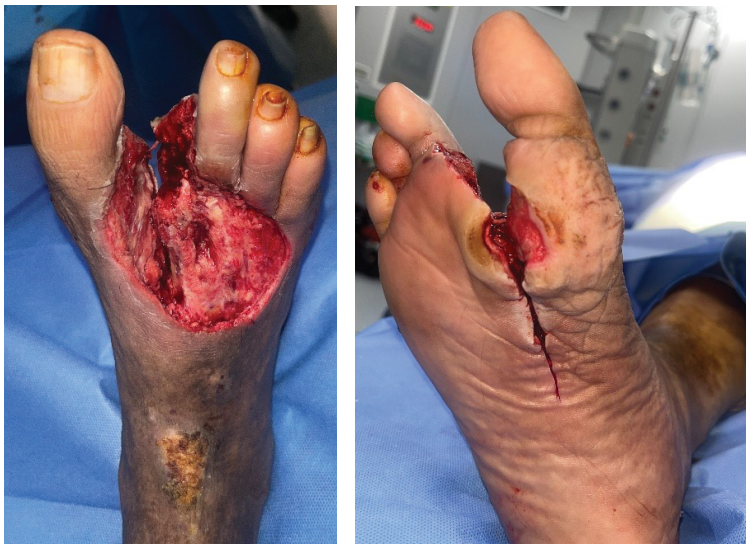
DURING PROCEDURE

Both patients were diagnosed with Diabetic Foot and were admitted for further management:

Case 1

The patient underwent **Right Foot Ulcer Extensive Debridement with 2nd Toe Amputation:**

- The patient was treated with culture-specific IV antibiotics, and diabetic control was achieved.
- Regular daily wound care and dressings were done.
- His wound became better, and he started developing healthy granulation tissue.

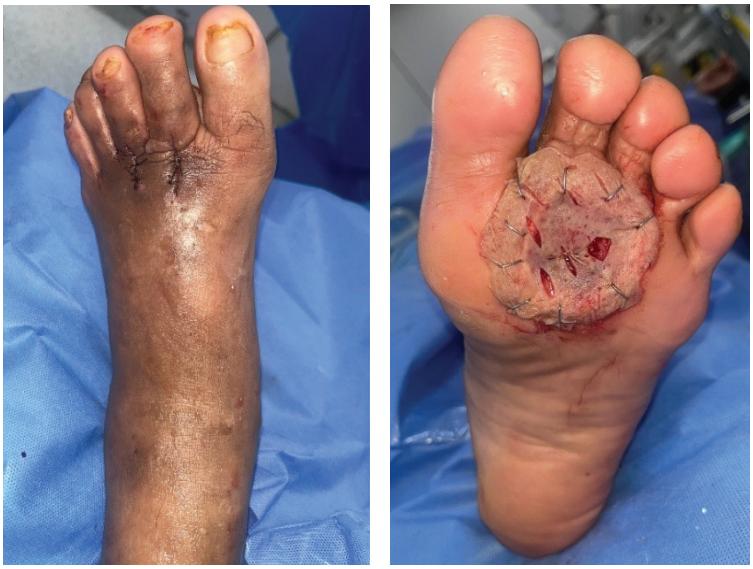


Intra-op Images

Case 2

The patient underwent **Left 2nd and 3rd Toe Metatarsal Head Weil Osteotomy with Foot Ulcer Debridement and Split Skin Grafting:**

- Left foot plantar ulcer base necrotic tissue and muscle were thoroughly debrided with sharp scissors, ulcer bed was prepared, and wound wash was given.
- Two dorsal foot incisions were made corresponding to 2nd and 3rd toe metatarsal heads, and Weil metatarsal osteotomy was done using an electric saw.
- Skin incision was closed with 2-0 Ethilon after achieving haemostasis.
- Split skin graft of around 4x4 cm in size was harvested from the left thigh, meshed and fixed to the ulcer bed with staplers.
- Sterile dressing and posterior slab were applied.



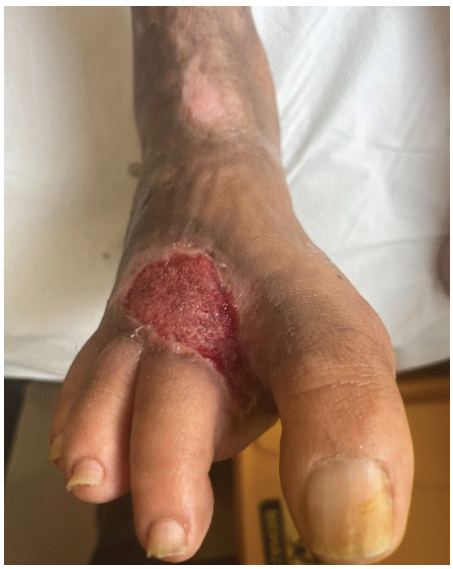
Intra-op Images

POST PROCEDURE

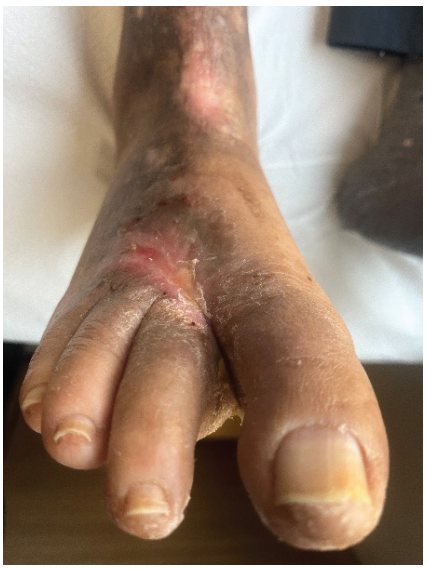
Both patients withstood the procedure well and were stable at the time of discharge. They were advised for non-weight bearing on their respective treated foot, to use the support of Walker / Ankle foot Orthoses (AFO) for additional protection. Daily wound dressing is required for the next 1-2 months until complete healing.

