THE ENDOVASCULAR TREATMENT OF TRAUMATIC CRANIO-CERVICAL VASCULAR INJURIES

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DECLARATION

I, Peter Kato Ssenyonga hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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Date: 15th March, 2013.

PART A: STUDY PROTOCOL (*1474 words*)

THE ENDOVASCULAR TREATMENT OF TRAUMATIC CRANIO-CERVICAL VASCULAR INJURIES

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Introduction:

Cranio-cervical vascular injuries result from either blunt or penetrating trauma. The injuries sustained may be intradural or extradural. Because some of these lesions are located near the skull base, they are particularly hazardous to approach and difficult to repair surgically. The vertebral arteries run in the transverse foramina of C6 to C1, making them difficult to access surgically. [1]

Vascular injuries may be caused by high or low velocity penetration. The mortality from these injuries is approximately 2% to 6 %. [2]

Blunt traumatic cerebro-vascular injury is defined as an extracranial or intracranial cerebro-vascular structural defect that is directly attributable to a known high-energy non-penetrating injury. [3] Blunt vascular injuries are mainly caused by high-speed motor vehicle accidents, motorcycle crashes, pedestrians struck by motor vehicles, falls, assaults, hangings and near hangings. [2] Vascular injuries of the neck occur in approximately 1% to 3% of all major blunt trauma. The overall mortality related to blunt injuries is 20% to 30%. In addition, 37% to 58% of patients develop permanent neurologic deficits attributable to loss of blood supply to the central nervous system.[2]

Risk factors for blunt cranio-cervical vascular injuries as per The Eastern Association for the Surgery of Trauma Guidelines 2010 include:
- GCS ≤8
- Petrous bone fracture
- Diffuse axonal injury
- Cervical spine fracture
- Fracture through the foramen transversum
- Lefort II or III facial fractures [4]

With both blunt and penetrating injuries, a spectrum of vascular injuries may result which include spasm, thrombosis, arterio-venous fistulas, arterial dissections, dissecting aneurysm and complete transections. [1]

Blunt cranio-cervical vascular injuries may be graded according to the Biffl et al grading scheme as:
Grade I – intimal irregularity with <25% narrowing
Grade II – dissection or intramural hematoma with >25% narrowing
Grade III – pseudoaneurysm
Grade IV – occlusion
Grade V – transection with extravasation [5]

Patient presentation is variable and may be asymptomatic. Symptomatic patients may present with:
• Arterial hemorrhage
• Cervical bruit
• Expanding cervical hematoma
• Focal neurological deficit
• Neurologic examination incongruous with CAT scan findings
• Ischemic stroke on secondary CAT scan [4]

The principles of management of an arterial injury are to repair the artery while ensuring that the lumen remains patent. However, in some cases such as an arterio-venous fistula, occlusion of the artery may be required. This may be accomplished by deploying balloons, the use of stents (open structured or covered) or the use of coils or NBCA glue. The procedure is usually done transarterial but may be done transvenously in certain cases.

Cranio-cervical vascular injuries are often associated with other local injuries, which particularly in an acute setting make accessibility more complicated.

Rationale for the Study:

Surgical repair of injured vessels requires adequate vessel exposure to achieve proximal and distal vessel control. This may result in significant surgical mortality and morbidity. Some studies report the surgical morbidity as high as 16% and neurological complications from the surgical procedure in nearly 25% of survivors. [6] Ligation or occlusion of the Internal Carotid Artery carries a mortality of 47%. Preservation of the carotid artery circulation by either direct repair or interposition of graft where possible has a mortality of 15-20%. [7]Endovascular techniques and devices however, permit minimally invasive arterial repairs to be performed from easily accessible sites, remote from the area of arterial trauma. This study seeks to assess if endovascular treatment of cranio-cervical vascular injuries due to trauma is an effective treatment strategy that lowers morbidity and mortality.

Hypothesis:

Endovascular treatment of cranio-cervical trauma provides a treatment modality comparable to the traditional open surgery methods as per the literature.

