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- Title
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Editorial

Science practice and intent of better outcomes are keynote of the fourth edition of The Kenya Journal of Anaesthesiology and Critical Care Medicine. Discussion on actual case reports detailing management of critical and rare diseases as well as augmenting skills in safe novel regional anaesthetic techniques remain a focus of our publication and educative forum.

Anaesthesia service provision is a the challenge in the face of an ageing population whose co-morbid loads pose an increasing intolerance to conventional anaesthetic endurance, as well as resource constraints, demands innovation in provision of safe surgical and procedural medication. With an evolving improved safety profile, regional anaesthesia forms an important core of progressive regular anaesthetic delivery.

This edition features an anecdotal article on the conduct of The Supraclavicular block, commonly considered the “spinal anaesthetic” of the upper limb.

Trigeminal neuralgia is a rare debilitating condition characterised by episodic severe facial pain often resulting from trivial stimuli. It is often misdiagnosed as migraine thus resulting in inappropriate pharmacological prescriptions. Common reference in terminology of its clinical and social effects aptly describe it as “The Suicide Disease”. A local case report of guided multidisciplinary approach and successful intervention is featured in this edition.

Idiosyncrasy in anaesthetic pharmacology is a reason for the continued contemporaneous monitoring and resultant intervention. Midazolam is a commonly used agent for premedication as well as an anxiolytic and amnestic. Various dysplastic syndromes have been noted to predispose patients to adverse drug reactions: we highlight a case report in which a profound hypotension presents after low dose midazolam without any pre-existent dysplasia.

Safety of the airway in the presence of gastrointestinal dysmotility syndromes under sedation or anaesthesia for intervention often present the anaesthesiologist with critical challenges. Potential fatal of aspiration from delayed gut emptying, dehydration and electrolyte imbalance all can result in morbid peri-procedural outcomes. Rarely is paediatric achalasia cardia encountered in regular practice and one such clinical scenario and management is featured in our current publication.

Various mortality predictors based on acute physiological parameters have been highlighted in literature for clinical triage and acute medical interventions. Non traumatic sub arachnoid haemorrhage is one such debilitating condition which if not well managed may result in morbid outcome. The coexistence of vitreal haemorrhage, described as Tersons’s syndrome, has also been cited as a potential harbinger of potential visual loss and mortality in concurrence with non-traumatic subarachnoid events. The case report we highlight here clearly outlines the role of early ophthalmological consult and intervention in acute care for sub arachnoid vascular catastrophes’.

A case presentation on the anaesthetic management of a carotid body tumour is also highlighted in this edition.

Supraclavicular Block – The 'Spinal' of The Upper Limb

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Abstract

Regional anesthesia for the upper limb has grown in leaps and bounds. One of the greatest achievements was the provision of surgical anesthesia using a single shot approach that is very simple, highly reliable and rarely requires any block supplementation since patchy blocks are rare. Indwelling longer-term infusion catheters may also be safely placed.

Brachial plexus blocks and more so the Supraclavicular block should be in the armamentarium of all practicing anesthesiologists as it has been shown to be effective and easy to perform safely. General anesthesia and in many instances, procedural sedation may be avoided, in many ways simplifying and effectively making a safer profile of the perioperative experience.

Introduction

The supraclavicular block (herein after referred to as 'the block') was once considered a high-risk advanced anaesthetic approach that would only be performed by highly skilled and experienced practitioners. Prior to the introduction of the Ultrasound assisted peripheral nerve block techniques, it was associated with an up to unacceptably 6% risk of pneumothorax even when handled by the very experienced anaesthesiologist.

With the advent and wide distribution/availability of ultrasound machines ease of performance, safety and effectiveness in application have meant its more widely accessibility and acceptance. The hall mark has been the ultrasonographic mapping of the associated anatomical landmark of the plexus innervation–the Subclavian Artery.

It is the recommended upper limb which has a wide sensorimotor distribution, of ease to learn and perform mimicking the definition of upper limb spinal anaesthesia (REF) Respectively however, as with all regional anesthetic applications, protocol approach and safety precautions should be taken in its performance.

Historical Background

In 1911, a German surgeon by the name Diedrich Kulenkamph performed the first infraclavicular block and as was the trend in those days, subjected himself as experiential control to the block to get a true understanding of its extent of its anaesthetic profile.

Clinical Uses

The block is suitable for anesthesia and analgesia for arm (excluding the upper medial aspect), elbow, forearm, wrist and hand surgery. Recent evidence suggests that it may be effective for shoulder surgery but with greater supplementation and/or sedation compared with the interscalene block.

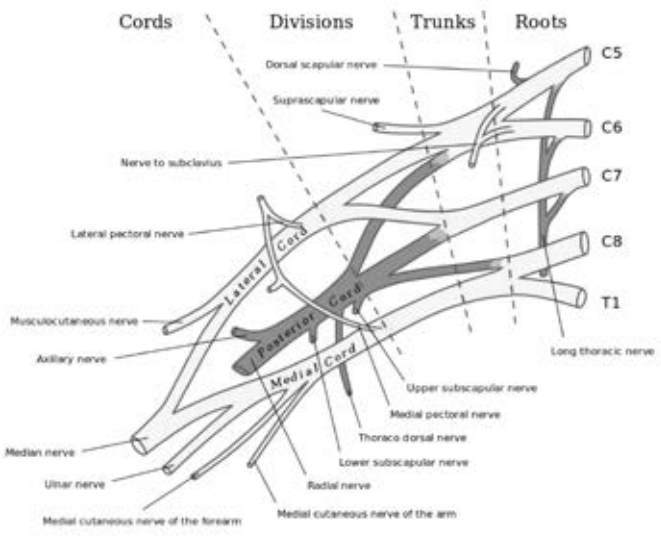
For hand surgery lidocaine has been used in the supraclavicular block for surgery and management of tourniquet pain. A supplemental wrist block for post operative pain management is performed with bupivacaine and occasional adjuvants prolonging therefore effective analgesia from the regional anaesthetic application.

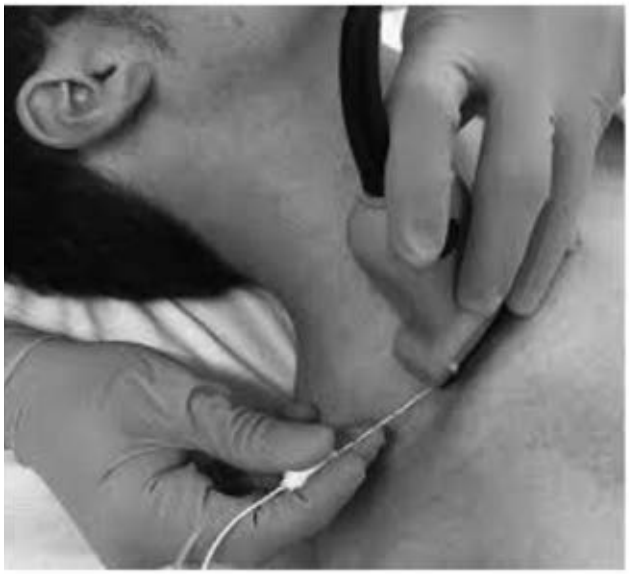
Anatomy – Basic and Ultrasonography

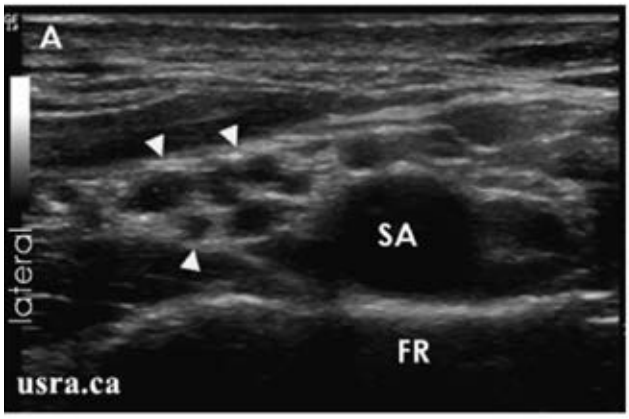
The trunks of the brachial plexus (C5-T1) are compactly bundled up in a neurovascular sheath together with the highly ultrasonographically visible Subclavian Artery. This in theory (and as seen clinically) makes this location ideal for plexus local anesthesia since the drugs used are contained in this location and also being a tight space, small volumes are required to achieve surgically acceptable neural blockade.