FOLLOW-UP

Case 1

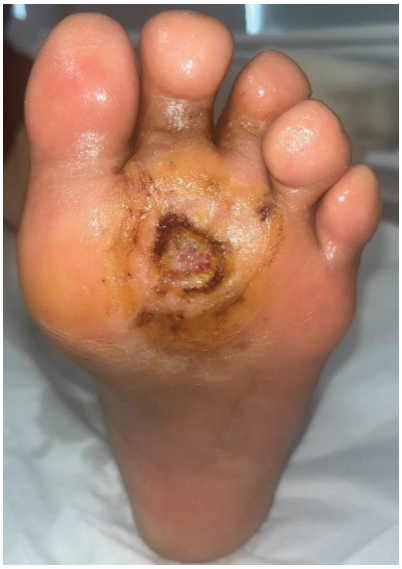


Month 1



Month 2

Case 2



Week 3

DISCUSSION

Diabetic Foot Surgeries are classified into the following four categories:

Class I: Elective

Reconstructive procedures are done on patients who do not have a loss of protective sensation (LOPS).

Class II: Prophylactic

Reconstructive procedures are performed to reduce the risk of ulceration or re-ulceration in patients who have LOPS and do not have a wound present.

Class III: Curative

Procedures performed to assist in the healing of open wounds.

Class IV: Emergent

Procedures performed to arrest or limit the progression of infection

The majority of diabetic patients visit the hospitals either in Class IV or Class III stages, as seen in the above cases. Surgery is only a part of the management of patients with diabetic foot ulcers.

These patients need a holistic approach of a dedicated multidisciplinary team which includes Diabetologists (Physicians and Endocrinologists), Diabetic Foot / Podiatric Surgeons (Vascular Surgeons, Plastic Surgeons, Orthopaedic Surgeons, and General Surgeons), Podiatrists and wound care certified Nurses.

REFERENCES

1. Hingorani A, LaMuraglia G M, Henke P, et al. The management of diabetic foot: A clinical practice guideline by the Society for Vascular Surgery in collaboration with the American Podiatric Medical Association and the Society for Vascular Medicine. Journal of Vascular Surgery. Volume 63, Issue 2, Supplement, 3S-21S, February 2016.
2. Sabapathy S R, Periasamy M. Healing ulcers and preventing their recurrences in the diabetic foot. Indian J Plast Surg. 2016 Sep-Dec; 49(3):302-313. PMID: 28216809. DOI: 10.4103/0970-0358.197238.

Impact of Systemic Conditions on Retinal Health

Dr. Aaliya Ambereen
Ophthalmology (Specialist)
Aster Hospital, Muhaisnah

INTRODUCTION

Several disorders affecting systemic health can have ocular manifestations (1). The eye is considered as an extension of the central nervous system (CNS), which makes it sensitive to changes in the CNS (2). Moreover, direct ocular involvement is seen in various vascular, inherited, autoimmune, oncologic and genetic diseases (3). These ocular manifestations may occur either as primary symptoms or secondary to the underlying systemic disease (4). Symptoms generally include floaters, blurred vision, peripheral vision loss, and progression to complete blindness (5). Routine ophthalmic examination including slit-lamp examination & fundoscopy along with investigations like optical coherence tomography can be used to identify retinal manifestations (4,6). It is important to diagnose and treat these manifestations to reduce their severity (1).

This article provides an overview of different systemic diseases that can impact the retina and their management options.

MECHANISM ON HOW SYSTEMIC DISEASE AFFECTS RETINAL HEALTH:

Systemic diseases can show ocular manifestations through various mechanisms: retinal vasculature mimics systemic circulation, an increase in blood pressure 10 mmHg narrows the retinal vessels by 3µm (7). This leads to hypertensive retinopathy which causes haemorrhage, oedema etc. (8). In diabetes, the retinal arterioles show impaired myogenic constriction when blood glucose spikes, chronic diabetes causes permanent remodeling/ dilation of retinal arterioles and veins leading to blurred vision or complete vision loss (7). Inheritable cancer syndromes like retinal hemangioblastomas and suppressed activity of tumour infiltrating lymphocytes lead to ocular malignancy and progressive vision loss (9).

Several autoimmune diseases produce autoantibodies affecting various regions of the body (10). Antinuclear antibodies in systemic lupus erythematosus deposits in the eyes leading to optic neuritis, occlusion and blindness (10). Spread of systemic infections to the eye causes ocular inflammation and autoreactive T cells that bring about macrophage activation further damaging the eye (11).