Methods:

1. Study Design

A retrospective review of all patients with cranio-cervical vascular injury secondary to trauma treated endovascularly at Groote Schuur Hospital in a five year period from January 2006 to December, 2010.

2. Subject identification or selection

Patients will be selected by the Principal Investigator from a neurovascular database that includes all patients that underwent cervical and cerebral angiograms. All patients with lesions secondary to trauma and were endovascularly treated will be selected. A folder review of the selected patients will be done to determine the mechanism of injury, location and nature of the vascular lesion, time interval between injury and angiogram, ancillary investigations done and outcomes.

The angiograms and ancillary investigations done will be reviewed by the Principal Investigator and the Neurovascular consultants; (Professor Allan Taylor and Dr. David Le Feuvre) The nature and location of lesions, the modality of treatment used and the outcome will be identified.

3.Exclusion Criteria include:

Patients under the age of 13 years
Patients without evidence of cranio-cervical trauma
Patients with incomplete data fields

4. Outcome Variables:

The primary outcomes measures for this study will include Glasgow Coma Scale score on admission, Glasgow Outcome score post procedure; Achievement of set out goal to treat the vascular injury; Procedure related complications and neurological deficits attributed to the intervention. Secondary outcome measures will include location of the vascular lesion, type of vascular lesion, time interval to treatment, retreatments and maintenance of patency of the parent artery.

5. Data Collection

Instruments:

An electronic data capture sheet was designed for collation of data from patient folders, Brain CT scans and Cerebral angiograms.

List of Variables:

Demographic Data: Gender and age

Clinical Data: Date of admission, time from admission to diagnosis, time from diagnosis to intervention and deterioration if any will be noted, mechanism of injury, Admission Glasgow Coma Scale Score, presenting features due to vascular lesion, associated injuries.

Brain CT scan: Date of imaging, time interval between imaging and presentation, presence of infarcts or hemorrhage.

Cerebral Angiogram (CTA/DSA): Location of lesion (intradural or extradural), number of lesions, nature of lesion, type of intervention,

Intervention: Intervention Modality (glue, coils, detachable balloons, stents or a combination), whether patency of parent artery maintained, goal of treatment achieved, procedure complications, retreatments and neurological deterioration attributable to the procedure.

Follow up: Glasgow Outcome Score, Duration of follow up and development of new symptoms and/or signs if any.

6. Analysis:

The collated data will be statistically analyzed.

Ethics and Communication:

Ethics:

The study protocol will be presented to the Surgical Departmental Research Committee (DRC) for approval, following which it will be sent to the Ethics Committee with a copy of the synopsis for Ethics approval.

The data collation will commence as soon as the protocol has been approved. The folders of patients that had traumatic cranio-cervical vascular trauma as per the database will be requested for analysis. The folders will be reviewed in the Records Department for safety and to maintain patient privacy.
The radiographic imaging will be requested from Radiology filing. This will be kept safe in a locked room that is only available to the research team. The imaging will only be requested for a short period of time to allow for the analysis by the research team. As soon as the review process is finished, the imaging will be sent back to the records department. The collated data will be entered into an electronic data sheet.
Patient confidentiality will be maintained by following a strict protocol. The patient data will be entered into a computer immediately. The computer and research data file will be secured by a password that is only known to the reviewers. A computerized data sheet will be used to prevent having multiple data cards that can be lost with patient information.
As patient names are not required, the information will remain confidential and anonymous.

Reporting and Implementation:

The results of the study will be submitted to a peer review journal for publication and will be the basis of an MMed thesis.

Logistics:

This will be a retrospective study.
There is no funding allocation for the study.