As shown in the diagram below, the only nerves in the entire brachial plexus that are not blocked by this technique are the nerve to subclavius, the long thoracic nerve of Bell and the Dorsal Scapular Nerve (which is considered important for shoulder surgery). The rest of the nerves supplying the upper limb are effectively anaesthetized hence the popularity of the block.

The subclavian artery is the key landmark for the block. It crosses the upper border of the first rib between anterior and middle scalene muscles. The Brachial plexus will be found superficial and posterolateral to the artery.







Block Performance

The appropriately counseled, consented and monitored sedated patient lies supine with the upper limb to be operated on adducted and lying comfortably on the bed/ table. A high frequency linear ultrasound probe is placed in the supraclavicular fossa in the midpoint of the clavicle facing caudad. The subclavian artery will be readily visualized in short axis (transverse view).

A fine needle (G30) can be used to anaethetise the needle insertion point with lidocaine after which a 50 mm peripheral nerve block needle is inserted in a lateral to medial orientation aiming initially at the corner formed by the Subclavian artery and first rib (described as the 'eight ball in the corner pocket' in reference to table pool games) .5-10 mL of the anaesthetic solution is initially deposited here with subsequent redirection of the needle, injecting the solution to ensure that the subclavian artery is surrounded by the solution. 20 mL of solution is usually more than adequate for a successful surgical block. It is important to note that most of the targeted brachial plexus lies posterolateral and superficial to the subclavian artery.

The medial aspect of the upper to mid arm is not blocked with this technique due to the fact that the skin on this aspect of the upper limb is innervated by the intercostobrachial nerves which come from T1-2. These superficial nerves can easily be blocked near the axilla by a subcutaneous wheal of local anesthetic placed on the upper medial arm from the anterior to posterior axillary lines.

Drugs Used

- Bupivacaine 0.1-0.5% depending on density of block required
- Adrenaline usually 1:200,000
- Dexamethasone 4 mg in 20 mL of solution
- Dexmedetomidine – 50 mcg barring any contraindications

Adverse Effects/Difficulties

- Inadvertent phrenic nerve block may occur in 50% to 2/3 of patients and utmost care must be observed in patients with pulmonary compromise or those with contralateral phrenic nerve palsy.
- It is usually difficult to maintain an indwelling catheter for continuous nerve blockade.
- Obese patients may present challenges as the structures of interest may be surprisingly and disturbingly deep.

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Trigeminal Neuralgia; Overview and Case Report

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Summary

Trigeminal neuralgia is a chronic, debilitating condition with episodes of extreme facial pain; sporadic and sudden and often like 'electric shocks' lasting a few seconds or minutes. We report a case of a 50 year old business lady who, in 2014, began feeling an abnormal sensation in her right lower jaw and face. She would sneeze a lot in the cold mornings, and washing her face resulted in sharp pain which would subside as the day progressed. She had initially ignored it but it had recently increased in intensity, worsened by chewing, turning the head, walking fast, opening the mouth, yawning, wind blowing on her face, and salty/sugary food. The painful attacks were now increasing in sharpness, frequency, and lasting 2-5 minutes. The jaw pain was initially diagnosed as being of dental origin leading to extraction of the right second upper pre-molar. In her estimation, the pain was beyond the limit of the visual analogue scale (VAS) when provoked, and frequently kept her awake at night. Pain medications reduced it only slightly.

When she first presented to the pain clinic in December 2017, she was diagnosed with trigeminal neuralgia and ultra-sound guided neurolysis was recommended and performed on the maxillary and mandibular nerves. However, the resultant pain relief lasted only one week. She, therefore, underwent a second neurolysis in January 2018 and has remained mostly pain-free with minimal medication since then.

Introduction

Trigeminal neuralgia or tic Douloureux (hemifacial spasm) is a neurological disorder of the trigeminal nerve that causes episodes of intense pain in eyes, lips, scalp, forehead and jaws. It is also known as prosopalgia, Fothergill's disease, Forthergill neuralgia, prosoponeuralgia, or trifacial neuralgia.

It has been labelled as "suicide disease" due to a significant number of people taking their own lives in desperation because they were unable to have their pain controlled by medications or surgery.

Case Report

We report a case of a 50 year old lady who, in 2014, began feeling an abnormal sensation in her right lower jaw and face. She would sneeze a lot in the cold mornings, and washing her face resulted in sharp pain which would subside as the day progressed. She had initially ignored it but it had recently increased in intensity, worsened by chewing, turning the head, walking fast, opening the mouth, yawning, wind blowing on her face, and salty/sugary food. The painful attacks were now increasing in sharpness, frequency, and lasting 2-5 minutes. The jaw pain was initially diagnosed as being of dental origin leading to extraction of the right second upper pre-molar. In her estimation, the pain was beyond the limit of the visual analogue scale (VAS) when provoked, and frequently kept her awake at night. Pain medications reduced it only slightly.

When she first presented to the pain clinic in December 2017, she gave a four year history of severe right upper jaw pain which had worsened over the last few weeks. She had trismus, and the pain was not responding to oral medications which included: Cymbalta 30mg bd, Imuran 50mb bd, Duzac 30mg bd, Dexamethasone 8mg tds, Lamictal 50mg od, Baclofen 10mg bd and Trileptal 450mg bd.

Her primary physician had administered Local Anaesthetics and steroids to facilitate travel to Nairobi for Neurolysis at the pain clinic where she was diagnosed with trigeminal neuralgia and ultra-sound guided neurolysis was recommended and performed on the maxillary and mandibular nerves. The resultant pain relief lasted only one week, and her pain returned with a vengeance. In January 2018, she underwent a second neurolysis (under CT guidance) with 10 ml of 70% alcohol and a cocktail consisting of 10mg plain bupivacaine 0.5% and depomedrol 240mg. She has since remained pain-free with minimal medication.

Although there was no more pain at the first post-procedure review, she complained of; dizziness, double vision, headache, numbness, heaviness of the tongue and was noted to have a more pronounced right Facial Nerve Palsy.

At her second review later in January, there was masseter weakness and diplopia which were improving as she continued to have physiotherapy for Facial Nerve Palsy. There was numbness of the right side of her face, with the ophthalmic region being more affected than the mandibular. She also reported to have bitten and cut her right lower lip without feeling pain. She had no corneal reflex and complained of hotness of both legs in the evening! She, however, had no more headache or pain. She has been on follow-up at the ophthalmology clinic with good progress.

Discussion

Aetiology

Trigeminal neuralgia is usually thought to be idiopathic. In most cases, however, it may be caused by compression of the trigeminal nerve by a loop of artery or vein (Figure 1). Another 5-10% of cases are attributed to intra-cranial tumours, demyelination of the nerve, petrous ridge compression, post-traumatic neuralgia, viral aetiology, multiple sclerosis, and abnormalities of the skull base or A-V malformations.