TYPES OF SYSTEMIC CONDITIONS THAT AFFECT RETINAL HEALTH:

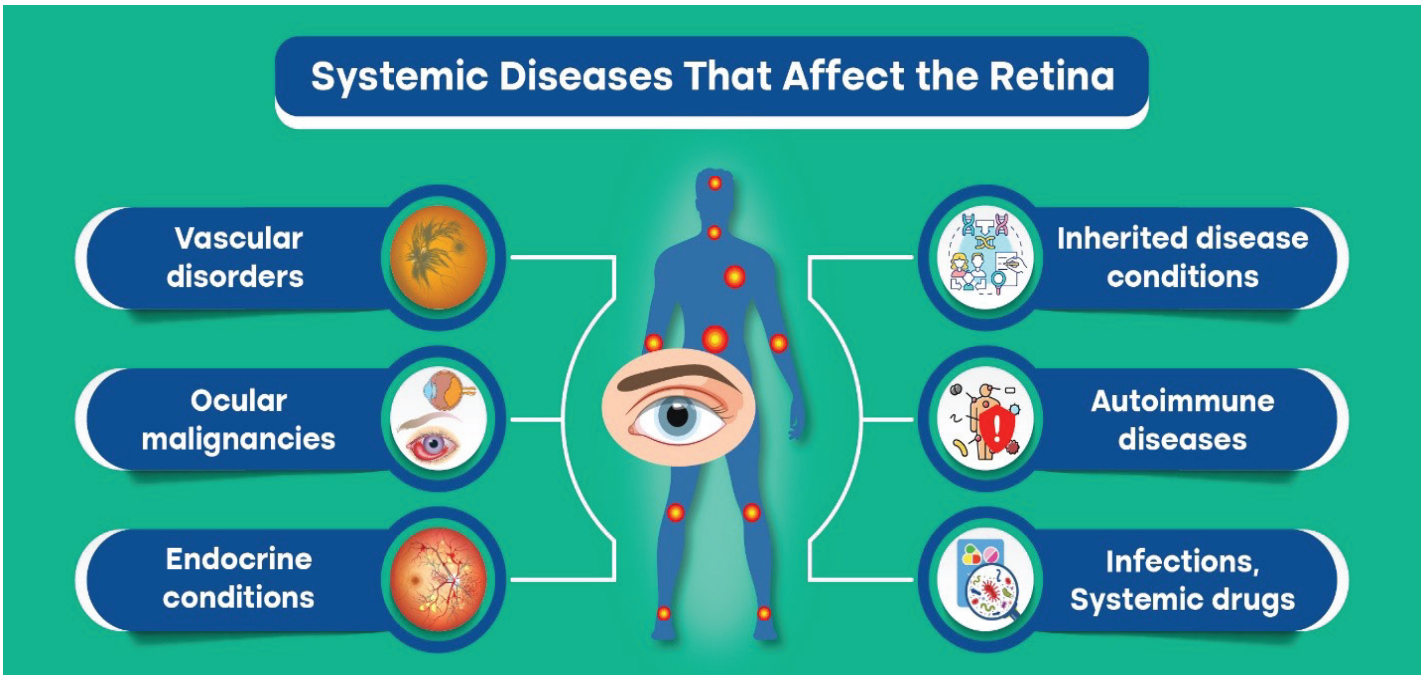


Figure 1: Effect of Systemic Conditions on The Retina

1. Vascular Diseases:

a) Hypertension:

Systemic hypertension can significantly affect the retinal microvasculature. Persistent hypertension leads to remodeling of the retinal vasculature, resulting in retinal haemorrhage, arteriovenous nipping, narrowing and microaneurysms. In some cases, this can also cause edema of the optic disc and macula (8). The retinal haemorrhage appears as dots and flame-shaped lesions, while cotton wool-type exudates may develop due to ischemia of the nerve fibres (12). Uncontrolled hypertension is a major risk factor for diabetic retinopathy (12).

Treatment:

Management and control of hypertension is the only way to treat mild-to-moderate retinopathy. Intravitreal corticosteroid and anti-vascular endothelial growth factor (anti-VEGF) agents may be used in severe cases (12). Urgent treatment is necessary due to its high mortality (12).

b) Ophthalmic Emboli and Central Retinal Vein Occlusion:

Retinal emboli can develop due to ipsilateral carotid lesions, atherosclerotic plaque or stenosis, hypertension, smoking and in some rare cases, diabetes (13). Retinal arteriolar emboli affect 1% of the adult population with a predominance in men (14). The presence of multiple emboli within a single eye may lead to retinal arterial occlusion and loss of vision (13). Additionally, indirect embolism stemming from haemodynamic changes can raise debris, leading to emboli formation and subsequent retinal occlusion (13).

Treatment:

Vasodilators like pentoxyphylline are used in peripheral vascular disease to improve tissue perfusion by reducing blood viscosity, and improving erythrocyte flexibility (15).

Inhalation of carbogen, a mixture of 95% oxygen and 5% carbon dioxide, is thought to prevent vasoconstriction caused by oxygen, thereby improving blood flow and oxygenation of the retina (15). This treatment, given at 10 minutes every hour after waking up and 10 minutes every 4 hours at night for 48-72 hours, can improve retinal vein occlusion (15). Hyperbaric oxygen can also be beneficial to improve the emboli (15).