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PART B: LITERATURE REVIEW *(4536 words)*

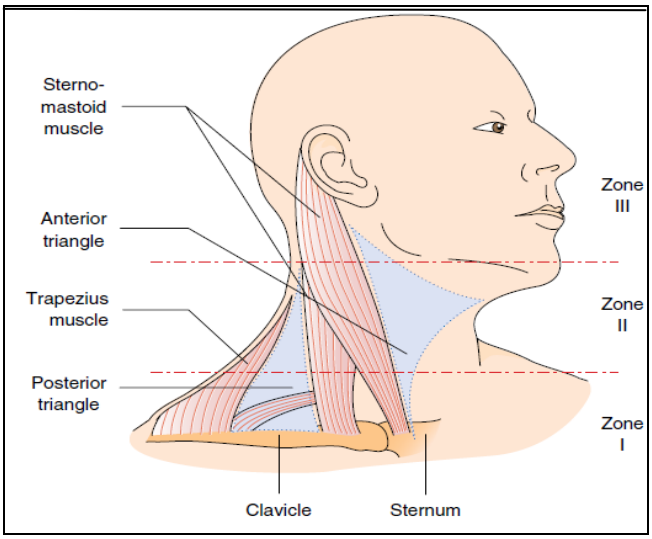
Introduction

French surgeon, Aristide Verneuil, in 1872 reported the first case of post traumatic carotid thrombosis. This was on necropsy findings in a case of multiple injuries resulting from a railway accident. Left cerebral infarction had resulted from thrombosis of the left internal carotid artery. The tunica media and intima were torn, although the adventitia had remained intact. The thrombus was extending up into the middle cerebral artery and its branches. There was some swelling of the sternocleidomastoid muscle. [1,2] J. Guibert described a traumatic intracranial aneurysm in 1895. This also, was on autopsy. The patient had presented with epistaxis. It was a bled traumatic aneurysm of the internal carotid artery in the cavernous sinus that communicated with the sphenoid sinus. [5] In 1943, Erikson reported a case of traumatic internal carotid artery occlusion at the skull base. [57] In 1944, Northcroft and Morgan reported a traumatic dissection of the left internal carotid artery in a patient who had been hanged accidentally. [58] In 1947, Dratz and Woodhall reported an intracranial internal carotid artery dissection. [59]

Since the 1970's, there has been an increase in the number of traumatic cranio-cervical vascular injuries reported. This is attributed to advanced imaging technology and adherence to protocols that screen for these injuries. [4]

Traumatic cranial vascular injuries may be classified into intracranial or extracranial lesions. The cervical vascular lesions are classified according to the zones of the neck. Zone I extends inferiorly from the cricoid cartilage. Zone III extends superiorly from the angle of the mandible to the skull base. Zone II includes everything between zones I and III, from the cricoid cartilage to the angle of the mandible. This helps with identification of the lesions and management.

Diagram showing the zones of the neck



Adapted from Van Waes et al, 2002 [12]

Incidence

Previously, traumatic cranio-cervical vascular injuries were estimated to occur in 0.08% of all blunt trauma. [4] With increased screening for these injuries, they are now estimated to occur in 1% - 3% of all patients of blunt trauma. [6,7,8,9,10] Vertebral artery injuries occur in 3% - 19% of all cervical vascular injuries. [11] Cervical vascular trauma occurs in 20% of all penetrating neck injuries with a mortality of about 50%. [12]

Cranio-cervical vascular injuries mainly affect the young. There is a male predominance which may probably reflect the participation of these patients in activities that put them at a higher risk of getting these injuries. [3,13] The age range is 5-38 years with a mean of 17 years.[3]

Risk Factors

Risk factors are best categorized under mechanism of trauma.

In blunt trauma, this can further be subdivided into intracranial and extracranial vessel injuries. For the extracranial vessel injuries, thoracic injuries and Glasgow Coma Scale (GCS) of 8 or less are the commonest predictors of vessel injury. [9,14] For intracranial vascular injuries, GCS of 8 or less and Facial fractures are the commonest predictors of vessel injury. [9] Berne and his colleagues found cervical spine fracture, basilar skull fracture, and mandible fracture to be most predictive of the presence of blunt cerebrovascular injury. [10] Blunt vertebral artery injury is associated with complex cervical spine fractures involving subluxation, extension into the foramen transversarium, or upper C1 to C3 fractures. [15,16]

Penetrating vascular trauma is suspected when there are hard signs like profuse bleeding, expanding haematoma, bruit and pulse deficit [13]