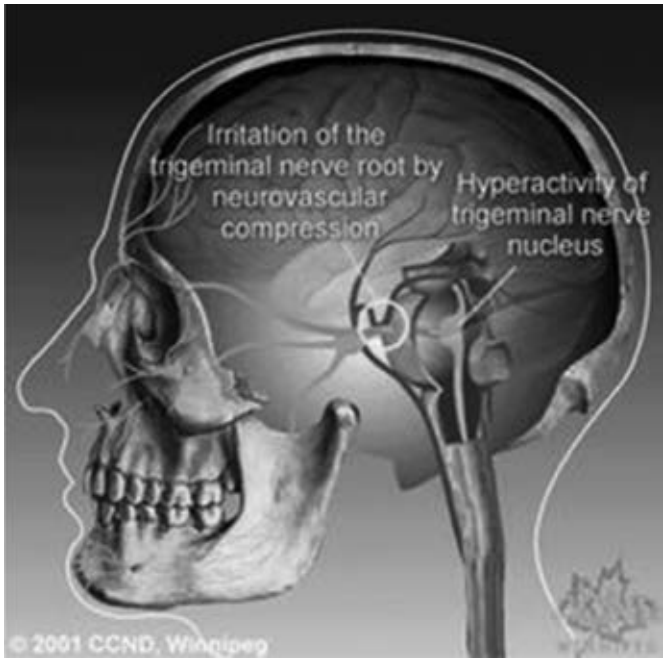

Figure 1. Neurovascular compression of the trigeminal nerve root.

Evidence that vascular compression commonly causes trigeminal neuralgia is based on the following facts:

i. An aberrant loop of artery, or less commonly vein, is found compressing the root entry zone of the trigeminal nerve in 80-90% of patients at surgery

ii. The trigeminal nerve is demyelinated next to the compressing vessel

iii. Eliminating the compression by surgery provides long term relief in most patients

iv. Intraoperative assessment reports immediate improvement in trigeminal nerve conduction on decompression

v. Sensory function recovers after decompression

Other causes such as compression by tumours or the demyelinating plaques of multiple sclerosis, produce similar lesions of the root entry zone of the trigeminal nerve.

Symptoms

Trigeminal neuralgia is a sudden, unilateral, stabbing, recurrent pain in the distribution of one or more branches of the 5th cranial nerve. Pain occurs in paroxysms which last from a few seconds to two minutes. The frequency of paroxysms range from a few, to hundreds of attacks daily. Periods of remission can last for months to years but tend to shorten over time. There may be preceding symptoms such as tingling or numbness, and patients may have certain triggers that set the pain paroxysms off. This is followed by sharp severe, shock-like pains which are usually on one side of the cheek or face but can involve the eyes, lips, nose and scalp. Episodes are intermittent but can last days, weeks or months and then subside for months or even years. 3-5% of patients will have bilateral pains.

Investigations

MRI scan of the brain is indicated to rule out other potential causes of pain if the diagnosis is uncertain. MRI may identify:

i. Sinusitis , or extra-cranial masses along the course of the trigeminal nerve

ii. Pathological enhancement of the trigeminal nerve that could indicate perineural spread of malignancy

iii. Carvenous sinus masses and demyelination plaques that might indicate multiple sclerosis

iv. Intrinsic brain lesions in the thalamus or trigeminal brainstem pathways such as lacunar infarctions

v. Cerebellopontine angle mass lesions such as tumour, epidermoid, dermoid or arachnoid cyst, aneurysm, or arteriovenous malformations.

Diagnosis

i. Well taken history and clinical examination

ii. CT Scan

iii. MRI Scan

iv. Diagnostic nerve block

Management Of Trigeminal Neuralgia

1. Medical Management

a. Carbamazepine (anticonvulsant)

b. Baclofen, Lamotrigine, Oxarbazepine, Phenytoin, Gabapentin, Pregabalin, Sodium valproate

c. Duloxetine (where neuropathic pain and depression are combined)

d. Opiates such as Morphine or Oxycodone

2. Surgical Management

a. Nerve block with long acting local anaesthetic agent or alcohol

b. Peripheral glycerol injection

c. Peripheral neurotomy (Nerve avulsion)

d. Open procedures (intracranial procedures) – microvascular decompression, percutaneous rhizotomy, Gama knife radiosurgery

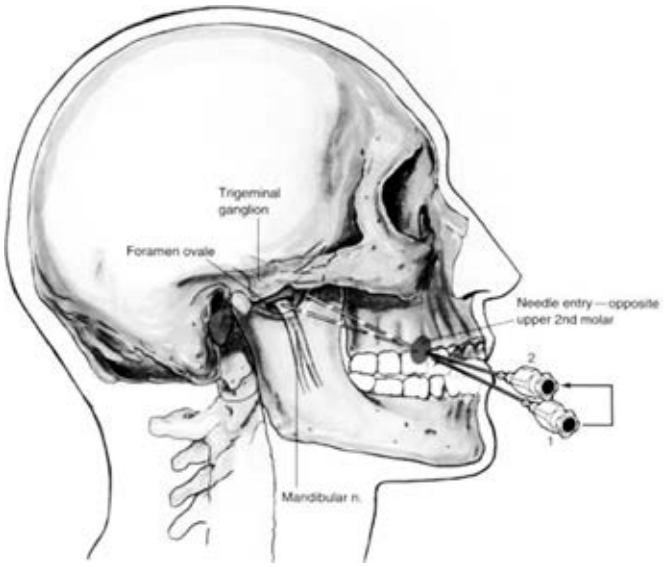

Figure 2: Anatomical Approach to Trigeminal Neurolysis.

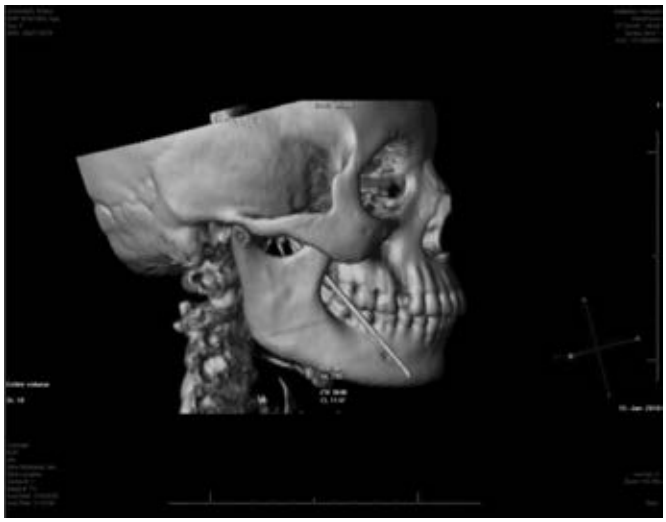

Figure 3: Actual Procedure of CT- guided Neurolysis of Trigeminal Nerve.(Lateral view)

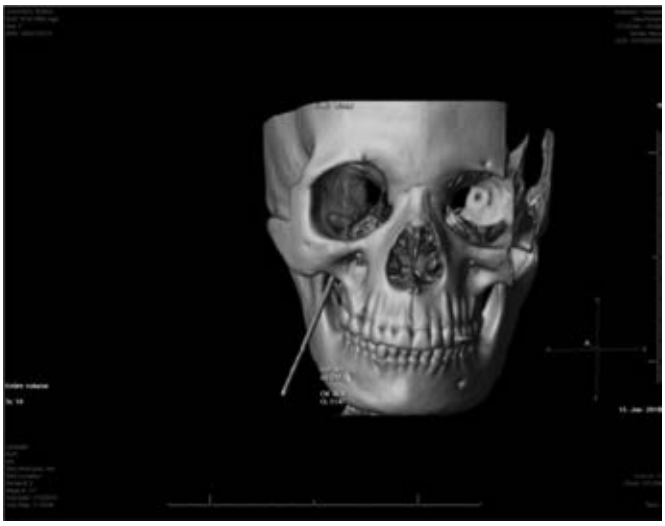

Figure 4: Actual Procedure of CT-guided Neurolysis of Trigeminal Nerve (AP view)

Acknowledgements

We wish to sincerely thank Dr. Patricia Otieno for taking care of the ophthalmic complications following the neurolysis of the trigeminal nerve. Dr. Divya Patel played a major role in meticulously providing conscious sedation to the patient during the procedure, and for this we are most grateful. To the radiology staff led by Mr. Thiong'o, who made it possible for us to carry out the procedure without any hitches, we feel eternally indebted.