Compression of the globe with ocular massage, using either a three-mirror contact lens or digital massage over closed eyelids, helps to mechanically disintegrate and dislodge the emboli (15). Ocular massage can be combined with acetazolamide to further improve the treatment effect (15).

2. Ocular Malignancies:

a) Retinoblastoma:

Retinoblastoma is the most common retinal malignancy affecting approximately 1 in 16,000 children (16). The appearance of a white pupillary reflex or a leukocoria is the first sign of retinoblastoma (16). Non-hereditary tumor presents unilaterally whereas hereditary retinoblastomas show bilateral and multifocal presentation (16).

Treatment:

Chemotherapy has been the standard treatment for retinoblastoma (17). Depending on the stage of presentation chemotherapy generally consists of one, two, or three drugs e.g.: vincristine, carboplatin and etoposide (17). In addition to chemotherapy, other globe-preserving treatments include cryotherapy, transpupillary thermotherapy, external-beam chemoradiotherapy (EBCRT), and brachytherapy (17).

b) Primary vitreoretinal lymphoma:

Primary vitreoretinal lymphoma (PVRL) is a rare type of lymphoma originating in the central nervous system (18). Reported ocular manifestations include complaints of haze and floaters in the vision (19). The infiltrated lymphoma cells manifest as cream-coloured retinal spots or as a 'leopard spot" pattern (18).While ocular imaging is the standard for diagnosing PVRL, it can be difficult to differentiate it from uveitis (18). Despite its slow progression, PVRL carries a significant risk, with 56-90% of patients experiencing central nervous system relapse within 30 months, leading to vision loss and ultimately, death (18).

Treatment:

The treatment focuses on controlling retinal disease and preventing CNS dissemination (18). Local systemic therapy with methotrexate and radiotherapy can be selected to prevent CNS relapse (18). Patients who don't show CNS involvement, are recommended EBCRT of 35–40 Gy delivered in 15 fractions of 2 Gy each (19). Other options include thiotepa-based chemotherapy followed by autologous stem cell transplantation, cytarabine, and pemetrexed, lenalidomide, pomalidomide or ibrutinib (19).

c) Retinal hemangioblastoma:

Retinal hemangioblastomas (RH) occur as a consequence of familial Von Hippel-Lindau (VHL) disease, presenting in up to 45%-60% of patients (9). In some cases, hemangioblastoma may be the only presenting tumour or sign of the disease (9). On fundus examination, these lesions appear as reddish, globular masses with a dilated, tortous draining vein (9). The lesions appear as a pinpoint, vascular tuft or large globular tuft. Despite its slow progression, the lesion can lead to significant vision loss (9).

Treatment:

The efficacy of the treatment depends on the tumour size, relative tumour location, haemorrhage, exudation, epiretinal fibrosis, and prior ocular surgeries (20). Small retinal hemangioblastomas of 1.5 mm diameter can be ablated, and large RH's that do not respond to cryotherapy or laser photocoagulation may be surgically excised (20).

3. Endocrine Conditions:

a) Diabetic retinopathy:

There is a wide spectrum of ophthalmic conditions that affect patients with diabetes mellitus (DM) (5). Several micro- and macrovascular changes occur in DM due to the hyperglycemic state that impairs vascular endothelial cell function leading to abnormal retinal vascularization (5).

Diabetic retinopathy (DR), which occurs in 60-90% of patients with DM due to infarction of the microaneurysms, resulting in cotton-wool spots or flame-shaped haemorrhages (5). Some patients present with bilateral DR with complaints of floaters, decreased visual acuity, partial vision loss, distorted visions, and new-onset flashes (5). In addition, patients with DR also present with macular oedema, cataracts and sixth nerve palsy (5).

Treatment:

Pan retinal photocoagulation (PRP) is the preferred treatment in proliferative stages of DR. Additionally, anti-VEGF agents can be used in the treatment of diabetic macular oedema (5).

4. Inherited Diseases:

a) Wolfram syndrome:

Wolfram syndrome occurs due to mutations of the gene WWFS1 which encodes the protein wolframin involved in calcium homeostasis in the endoplasmic reticulum (5). This protein is found in optic axons, optic nerve and retinal ganglion cells (5).

The initial presentation of this syndrome is bilateral optic nerve atrophy in the first decade of life. The early symptoms are relatively mild, involving loss of colour and peripheral vision, but the condition progresses to complete blindness within 8 years of the initial atrophy diagnosis (5). As the disease progresses, additional ocular manifestations emerge, including retinal thinning, retinal pigmentation, DR, glaucoma, nystagmus (5).

Treatment:

There are currently no curative treatments available for Wolfram syndrome, however, the disease progression can be delayed with idebenone and docosahexaenoic acid (5).

b) Bardet-Biedl syndrome:

Bardet-Biedl syndrome occurs due to mutation in the genes BBS1-BBS20 that encode for ciliary proteins (5). Mutations of these genes cause ciliary protein dysfunction and impair other associated systems (5). Ocular manifestations of Bardet-Biedl syndrome are nyctalopia, retinitis pigmentosa, loss of peripheral vision and complete blindness by the third decade of life (5).

Treatment:

There are no treatments available for Bardet-Biedl syndrome, vitamin A supplement may help to preserve retinal function (5).