Pathogenesis

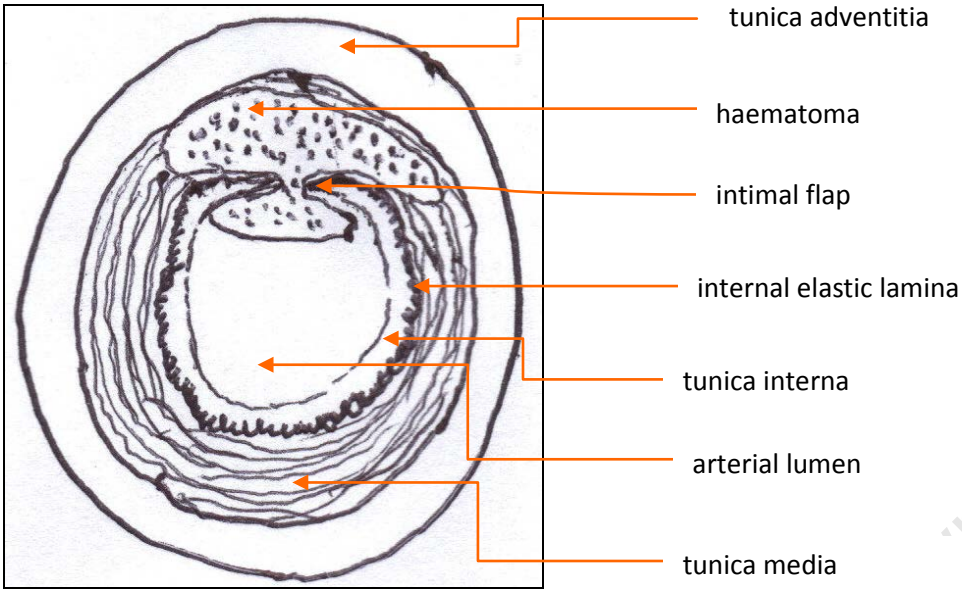
The vessel wall has three layers, the tunica intima, tunica media and tunica adventitia. The tunica intima consists of the endothelium and subendothelial connective tissue. In between the intima and media is the internal elastic lamina which provides mechanical strength to the blood vessel. [18] The tunica media consists of closely packed layers of smooth muscle cells surrounded by collagen and elastin fibers. The external elastic lamina divides the tunica media and tunica adventitia. The tunica adventitia is composed of collagen fibres. It is responsible for protection and anchoring of the vessel to surrounding tissue. Within this layer are the vasa vasorum. The intracranial arteries lack the external elastic lamina and have a thinner adventitia when compared to vessels of similar luminal diameter in other parts of the body. Intracranial arteries also have a lower wall:lumen ratio than extracranial arteries. [18,19]

Intradural arteries are surrounded by subarachnoid space. The vertebral artery and carotid artery have sections where they traverse a venous plexus namely; cavernous sinus and vertebral venous plexus respectively. [20]

Blunt trauma, mainly due to motor vehicle accidents, assaults, seat belt injuries, extreme neck movements like in chiropractic manoeuvres may result into disruption of the intima. This leads to exposure of the subendothelial tissue which is thrombogenic causing platelet aggregation and clot formation. Distal extension of the thrombus with or without embolisation may explain the interval between incidence of trauma and presentation of neurological symptoms. [2] Trauma also weakens the internal elastic lamina (IEL). Vibration from arterial blood flow lead to widening of the tear in the IEL. [21]

Penetration of blood into the media leads to a dissection. The haematoma may remain within the layers of the arterial wall and progressively continue to dissect along the plane of the circular muscle layers. The increase in size of the subadventitial haematoma may lead to occlusion of perforators from the parent artery leading to local ischaemia or stenosis of the parent lumen. [20] A narrowed lumen causes disturbances in blood flow, which acts as a catalyst for further clot formation. Sometimes, the narrowing may be critical leading to insufficient blood supply with resultant ischaemia. [20]