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Case Presentation

Severe Hypotension After Low Dose Midazolam

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Summary

We report the occurrence of severe hypotension following low dose intravenous midazolam premedication, in a 52-year-old patient scheduled to undergo total knee arthroplasty under regional anaesthesia. During the positioning for performance of the prescribed regional anaesthetic, severe hypotension was picked up with procedural protocol monitoring requiring vasopressors for effective resuscitation and management.

Introduction

Midazolam is a commonly used sedative/anxiolytic in the clinical set up. It is a short-acting imidazobenzodiazepine central nervous system depressant. It is generally a safe drug when used properly. It is mainly indicated for procedural sedation. Severe adverse effects of midazolam are uncommon and include respiratory depression and anaphylactic reactions. Hypersensitivity reactions to midazolam although rare have been reported worldwide. We report a case of severe hypotension to midazolam in the absence of other signs of a hypersensitivity reaction. The aim of this report is to create awareness to anaesthesia providers and other health care providers in general on the likelihood of severe adverse reactions to midazolam, even in low doses. It also emphasizes early recognition and treatment of adverse events.

Case Report

A 52-year-old female patient was admitted for left total knee arthroplasty during an outreach orthopaedic camp. She had severe osteoarthritis of the left knee and was on oral diclofenac for pain relief. The patient had no history of co-morbid states though she had congenital hypoplastic feet. She had no history of food and drug allergy. She was a peasant farmer with two group children. She denied any alcohol, cigarette or any illicit substance use.

On examination preoperatively, she was in fair general condition, weighed 64kg with a height of 172cm. Her blood pressure was 127/82 mmHg, pulse rate of 82 beats per minute and respiratory rate of 16 breathes per minute. The examination of her organ systems yielded normal findings except for the hypoplastic feet and tender left knee with limited active range of motion.

The laboratory work up included total blood count, and urea, electrolytes and creatinine levels. The results were within the normal ranges.

After preoperative evaluation, regional anaesthesia (combined spinal anaesthesia and left femoral block) was planned. This was to be her first anaesthesia and surgical exposure and she was counselled and educated on the planned anaesthesia and perioperative pain management plan. Gabapentin was prescribed as premedication and diclofenac was continued and appropriate fasting instructions issued.

On the day of the surgery, the patient was transported to the holding area for the femoral block to be performed. Standard monitors were attached, and intravenous access established. The blood pressure was 116/76mmHg, heart rate of 82/min and peripheral oxygen saturation (SPO2) of 99%. Ringers lactate infusion was started. Resuscitative and procedural medication was prepared and labelled appropriately. The patient was positioned for the block and 1mg of midazolam administered intravenously. Within a minute of midazolam injection, the patient complained of "not feeling right", and looked apprehensive. The heart rate dropped to 68 beats/min and the blood pressure to 62/32mmHg. After a quick evaluation and in the absence of other signs of anaphylaxis, 6mg of ephedrine were administered intravenously. The blood pressure rose to 90/51mmHg with a heart rate of 92/min. The Ringers lactate infusion was continued, and supplemental oxygen given via face mask. She required further boluses of ephedrine to stabilise her blood pressure. After one hour her blood pressure and heart rate stabilised to pre-event levels. The recovery was uneventful.

The surgery was postponed, and the patient was referred for allergy testing and cardiologist evaluation. The vial of midazolam used for this patient was from a batch that was used in other patients with no adverse effects reported.

Discussion

Midazolam is frequently used just before performance of regional anaesthesia/analgesia techniques. This is partly to allay anxiety and to provide comfort during needling in the patient.

Our case was unique from other reported cases of severe adverse reaction to midazolam. The severe hypotension occurred in the absence of other clinical signs of hypersensitivity reaction. This was in a medical outreach setting with expected high turnover

rate of patients and visiting health care providers. It is because of this setting that laboratory tests to confirm anaphylaxis (blood tryptase levels) we not immediately available .

Anaphylactic reactions are IgE-mediated. There is massive release of mediators from tissue mast cells or from circulating basophils. These reactions manifest as severe hypotension or cardiovascular collapse, bronchospasm, and cutaneous manifestations. Serum tryptase levels should ideally be measured in suspected anaphylactic reactions especially in the absence of other signs and symptoms of anaphylaxis. Serum tryptase levels begin to raise 1-2 hours after the event. Thereafter, other confirmatory tests like skin testing should be carried to ascertain the causal relationship of the suspected allergen.[1,2,3,4]

Anaphylactoid reactions also occur. These reactions produce a similar clinical picture as anaphylactic reactions, but they are not IgE-mediated.[3,5,8]

For this patient, we also reviewed other possible aetiologies for occurrence of sudden and severe hypotension. We considered the fact whether there could be an undetected cardiovascular lesion. This is partly in consideration of the presence of congenital lower limb hypoplasia. From the reported studies, careful titration of midazolam seems well tolerated in patients with congenital and acquired cardiac lesions. There have been descriptions of development of an acute coronary syndrome, the Kounis syndrome, after a hypersensitivity reaction.

This is characterised by increased cardiac biomarkers and can progress to heart failure and cardiovascular collapse.[6,7]

We also considered the possibility of drug interactions. There are no reports of severe hypotension after midazolam in a patient on gabapentin or diclofenac. This was quickly ruled out as a possible cause. There is however a possibility of hypotension in patients on concomitant opioids.[5,8]

Treatment of hypotension is dependent on the severity of the hypotension and the possible aetiological factors. In suspected anaphylaxis, epinephrine is the treatment of choice. It induces vasoconstriction, reduces mucosa oedema and causes bronchodilation. It has inotropic/chronotropic effect. Other supportive measures include hydration, supplemental oxygen, antihistamines and steroids. Other vasoconstrictors, e.g. ephedrine, are useful in severe hypotension caused excessive vasodilation, which is also occurs in anaphylaxis.[1,2,5,10,11] For this patient the hypotension was responsive to ephedrine and intravenous fluids.

In conclusion, severe adverse drug reactions require early recognition and prompt management. Objective causal relationship should be sought and documented. Unexpected reactions to midazolam, despite its safety profile have occurred and high index of suspicion is warranted. Early recognition and treatment of adverse drug reactions is warranted. This is to prevent the negative consequences late recognition.

Conflict of interest issues regarding the authorship or article: None declared.

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Case Report:

Achalasia Cardia: A Rare Encounter for The Paediatric Anaesthetist

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Abstract

Achalasia cardia is an oesophageal motility disorder characterized by the absence of oesophageal peristalsis and impaired relaxation of the lower oesophageal sphincter (LES) in response to swallowing. The abnormalities cause a functional obstruction at the gastro-oesophageal junction (GEJ).

Overall incidence is approximately 1/100,000 of which paediatric cases represent up to 1/10th and the incidence of childhood achalasia is therefore frequently reported as 0.11/100,000. **[1].**

Paediatric achalasia is often misdiagnosed as gastro-oesophageal reflux disease (GERD) with many children treated with anti-reflux medication before the diagnosis is made. The diagnosis may be delayed for many years in children who are typically unsuccessfully managed for failure to thrive, eating disorders, oesophagitis or asthma.