5. Autoimmune diseases:

a) Bechet's disease:

One of the features of Bechet's disease is relapsing uveitis, which is characterised by chronic relapsing bilateral non-granulomatous panuveitis and retinal vasculitis (21). It causes inflammation of the branch retinal vein occlusion, which results in retinal non-perfusion, and neovascularisation, which can increase the risk of retinal haemorrhage and loss of vision (21).

Treatment:

Disease management varies depending on which eye structures are affected (21). Treatment involves aggressive treatment with corticosteroids to prevent vision loss, corticosteroids could be combined with cyclosporine and azathioprine (21). For patients who are refractive to immunosuppressants, infliximab and adalimumab can be used as second-line therapy (21).

b) Lupus retinopathy:

About 29% of patients with lupus erythematosus suffer from lupus retinopathy (6). Pathogenesis includes the

deposition of immune complexes, microemboli, intraretinal haemorrhages, and cotton wool spots (6). In acute cases, lupus retinopathy manifests as retinal vasculitis, floaters, visual field distortion, and sudden vision loss (6). Immune complex deposition shows mild retinal vasculopathy and inflammation whereas fibrinoid degeneration is associated with severe vasculopathy, retinal vein occlusion, retinal ischemia, vitreous haemorrhage, and neovascularisations (6).

Treatment:

Retinopathy can be improved with immunosuppressants, a combination of immunosuppressants and anticoagulants can stabilise vascular complications (6). For severe diseases, plasmapheresis and plasma exchange can be used (6). Treatments used in DR can be used for lupus retinopathy (PRP, intravitreal anti-VEGF, and vitrectomy)(6).

c) Multiple sclerosis:

Multiple sclerosis (MS) causes thinning of the retinal fibre layer (RNFL) of the peripapillary (pRNFL) (22). Patients who observe optic neuritis and relapsing-remitting MS show a more progressive thinning of the ganglion cell and inner plexiform layer and retinal ganglion cells (22). The exact pathology behind these neurodegenerative changes is unknown but reduced metabolic activity and vascular dysfunction are presumed (22).

Treatment:

Certain disease-modifying drugs have shown efficacy in retinal thinning (22). Natalizumab shows reduced RNFL thinning while alemtuzumab has demonstrated stable RNFL and GCIPL parameters and rituximab has depicted preserved atrophy of GCIPL (22). Fingolimod shows effect on the visual outcome but is associated with macular oedema (22).

6. Other causes:

a) Infectious diseases:

Infectious diseases like cytomegalovirus, herpes simplex virus, toxoplasmosis, candida, Lyme disease cause infectious retinitis (11). Infections may be due to maternal-fetal transmission, immunosuppressed states and exposure to endemic regions (11). Infections of the eye can cause retinal necrosis and oedema that leads to scarring and subsequent retinal detachment (11). Typical ocular findings of these infections include cotton wool spots, white lesions, retinal vasculitis, and haemorrhage (11).

Treatment:

Adequate treatment of the underlying infectious disease is essential to improve visual outcomes.

b) Systemic drugs:

Retinal folds are seen in sulfa-drugs like diuretics and antibiotics, as well as choroidal effusion and swelling of the ciliary body (23).

Progestins in oral contraceptives may cause occlusive retinopathy, occlusion, retinal oedema, and macular neuroretinopathy (23). Intravitreal aminoglycosides may cause haemorrhage, oedema, cotton wool lesions, retinal vasculitis and retinal thinning (23).

MEK Inhibitors like trametinib, vancomycin, immune checkpoint inhibitors also cause retinal vasculitis (23). Cisplatin and carmustine have shown ocular effects like retinal ischemia and retinal thinning (23).

Treatment:

Drug discontinuation, dose reduction, and appropriate corticosteroids can be used to manage the retinal side effects (23). In some life-saving drug classes discontinuation is not possible and regular monitoring is recommended (23).

Key Highlights

- Systemic disease can directly or indirectly affect the ocular system and retinal health. Ocular manifestations may be the primary symptom or secondary to the disease (4,24).
- Diabetes & chronic hypertension are the most prevalent causes of systemic-ocular manifestation. Ocular malignancies, endocrine disorders, autoimmune effects, and genetically inherited diseases are few other factors affecting retinal health (5,20).
- Ocular effects depend on the underlying disease but typical manifestations include cotton wool lesions, vasculitis, ischemia, occlusion, oedema, atrophy/ detachment of the retina (23).
- Management options comprise symptomatic treatment with corticosteroids, anti-VEGF agents, and surgical treatment as well as effective management of systemic disease (23).