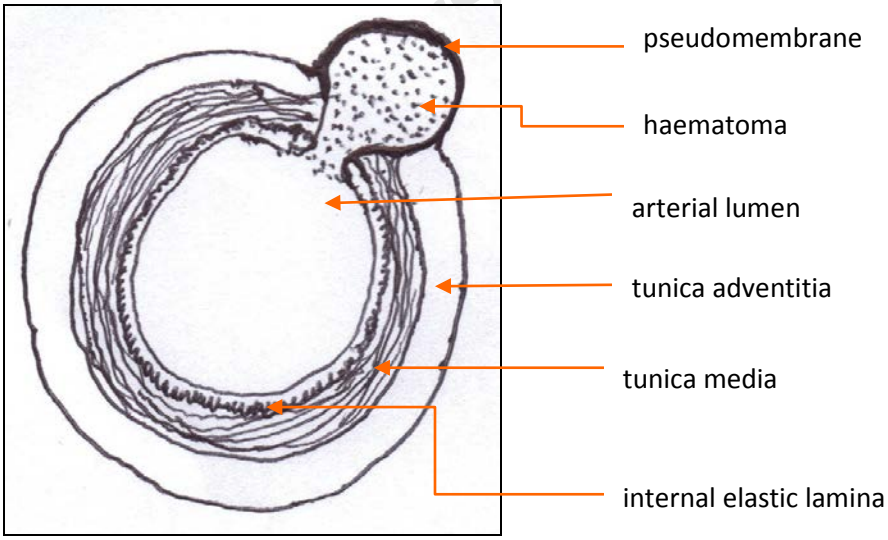
Dissection:



The dissecting haematoma may regain access to the parent vessel lumen. This can lead to distal embolization of the clot.

If the haematoma penetrates all the layers of the blood vessel wall, a transmural dissection is formed. If blood gets into the subarachnoid space, this leads to a subarachnoid haemorrhage. At times, the haematoma forms a pseudo membrane that is contained by the surrounding soft tissues leading to a false aneurysm or it may communicate with a surrounding venous system leading to an arteriovenous fistula. [20]

False Aneurysm:



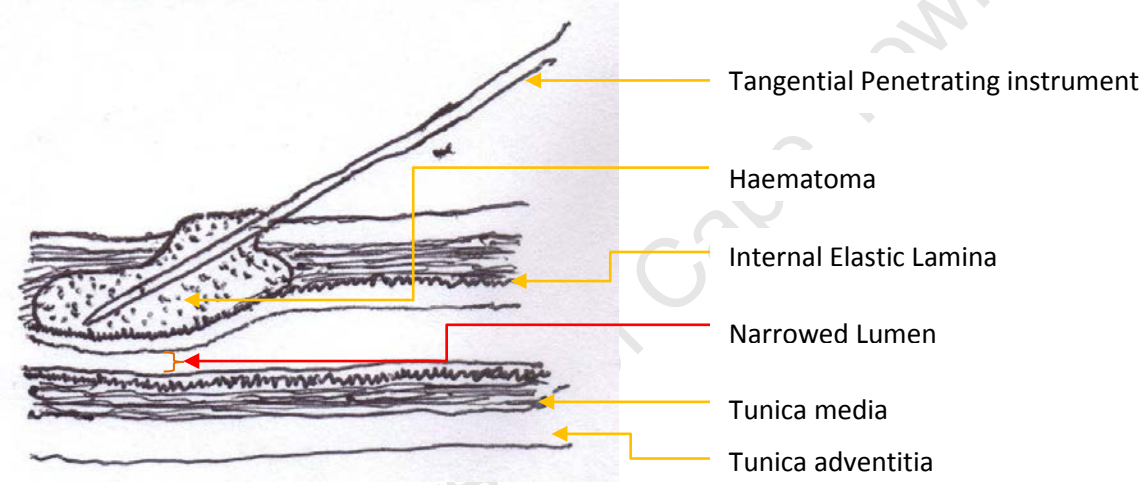
Following the injury, the vessel undergoes an adaptation and remodelling process in response to the hyperdynamic stress. There is thickening of the intima with

accumulation of collagen. This may be followed by complete or incomplete vessel healing. [21] If the healing is incomplete, it will lead to a chronic disease process.