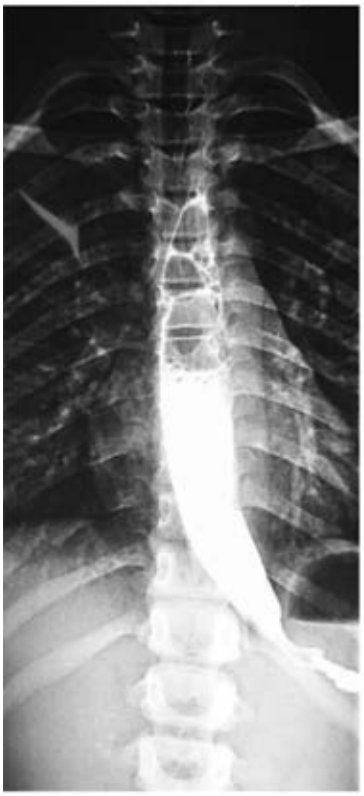
Data concerning the epidemiology of achalasia in Africa are lacking **[2-3]** and within the paediatric population consists mostly of case reports. **[4-5].**

We present a case of childhood achalasia managed in our institution in the last year.

Case Presentation

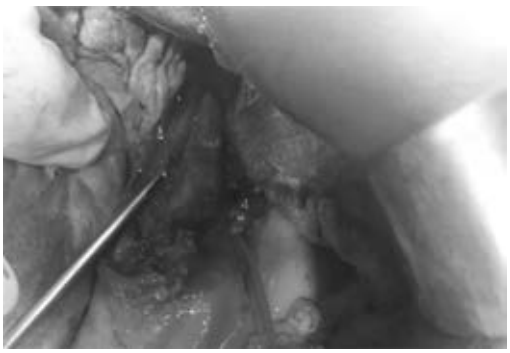
A 9-year-old male patient presented as a referral from a peripheral facility with a 4-month history of progressive dysphagia, vomiting, abdominal pain and 20 percent weight loss over 4 months. During this period treatment for GERD was unsuccessful and a barium swallow demonstrated oesophageal dilatation and narrowing at the LES prompting referral.

Pre-operative Barium Swallow showing the typical 'birds beak' appearance of the LES and oesophageal dilatation.

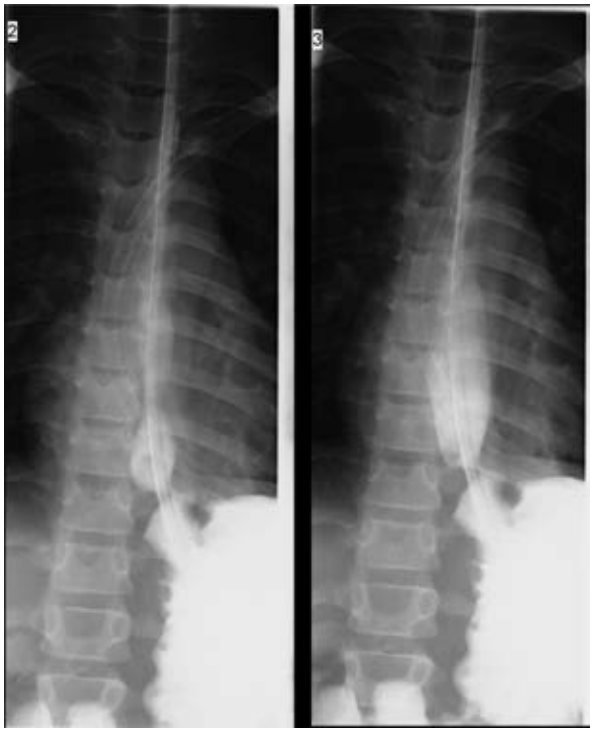


Pre-operative oesophagogastroduodenoscopy (OGD) excluded stricture, ulceration and hiatus hernia.

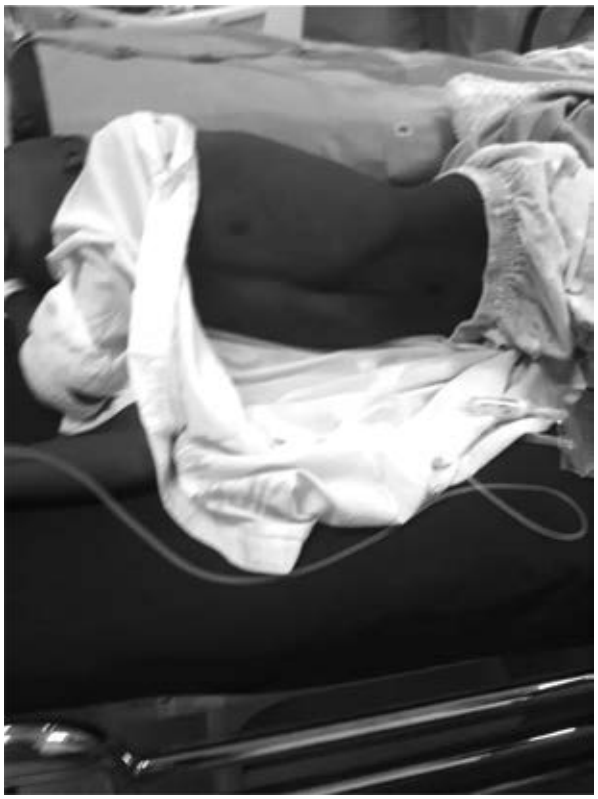
An open Heller's cardiomyotomy and partial Nissen's fundoplication was done uneventfully under general anaesthetic with discharge from High Dependency Unit after 24 hours.



Oral intake was resumed on the 4th post-operative day after a barium meal. This showed post-operative findings of Heller's cardiomyotomy and Nissen's fundoplication. It suggested possible over-constriction at the fundoplication site.



The patient was able to tolerate liquids but experience dysphagia with solids. A post-operative OGD and dilation of stricture at fundoplication site was done on the 9th post-operative day with discharge from hospital on the 10th post-operative day with instructions to take pureed food.



Pre-operative Barium Swallow showing the typical 'birds beak' appearance of the LES and oesophageal dilatation.

A second post-operative OGD and dilation was done 2 weeks after discharge when it was noted that the patient had began to gain weight. The patient remains under follow-up and further oesophageal dilatations will be done only if indicated.

Discussion

Achalasia is diagnosed by radiographic studies (barium swallow) and oesophageal manometry **[6].** Barium swallow studies classically demonstrate a dilated esophagus with "bird's-beak" like tapering of the distal esophagus.

Oesophageal manometry shows elevated resting LES pressure, absent or low amplitude peristalsis, or non-relaxing LES upon swallowing. However, absence of these findings does not rule out the diagnosis of achalasia since LES function in children is heterogeneous. Partial relaxations are common and normal relaxations may also be present on manometry. **[1]**

Upper endoscopy and biopsy is reasonable to rule out esophagitis, tropical infections, malignancy, and other secondary causes of achalasia although these are much less common in children.

In this patient, the diagnosis of achalasia was made on the history, barium swallow and OGD ruling out secondary causes of dysphagia.

Socio-economic status is a barrier to diagnosis and intervention. Increasing coverage of the Kenyan population with the National Health Insurance Fund has enabled increased access to healthcare as in this case.

Treatment aims to reduce LES pressure in order to facilitate oesophageal emptying. Medical options include oral injection of botulinum toxin and oral administration of calcium channel blockers (Nifedipine). Physical interventions are either serial pneumatic dilatations or oesophageal myotomy (Heller's) with or without an anti-reflux procedure. A novel technique, per oral endoscopic myotomy (POEM) is increasing being utilized.