REFERENCES

1. Singh M, Deokar K, Sinha BP, Keena M, Desai G. Ocular manifestations of common pulmonary diseases: a narrative review. Monaldi Arch Chest Dis [Internet]. 2023 Mar 3 [cited 2024 Aug 6];94(1). Available from: https://www.monaldi-archives.org/macd/article/view/2535
2. Marchesi N, Fahmideh F, Boschi F, Pascale A, Barbieri A. Ocular Neurodegenerative Diseases: Interconnection between Retina and Cortical Areas. Cells [Internet]. 2021 Sep 1 [cited 2024 Aug 16];10(9):1–15. Available from: https://www.mdpi.com/2073-4409/10/9/2394
3. Roszkowska AM, Fogagnolo P, Neri P. Editorial: Eye in systemic diseases. Front Med [Internet]. 2023 Mar 24 [cited 2024 Aug 6];10:1–12. Available from: https://www.frontiersin.org/journals/medicine/articles/10.3389/fmed.2023.1171238/full
4. Nowinska AK, MacHalińska A, Módis L, Koprowski R, Rechichi M. Ocular Manifestations of Systemic Diseases. J Ophthalmol [Internet]. 2018 [cited 2024 Aug 16];2018:1–6. Available from: https://onlinelibrary.wiley.com/doi/10.1155/2018/7851691
5. Lause M, Kamboj A, Faith EF. Ophthalmic manifestations of endocrine disorders—endocrinology and the eye. Transl Pediatr [Internet]. 2017 Oct 1 [cited 2024 Aug 14];6(4):28699–28299. Available from: https://tp.amegroups.org/article/view/16985/html
6. Palejwala N V., Walia HS, Yeh S. Ocular Manifestations of Systemic Lupus Erythematosus: A Review of the Literature. Autoimmune Dis [Internet]. 2012 [cited 2024 Aug 14];2012(1):1–9. Available from: https://onlinelibrary.wiley.com/doi/10.1155/2012/290898
7. Hanssen H, Streese L, Vilser W. Retinal vessel diameters and function in cardiovascular risk and disease. Prog Retin Eye Res [Internet]. 2022 Nov;91:1–26. Available from: https://linkinghub.elsevier.com/retrieve/pii/S1350946222000556
8. Bhargava M, Ikram MK, Wong TY. How does hypertension affect your eyes? J Hum Hypertens 2012 262 [Internet]. 2011 Apr 21 [cited 2024 Aug 6];26(2):71–83. Available from: https://www.nature.com/articles/jhh201137
9. Dollfus H, Massin P, Taupin P, Nemeth C, Amara S, Giraud S, et al. Retinal hemangioblastoma in von Hippel-Lindau disease: a clinical and molecular study. Invest Ophthalmol Vis Sci [Internet]. 2002 Sep;43(9):3067–74. Available from: https://iovs.arvojournals.org/article.aspx?articleid=2162471
10. K Kaviarasan P. Exigency of ocular complications of systemic lupus erythematosus. IP Indian J Clin Exp Dermatology [Internet]. 2020 Jun 28 [cited 2024 Aug 14];6(2):105–12. Available from: https://ijced.org/article-details/11548
11. Gupta Nikita TK. Retinitis - StatPearls - NCBI Bookshelf [Internet]. 2024 [cited 2024 Aug 15]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560520/
12. Shaheen AR, Sridhar J. Hypertensive Retinopathy. Retin Choroidal Vasc Dis Eye [Internet]. 2023 Jul 4 [cited 2024 Aug 7];427–36. Available from: https://www.ncbi.nlm.nih.gov/books/NBK525980/
13. Chen SN, Hwang JF, Huang J, Wu SL. Retinal arterial occlusion with multiple retinal emboli and carotid artery occlusion disease. Haemodynamic changes and pathways of embolism. BMJ Open Ophthalmol [Internet]. 2020 Jul 27 [cited 2024 Aug 7];5(1):ep000467. Available from: https://bmjophth.bmj.com/content/5/1/e000467

14. Kadonosono K, Inoue M, Yanagi Y. Retinal arterial and vein occlusion: is surgery ever indicated? Curr Opin Ophthalmol [Internet]. 2024 May 1 [cited 2024 Aug 7];35(3):210–6. Available from: https://journals.lww.com/10.1097/ICU.0000000000001045
15. Cugati S, Varma DD, Chen CS, Lee AW. Treatment Options for Central Retinal Artery Occlusion. Curr Treat Options Neurol [Internet]. 2013 Feb [cited 2024 Aug 7];15(1):63–77. Available from: https://link.springer.com/article/10.1007/s11940-012-0202-9
16. Kaewkhaw R, Rojanaporn D. Retinoblastoma: Etiology, Modeling, and Treatment. Cancers (Basel) [Internet]. 2020 Aug 16;12(8):2304. Available from: https://www.mdpi.com/2072-6694/12/8/2304
17. Ancona-Lezama D, Dalvin LA, Shields CL. Modern treatment of retinoblastoma: A 2020 review. Indian J Ophthalmol [Internet]. 2020 Nov 1 [cited 2024 Aug 8];68(11):2356–65. Available from: https://journals.lww.com/ijo/Fulltext/2020/68110/Modern_treatment_of_retinoblastoma__A_2020_review.9.aspx
18. Soussain C, Malaise D, Cassoux N. Primary vitreoretinal lymphoma: a diagnostic and management challenge. Blood [Internet]. 2021 Oct 28 [cited 2024 Aug 8];138(17):1519–34. Available from: https://ashpublications.org/blood/article/138/17/1519/476013/Primary-vitreoretinal-lymphoma-a-diagnostic-and
19. Giuffrè C, Menean M, Modorati GM, Marchese A, Cicinelli MV, Bandello F, et al. Primary vitreoretinal lymphoma: recent advances and literature review. Ann Lymphoma [Internet]. 2020 Dec 30 [cited 2024 Aug 8];4:1–16. Available from: https://aol.amegroups.org/article/view/6843/html
20. Wiley HE, Krivosic V, Gaudric A, Gorin MB, Shields C, Shields J, et al. MANAGEMENT OF RETINAL HEMANGIOBLASTOMA IN VON HIPPEL–LINDAU DISEASE. Retina [Internet]. 2019 Dec 1 [cited 2024 Aug 8];39(12):2254–63. Available from: https://journals.lww.com/10.1097/IAE.0000000000002572
21. Zając H, Turno-Kręcicka A. Ocular Manifestations of Behçet's Disease: An Update on Diagnostic Challenges and Disease Management. J Clin Med [Internet]. 2021 Nov 5 [cited 2024 Aug 14];10(21):5174. Available from: https://www.mdpi.com/2077-0383/10/21/5174
22. Olbert E, Struhal W. Retinal imaging with optical coherence tomography in multiple sclerosis: novel aspects. Wiener Medizinische Wochenschrift [Internet]. 2022 Nov 1 [cited 2024 Aug 15];172(15–16):329–36. Available from: https://link.springer.com/article/10.1007/s10354-022-00925-2
23. Somisetty S, Santina A, Sarraf D, Mieler WF. The Impact of Systemic Medications on Retinal Function. Asia-Pacific J Ophthalmol [Internet]. 2023 Mar 1 [cited 2024 Aug 15];12(2):115–57. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2162098923007879
24. Cho H. Ocular Manifestations of Systemic Diseases: The Eyes are the Windows of the Body. Hanyang Med Rev [Internet]. 2016;36(3):143–5. Available from: https://synapse.koreamed.org/DOIx.php?id=10.7599/hmr.2016.36.3.143