Penetrating trauma leads to disruption of the vessel wall. All the vascular layers could be penetrated leading to profuse bleeding with or without exsanguination. Formation of an clot with associated muscle spasm may taper the active bleeding leading to a false aneurysm.

There is a possibility of a tangential penetrating injury that may not penetrate the internal elastic lamina. Bleeding from the vasa vasorum will lead to a dissecting process with resultant luminal stenosis. This may lead to insufficient blood flow or turbulence at the site of narrowing. The clot formed may be disseminated distally leading to ischaemia.

Trans-adventitial Dissection:



Penetrating trauma may lead to complete vessel transection. This followed by vessel spasm and thrombosis may lead to complete vessel occlusion.

The internal carotid artery has three points of transition from a mobile segment to a fixed one. At these points it is susceptible to traumatic injury. These points are; the entry point of the internal carotid artery into the carotid canal of the petrous bone, at the point of dural penetration and immediately distal to the posterior communicating artery. [3]

The orbits are funnel shaped with the pointed end towards the cavernous sinus. The internal carotid artery is fixed as it passes through the cavernous sinus. Penetrating injuries through the orbits are inadvertently channelled towards the cavernous sinus making the internal carotid injury highly susceptible to injury as it traverses through. The temporal bone is the thinnest bone of the cranium and is easily penetrated with stabs. On penetration, the sphenoid ridge is in the way diverting the angle of penetration

towards the cavernous sinus. This explains why such injuries usually lead to carotid-cavernous fistulas.

Pathophysiology:

Penetrating injuries to the neck and head and blunt trauma to the face may lead to arterial injuries with resultant profuse bleeding. Patients may present with severe hypotension or in haemorrhagic shock. Venous injuries are easily controlled with pressure.

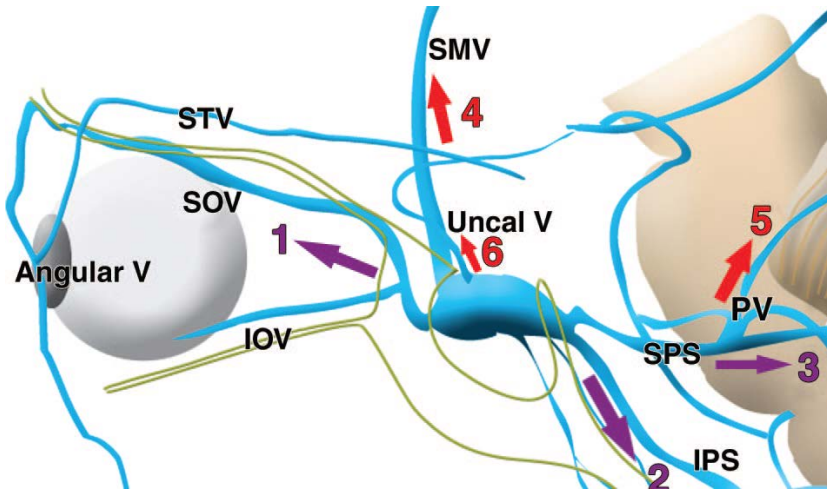
Sometimes, the transmural haematomas formed may be contained by the surrounding local tissue. With continuing extravasation of blood, this leads to formation of an expanding haematoma. In the cervical region, it may cause obstruction of the airway, pharynx or compression of the cranial nerves. If formed in the intracranial cavity, it will lead to an intracranial bleed with mass effect and raised intracranial pressure.

The intramural haematomas lead to parent lumen stenosis which may result into occlusions. Alternatively, they may compress distal perforators resulting into distal ischaemia. [20]

Fistulas result from direct communication between two vascular structures. Direct communication of the artery with a vein leads to increased blood flow and pressure transmitted into the venous system. The venous hypertension and hyperdynamic circulation may cause cardiac failure and venous compression of the nerves.