The low numbers of cases hampers evidence for the optimal treatment of achalasia in different age groups. A recent systematic review concluded adequate comparative data are lacking to determine the ideal treatment for paediatric achalasia **[7].** Nevertheless, in clinical practice surgery is the preferred treatment. This involves a Heller's cardiomyotomy, either laparoscopic or open, which is usually combined with a partial fundoplication **[1, 8-9].** Oesophageal perforation and dysphagia recurrence are the two most common complications of this surgery. Therefore a contrast study is a requirement of the postoperative period. Dysphagia recurrence at up to 26% of cases compares favourably with oesophageal dilatations where it occurs significantly more frequently. Treatment of post-operative dysphagia is by dilatation under OGD and/or re-do operation.

In this case the patient was discharged 10 days post-operatively due to partial relief of symptoms with surgery alone necessitating post-operative oesophageal dilatation.

Anaesthetic management for achalasia surgery includes pre-optimisation of a cachexic patient with nutritional and biochemical abnormalities, precaution against pulmonary aspiration and expectation of prolonged surgery with possible complications of GIT perforation, blood loss and requirement for thoracotomy. **[10]** Multimodal analgesia with systemic and local anaesthetic agents is ideal. Most patients are able to begin oral intake in 48 hours and are discharged within a week.

When the newer POEM procedure is used, additional anaesthetic considerations include complications of intraoperative carbon-dioxide insufflation such as increased intra-abdominal pressure intra-operatively and post-operative surgical emphysema.

Achalasia patients are subject to multiple anaesthetics for diagnostics and interventions and strategies to reduce perioperative anxiety should be employed.

Conclusions

Due to its low incidence the adult or paediatric anaesthetist rarely encounters achalasia. In low and middle-income settings, appropriate referral guidelines and pathways can assist in shortening the commonly encountered delay in diagnosis. **[11]** Furthermore, adequate treatment and follow-up can only be guaranteed in settings where there is universal access to these interventions.

This case report has illustrated many of the challenges in diagnosis and management of paediatric achalasia and adds to the growing body literature on the subject.

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Terson's Syndrome: Intraocular Hemorrhage in Subarachnoid Hemorrhage

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Abstract

Terson's syndrome is vitreous hemorrhage occurring consequent to subarachnoid hemorrhage. The syndrome of vitreous hemorrhage in association with subarachnoid haemorrhage (SAH) was first described by Albert Terson, French ophthalmologist 1900. The patient with Terson's syndrome will present with symptoms of deterioration in visual acuity, which degree depends on the extent of the bleeding, along with characteristic neurological symptoms, the vitreous haemorrhage can be unilateral or bilateral. The cause of vitreous haemorrhage in SAH is raised intracranial pressure causing distension and rupture of peripapillary and retinal capillaries. Terson's syndrome, in association with diffuse SAH, has significant increase of risk of death. Reported complications of Terson's syndrome include visual loss, macular holes, epiretinal membrane formation, retinal folds, proliferative vitreoretinopathy, and retinal Detachment. Visual loss is reversible after removal of vitreous haemorrhage by vitrectomy. We present here the case of 33 years old man who presented with spontaneous subarachnoid hemorrhage associated with progressive visual loss. He was diagnosed as Terson's syndrome and his vision was restored successfully after vitrectomy.

Key Words: Terson Syndrome, Subarachnoid hemorrhage, intraocular haemorrhage, Intracranial Aneurysm, Vitreous haemorrhage.

Introduction

Intraocular hemorrhage is very rare and frequently missed ophthalmic finding in Subarachnoid hemorrhage. The subarachnoid haemorrhage associated with retinal hemorrhage was first described in German literature by Litten in 1881 **(1).** The syndrome of vitreous hemorrhage in association with subarachnoid haemorrhage (SAH) was first described by Albert Terson, French ophthalmologist 1900 **(2).** Intraocular hemorrhages can be sub-retinal, retinal, subhyaloidal or intra-vitreal **(3).** Terson's Syndrome defined as retinal or vitreous hemorrhage in the presence of intracranial hemorrhage mostly SAH, occurring in 12.5% to 40% of cases **[4,6-9].** The patient with Terson's syndrome will present with symptoms of deterioration in visual acuity, which degree depends on the extent of the bleeding, along with characteristic neurological symptoms. Vitreous haemorrhage in the course of Terson's syndrome can be unilateral or bilateral. Other clinical symptoms include optic disc oedema and formation of preretinal membranes in the macula region.

The theory about pathogenesis of Terson's syndrome has been much debated **(10-13).** Subarachnoid hemorrhage due intracranial aneurysmal rupture or head trauma rapidly rises the intracranial pressure **(4,8)**. This sudden rise in intracranial pressure may force blood into the subarachnoid space along the optic nerve sheath into the preretinal space, or may lead to decrease in venous return to the cavernous sinus or obstruct the central retinal vein and retinochoroidal anastomoses, culminating in venous stasis and hemorrhage, i.e. causing distension and rupture of peripapillary and retinal capillaries resulting in remarkable hemorrhage in the vitreous cavity or subhyaloid space **(5,11-14).**

The diagnosis of Terson's syndrome can be made on the basis of clinical symptoms, fundoscopy, and computed tomography of brain tissue with orbital structures. A B-mode ultrasound scan allows for establishing the extent of haemorrhage and assessing the possibility of its spontaneous resorption. A close cooperation of ophthalmologist and neurologist is essential in the therapeutic process.

We present here the case of 33 years old man who presented with spontaneous subarachnoid hemorrhage associated with progressive visual loss. He was diagnosed a Terson's syndrome and his vision was restored successfully after vitrectomy.

Case Report

33 year old male, presented to the Casualty department with complaints of a headache for 10 days. Headache was of sudden onset, persistent, worsening in severity over the last 3 days, radiating to the neck. It was associated with blurry vision, projectile vomiting and having had lost consciousness for approx. three hours. On examination, patients vitals: HR 93 BP 126/69 RR 24 SPO2 95%. Central nervous system: GCS 15/15, Nuchal rigidity, pupils equally reacting to light and no focal neurological deficits. CT SCAN – Acute subarachnoid haemorrhage with haemorrhagic hyper intensities within both optic globes (Figure 1&2). CT CEREBRAL ANGIOGRAM –Bi lobed Saccular aneurism of the anterior communicating artery with non-visualised A1 segment of right anterior cerebral artery? Vasospasm. 4 Vessel Angiogram was done to rule out vasospasm: showed the large bi lobed anterior communicating artery aneurism.

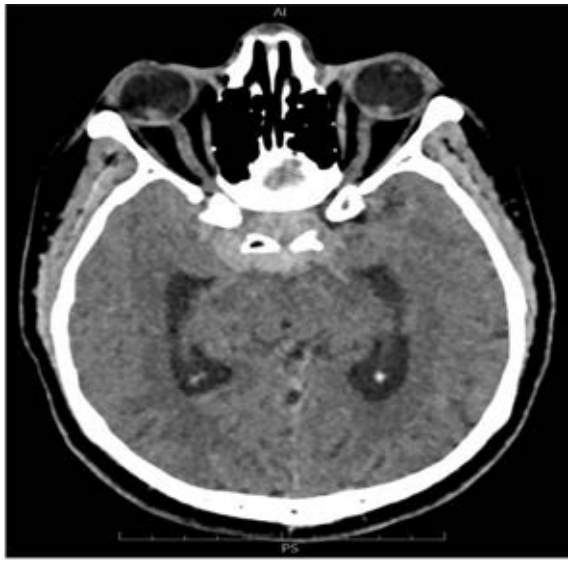

Figure-1 (Non-ContrastCT-Brain demonstrating subarachnoid haemorrhage with ocular extension).