Aster HOSPITALS & CLINICS
We'll Treat You Well

Arthroscopic Decompression of an Entrapped Suprascapular Nerve due to Ganglion Cyst at Suprascapular Notch and Posterior Labral Repair performed successfully at Aster Cedars Hospital and Clinic, Jebel Ali

Dr. Shafeed Thadathil Parambil
Orthopaedics (Specialist)

PRESENTATION

- 35 year old male, volleyball player
- No medical history
- No family history of medical illness
- Admitted with:
  - Complaints of pain in the right shoulder for 5 months

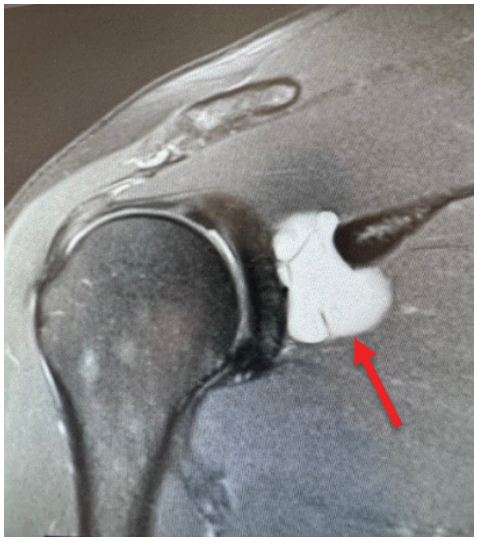
FINDINGS

During Examination:

- Physical examination revealed a full passive range of movement of the shoulder
- No wasting of supraspinatus or infraspinatus muscles was seen
- X-ray of the shoulder was normal

MRI showed:

- Large 45x30x11 mm sized thin walled multiseptated fluid signal intensity lesion in suprascapular region and posterior labral tear

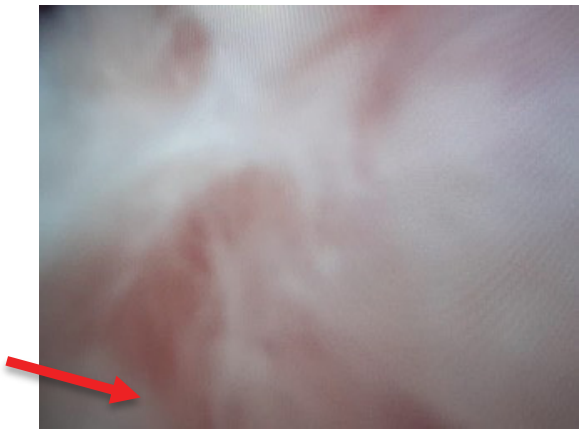


MRI image showing Cyst at the Suprascapular Notch

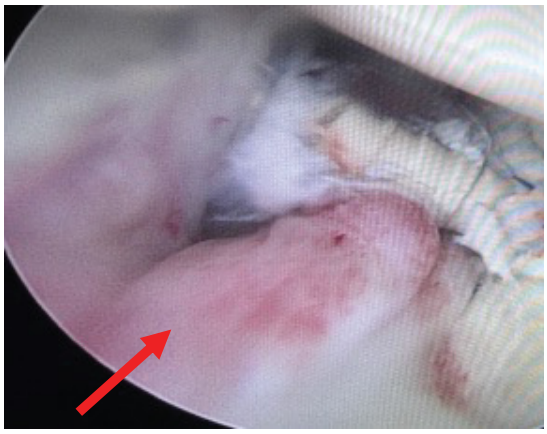
DURING PROCEDURE

- The procedure was done under general anaesthesia.
- The right shoulder was prepared and draped in the lateral decubitus position.
- Five portals were used: classic posterior portal, lateral subacromial portal, anterior portal, Neviaser portal and posterolateral portal.
- After inspection of the glenohumeral joint, the posterior labral tear was repaired using a knotless PushLock suture anchor.
- Arthroscope was introduced into the subacromial space till the suprascapular notch.
- Suprascapular nerve was found to be entrapped at the notch because of the large ganglion and tight superior transverse scapular ligament (STSL).
- The ligament was sectioned using arthroscopic scissors, and decompression of the cyst was done using an arthroscopic probe.
- Cyst wall was excised using an arthroscopic shaver.