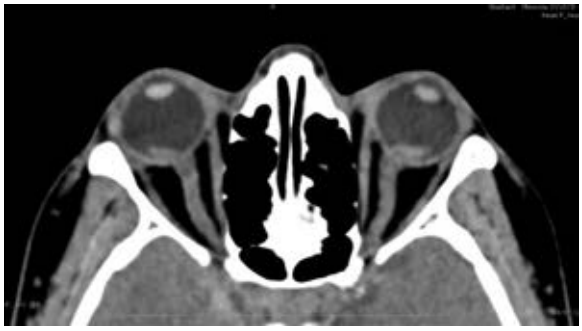

Figure-2 (Non-ContrastCT-Brain demonstrating Haemorrhagic hyper intensities within both optic globes)

On ophthalmic evaluation the patient had poor visual acuity CF at 1metre, normal adnexa, sclera, conjunctiva and a clear lens with pupils reacting equally to light. Fundoscopy confirmed bilateral dense haemorrhages in the vitreous bilaterally with retina being unable to be visualised. Recommended for vitrectomy if no visual improvement in fourteen days.

The patient was planned for aneurismal clipping. Aneurismal clipping was done on the third day since admission, after which the patient faired on well post operatively but with some confusion and disorientation. He had no improvement in vision up to 14 days post admission, now having only some appreciation of bright light with the both eye. Patient had developed neurological deterioration, repeat CT SCAN brain done which revealed hydrocephalus. A ventriculo-peritoneal shunt was done and patient improved, fully alert and mobile but with no visual improvement. Ocular ultrasound: Bilateral vitreous echogenic debris (Figure 3&4) with the posterior vitreous detachment- left being more then the right (right =2.1mm, left 2.3mm). Vitrectomy was plan after one week.

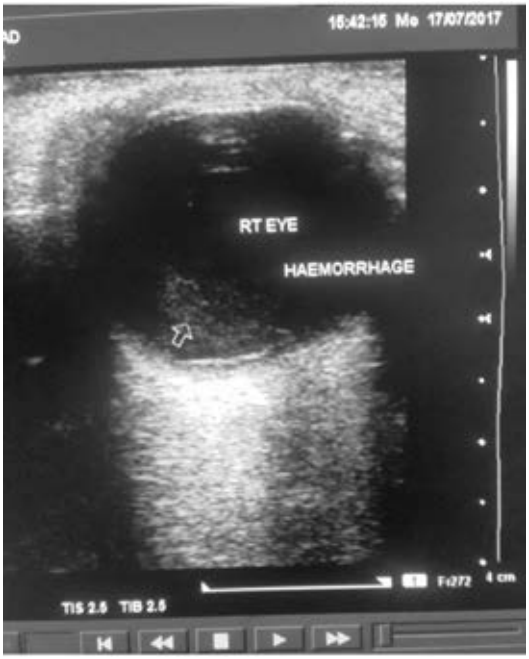

Figure-3 (Ocular Ultrasound demonastrating Right Vitreous Haemorrhage)

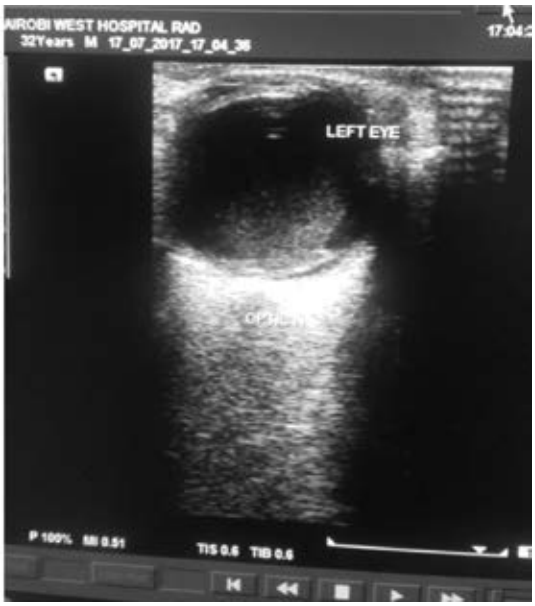

Figure-4 (Ocular Ultrasound demonastrating Left Vitreous Haemorrhage)

Patient was discharged home to be followed up at the ophthalmology clinic during which a vitrectomy was done and the patient's vision was restored successfully

Discussion

One of the known complications of SAH is the occurrence of bleeding in posterior eye called Terson Syndrome. This finding often missed in patient with SAH. The reported frequency of Terson's syndrome in such patients ranges between 20% and 50% **(15, 16, 17, 18).** Recent studies suggest that vitreous haemorrhage on its own is an indicator of poor prognosis in patients with subarachnoid haemorrhage. Terson's syndrome is associated with a worse outcome than in patients with subarachnoid haemorrhage without vitreous haemorrhage **(19).** Thus documentation of vitreous haemorrhage in CT image and ophthalmic examination is required in all patients with subarachnoid haemorrhage, especially as most deaths are a direct result of the neurological consequences of subarachnoid bleeding **(20).** The theory about pathogenesis of Terson's syndrome has been much debated **(10-13).** The most probable theory seems to be the one presuming that the primary cause of Terson's syndrome is the elevation of intracranial pressure which causes oedema of the retrobulbar part of the optic nerve, which in turn hampers the venous return from retinal veins to cavernous sinus by closing the retinal and choroidal vessels on cribrum level. This creates a venostasis which causes minute superficial retinal vessels to rupture, resulting in vitreous bleeding. This theory also explains the occurrence of Terson's syndrome in cases other than subarachnoid bleeding **(3).** Characteristically patients developing intracranial hemorrhage and Terson's syndrome usually have lost consciousness, and may be in a coma for an extended period. Usually the intraocular bleeding occurs in both eyes. Although the vitreous blood can clear spontaneously, it can take a year or more, hence an operation called a vitrectomy is often performed to remove the blood and restore vision more rapidly especially in bilateral cases. Occasionally the intravitreal bleeding damages the retina, and sometimes later a layer of scar tissue develops on the surface of the retina. This so-called epiretinal membrane can be surgically removed if it interferes with vision. Occasionally retinal detachments follow Terson's syndrome, presumably caused by changes in the vitreous body influenced by the blood. These can be repaired with surgery as well **(21).**

Conclusion

The vitreous haemorrhage, including bilateral haemorrhages, is a relatively common finding in patients with subarachnoid haemorrhage and can be identified by careful serial clinical examination. Univariate analyses suggest that vitreous haemorrhage is an indicator of poor prognosis in subarachnoid haemorrhage. Thus it is important to note that although the cause of Terson's syndrome seems to be clearly identified, this disease can still pose a threat to patient's sight and life and requires an individual approach to every patient. For surviving patients, close ophthalmological evaluation and surgical treatment are recommended.

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A Case Report On Carotid Body Tumour: Anaesthetic Challenges and Literature Review

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Abstract

Carotid body tumours (CBT) are rare tumours arising at the bifurcation of the carotid artery. They are largely benign and present with pressure symptoms to surrounding structures, including the pharynx and cranial nerves IX-XII.

Introduction

The carotid body is a peripheral chemoreceptor located at the carotid bifurcation that is involved in mediating ventilatory responses to hypoxia and hypercapnia. Carotid body tumours are rare and usually benign tumours that arise from it. They are commonly seen in the third and fourth decade. Surgical excision is the recommended therapy for these tumours, and this poses several anaesthetic challenges. We present a case of difficult excision of an adherent tumour.

Case Presentation

A 32 year old male was reviewed in the surgical clinics with a 7 year history of a painless, gradually enlarging mass over the right neck of insidious onset. He reported a history of a sensation of choking when sleeping, but no dysphagia, odynophagia, or hoarseness of voice. There was no history of Trauma, Fever or Cough.

He had no known comorbidities and his past surgical history was only positive for repair of non-cervical injuries sustained following assault. He had no history suggestive of a functional tumour. His family history was unremarkable.

On examination a non-pulsatile mass was seen on the right side of the neck. The mass was non-tender, non-collapsible, and immobile in the vertical plane with absence of a bruit. On general examination, the patient was 80Kgs, 166cm in height, PR of 74beats/minute and Blood Pressure of 136/74MmHg. The airway examination was normal. CNS, CVS and abdominal examination were unremarkable.

Preoperative hemogram, Random blood sugar, electrocardiogram, electrolytes, renal function tests, and chest radiograph were normal. On magnetic resonance imaging, large well-defined ovoid intensely enhanced mass over right carotid bifurcation insinuating both internal and external carotid arteries and confirmed the findings of the Doppler study. The '125cc right carotid body tumour was encasing but not occluding 1.7cm of the distal common carotid and 6cm of the proximal carotid'.

The patient was diagnosed as having right CBT and planned for excision under general anesthesia.

The patient was premedicated with Tab. Midazolam 7.5 mg and Tab. Ranitidine 150 mg per oral night before surgery. He was kept Nil Per Oral for 6 h before surgery. On arrival to the operating room, noninvasive blood pressure (BP) 130/70 mmHg, heart rate of 81/min, respiratory rate of 17b/min, and oxygen saturation of 98% were recorded. He was put on continuous ECG Monitoring.

The anaesthetic plan included general anaesthesia with a volatile agent, endotracheal intubation and positive pressure ventilation, standard ASA, arterial and central venous pressure (CVP) monitoring, and recovery in a critical care unit. The allowable blood loss was calculated as 1300ml. We targeted to maintain mean arterial pressures (MAP) above 60mmHg to ensure adequate cerebral perfusion pressures, and to maintain CVP around 10. Temperatures of 34-36 Celsius were acceptable for neuroprotection. We also ensured oxygen saturations above 94%.

An 18G cannula in the right forearm was used for induction which involved a ringer's lactate drip, Midazolam intravenously at 0.05mcg/kg, Fentanyl 2mcg/kg, Sodium Thiopental 375Mg, Cisatracurium 16Mg. A size 7.0 endotracheal tube was inserted without difficulty under direct laryngoscopy and a Comark-Lehane I recorded. Isoflurane was then used for maintenance with end tidal values of between 1.1 and 1.6.

A left radial artery was cannulated with 20 G, aseptically using modified seldinger technique. It was transduced for Invasive Blood Pressure Monitoring. A 7Fr right subclavian central venous catheter was sited aseptically. Also inserted were a urethral catheter and a temperature probe was placed in the right nostril.

Other medications administered included 1 .5g Cefuroxime, 8mg Dexamethasone, 2g Tranexamic acid, 8 mg Morphine and 1g Paracetamol were given.

Total anaesthesia time was 4.5 hours. During the course of surgery, a phenylephrine infusion at 0.4-0.6mcg/kg/min was used to maintain MAP above 75mmHg. There was one episode of profound bradycardia associated with hypotension for which 0.6mg Atropine and Ephedrine boluses up to 24mg were given. Two capillary random blood sugar samples were within normal range.

There was profound blood loss intraoperatively estimated at 2000ml, associated with hypotension. This was attributed to the fact that tumour was found to have infiltrated the lumen of the internal carotid artery and become strongly adherent, with associated discovery of several enlarged cervical nodes. 4L Crystalloid, 500ml Voluven and two units of packed red cells were given in total with an accompanying urine output of 1300ml.

Local infiltration with 10ml of 0.5% Bupivacaine was done by the surgeons.

There were no marked derangements from normal in the arterial blood gas analysis at the end of surgery. Reversal of neuromuscular blockade was done, the patient successfully extubated awake, and transferred to the ICU once absence of respiratory distress was confirmed. A deviation of the tongue to the left was noted, and there were no other neurologic deficits at this time.

Post operatively, the patient developed a hematoma at the surgical site that required drainage under General Anaesthesia on the second post op day. He was subsequently discharged four days later with no other complications.

Discussion

The carotid body is a paired structure located at the bifurcation of the common carotid artery. It comprises tissue that is highly vascularized with branches from the carotid artery. It is made up primarily of Type 1 (Glomus) cells that are of neural crest origin, and Type 2 cells that are similar to glial cells of the CNS. It functions as a chemoreceptor that largely mediates the ventilatory response to hypoxemia and partly contributes to the response to hypercapnia. It is innervated by the Nerve of Herring, a branch of the Glossopharyngeal nerve.

Carotid body tumours are slow-growing hyper vascular neuroendocrine neoplasms that occur within the adventitia of the common carotid artery near its bifurcation. They are the most common type of paragangliomas of the head and neck. They arise within the glomus cells of the carotid body.

They are rare tumours with a reported incidence of 1-2 per 100,000. The male to female ratio is equal and they occur more commonly in the 3rd and 4th decades.

Three types of CBTs are described; these are Familial (10%), Sporadic (up to 85%) and Hyperplastic. The hyperplastic tumours occur in response to chronic hypoxic states including COPD, cyanotic heart disease and high altitude.

They occur bilaterally in 10% of sporadic cases and in 30% of familial cases. These tumour are largely benign but have a propensity to turn malignant in 10% of cases. They tend to be asymptomatic and are commonly discovered as incidental findings during physical examination or on imaging. They can also present as functional tumours that secrete hormones including histamine, serotonin, epinephrine and norepinephrine. They can also occur in association with pheochromocytomas.

CBTs that grow to be large can present with pressure symptoms to surrounding structures including the sympathetic chain and cranial nerves VIII-XII, causing dysphagia, hoarseness, choking, Horner's syndrome, or altered airway anatomy if there is encroachment of the para-pharyngeal spaces.

In order to avoid local invasion and metastasis, early surgical excision is considered as the curative treatment option for the treatment of CBTs. Radiotherapy is reserved for tumours that are inoperable, recurrent or bulky.

Excision of CBT has been associated with a morbidity of up to 30%. There are inherent risks of bleeding, cerebral hypoxia, hypertensive crises, and cerebrovascular accidents. Carotid cross-clamping is associated with a period of cerebral hypoperfusion and necessitates interventions to avoid hypoxia. Injury to the vagal nerve or its branches could potentially lead to respiratory embarrassment at extubation.

Our patient did not exhibit any features of a functional tumour and as such no endocrine evaluation was done pre-operatively. Functional tumours may require pre-operative use of alpha and beta blockers and intraoperative use of the potent vasodilators for blood pressure control.

Bradycardia due to stimulation of the carotid sinus as was encountered may be treated with atropine. Infiltration of lignocaine at the surgical site has been reported to reduce the sensitivity of the carotid sinus. Severe episodes may require pacing.

Our anaesthetic plan was aimed at reducing cerebral metabolic rate and maintaining adequate cerebral perfusion. Sodium thiopental was chosen for its properties of reducing CMR.O2 Isoflurane was appropriate since it reduces CMRO2 and causes cerebral vasodilatation with minimal increase in ICP. Hypocapnia was avoided to prevent cerebral vasoconstriction. A vasopressor infusion was used to maintain MAPs well above 60mmHg to ensure cerebral perfusion pressures above 80mmHg. We avoided cannulation of the contralateral internal jugular vein to ensure adequate venous drainage from the cranium so as to prevent rise in ICP. Random blood sugar testing was also done to rule out hypo- or hyperglycemia

Conclusion

A high vigilance is necessary for an anesthesiologist during the excision of CBT. Cerebral protection, hypotension, and management of arrhythmias are challenging. A high index of suspicion should be made for possible complications that may occur during the perioperative period. The successful management depends upon detailed history, specific investigation, invasive monitoring, optimization, and managing postoperative complications.

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Ketesse

Dexketoprofen trometamol 50 mg/2 ml

Solution for injection or concentrate for solution for infusion

High analgesic potency

Therapeutic Indications

Symptomatic treatment of acute pain of moderate to severe intensity, when oral administration is not appropriate such as postoperative pain, renal colic and low back pain [2]

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