Arthroscopic Images



Cyst at the Suprascapular Notch



Repair of Posterior Labrum with Suture Anchor

POST PROCEDURE

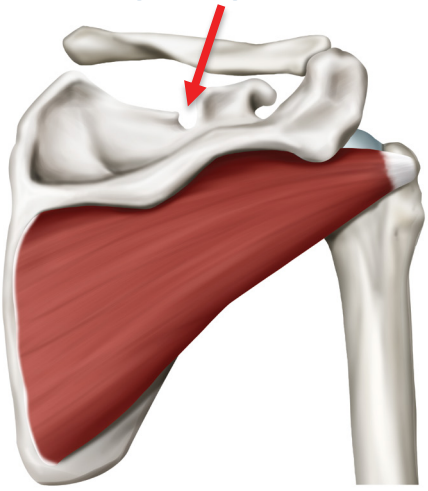
The patient experienced immediate pain relief after the surgery and was discharged the following day. He returned to his daily activities 2 months after the surgery.

DISCUSSION

The SSN (Suprascapular Nerve) is a mixed motor and sensory peripheral nerve arising from the superior trunk (C5, C6) of the brachial plexus with a variable contribution from the 4th cervical nerve root. The nerve travels below the transverse scapular ligament as it crosses the suprascapular notch to enter the supraspinatus fossa, whereas the suprascapular artery usually travels above the ligament.

Pressure on the SSN has been reported to be due to tumours or ganglion cysts (the most commonly reported mechanism), congenital structural changes of the scapular bone, a fracture of

Suprascapular Notch



the scapula that modifies the anatomic structure of the notch, and the sling effect phenomenon produced by the SSN being fixed at two points, one in the brachial plexus and the other in the supraspinatus muscle.

Diagnosing Suprascapular Nerve Entrapment Syndrome (SNES) can be challenging. A detailed history and physical examination are required. Radiographs, MRI, electromyography, and nerve conduction studies can provide essential information.

Initial treatment of suprascapular neuropathy without evidence of space-occupying lesion should be nonoperative, with activity modification, anti-inflammatory and analgesic medications combined with physiotherapy. Surgical intervention is indicated when there is no improvement after 6 months of nonsurgical care or in cases with clear evidence of a compressive lesion.

Arthroscopic decompression of SSN is technically challenging but less invasive and potentially a more effective way to treat suprascapular neuropathy, as it may provide a more rapid recovery.

REFERENCES

1. Thompson WAL, Koppel HP. Peripheral Entrapment Neuropathies of the Upper Extremity. N Engl J Med.1959; 260:1261–1265
2. Gosk J, Urban M, Rutowski R. Entrapment of the Suprascapular Nerve: Anatomy, Etiology, Diagnosis, Treatment. Ortop Traumatol Rehabil. 2007;9(1):68–74.
3. Cummins CA, Messer TM, Nuber GW. Suprascapular Nerve Entrapment. J Bone Joint Surg Am. 2000;82(3):415–424.
4. Aval SM, Durand P, Jr, Shankwiler JA. Neurovascular Injuries to the Athlete's Shoulder: part II. J Am Acad Orthop Surg. 2007;15(5):281–289.
5. Cohen SB, Dines DM, Moorman CT. Familial Calcification of the Superior Transverse Scapular Ligament causing Neuropathy. Clin Orthop Relat Res. 1997;334:131–135.
6. Khan MA. Complete Ossification of the Superior Transverse Scapular Ligament in an Indian Male Adult. Int J Morphol. 2006;24(2):195–196.
7. Osuagwu FC, Imosemi IO, Shokunbi MT. Complete Ossification of the Superior Transverse Scapular Ligament in a Nigerian Male Adult. Int J Morphol. 2005;23(2):121–122.
8. Alon M, Weiss S, Fisher B, Dekel S. Bilateral Suprascapular Nerve Entrapment Syndrome due to an Anomalous Transverse Scapular Ligament. Clin Orthop Relat Res. 1988;234:31–33.
9. Lafosse L, Tomasi A. Technique for Endoscopic Release of Suprascapular Nerve Entrapment at the Suprascapular Notch. J Shoulder Elbow Surg. 2006;7(1):1–6.
10. Millett P, Barton RS, et al. Suprascapular Nerve Entrapment: Technique for Arthroscopic Release. J Shoulder Elbow Surg. 2006;7(2):89–94.

Aster
HOSPITAL
We'll Treat You Well

ASTER HOSPITAL MUHAISNAH

Your friendly neighbourhood Family Hospital

Aster
HOSPITAL
We'll Treat You Well

State-of-the-art
Medical & Surgical Care

15+
Specialities

24/7
Emergency Care

Download
my Aster

Algeria Street, Muhaisnah