Traumatic direct carotico-cavernous fistula (CCF) results when the integrity of the cavernous portion of the internal carotid artery (ICA) is disrupted, creating an abnormal communication between the high-pressure arterial systems and the low-pressure venous system. [22] The cavernous sinus has numerous drainage systems and each gives rise to different symptoms. Raised pressure in the superior and inferior ophthalmic veins leads to ocular symptoms. Increased pressure in the inferior petrosal sinus, basilar and pterygoid plexuses leads to bruits and cranial nerve palsies. Hypertension in the superior petrosal sinus, cortical reflux of blood into the sphenoparietal sinus and superficial middle cerebral vein leads to venous infarction and haemorrhage. Compression of the oculomotor, trochlear and abducent cranial nerves as they traverse the cavernous sinus leads to an ophthalmoplegia. [23] In traumatic indirect CCF, the intracavernous portion of ICA is intact. Arterial blood flows through the meningeal branches of external carotid artery or ICA or both, into the cavernous sinus.[22,60]

Diagram to explain flow of blood out of the cavernous sinus



IOV - inferior ophthalmic vein
SOV - superior ophthalmic vein
IPS - inferior petrous sinus
SPS - superior petrous sinus
SMV - superficial middle cerebral vein
PV - petrous vein
STV - superficial temporal vein
Adapted from: Kiyosue et al, 2004 [23]

With the understanding of the pathogenenesis, it is easier to deduce the possible pathophysiology and resultant clinical presentations.

Clinical Presentation:

Blunt injury is more likely to result into delayed lesions like dissections. Symptoms from thromboembolic phenomena and distal ischaemia take some time to present providing a lucid interval. Traumatic intracranial aneurysms and fistulas may present acutely or more indolently well after the inciting event. [24]

Most of the symptoms in carotid thrombosis are typical of those caused by carotid – artery insufficiency, and include alteration in level of consciousness, dysphasia, and aphasia, convulsions and hemiparesis or monoparesis. [1]

An ipsilateral Horner's syndrome is a common mainifestation of extracranial internal carotid artery dissection and may precede the onset of ischemic symptoms. [3]

Penetrating trauma is often associated with acute presentation with profuse arterial bleeding with exsanguination and haemorrhagic shock and expanding haematomas causing compression of adjacent structures. [3]

Carotid-cavernous fistulas mainly present with ocular signs of proptosis, chemosis and a bruit. [25]

Investigations

Controversy still exists on whether it is necessary to screen asymptomatic patients for cranio-cervical vascular lesions. Those for the screening sight the high morbidity and mortality associated with untreated lesions. They show improved outcome in the treated patients. [26,27] The school against screening counters that it is not cost effective to screen for the asymptomatic injuries. [28] Long-term follow-up review generally shows that 40% of dissections and perhaps 100% of false aneurysms do not “heal” with anticoagulation therapy and constitute a long term risk of embolization or flow-related complications. [43]

Two well established screening criteria exist for blunt cerebrovascular injuries (BCVIs); the Denver criteria and the Memphis criteria. The Denver criteria was the earlier screening protocol. In their study, 4.8% of the blunt trauma patients underwent screening with BCVIs identified in 18% of these patients giving an overall incidence of 0.86%. [26] In the Memphis study, 3.5% of the patients were screened. BCVIs were identified in 29% of these patients resulting in an overall incidence of 1.03%.[29]

Screening criteria for blunt cerebrovascular injuries

Denver Criteria	Memphis Criteria
Signs/symptoms	
Arterial haemorrhage or expanding haematoma	Cervical spine fracture
Cervical bruit	Neurological exam not explained by brain imaging
Focal neurological deficit	Horner’s syndrome
Neurological exam inconsistent with head computed tomography (CT) findings	Le Forte II or III fracture pattern
	Basilar skull fracture with involvement of the carotid canal
Stroke on follow-up head CT	Neck soft tissue injury (seatbelt sign or hanging or haematoma)
Risk factors	
Le Forte II or III fracture pattern	
Cervical spine fracture	

Denver Criteria	Memphis Criteria
Basilar skull fracture with involvement of the carotid canal	
Diffuse axonal injury with Glasgow Coma Scale <6	
Near hanging with anoxic brain injury	

Arthurs ZM and Starnes BW. 2008 [30]

Digital Subtracted Angiography:

Digital Subtracted Angiography (DSA) is considered the gold standard in diagnosing cranio-cervical vascular injuries. It assists in defining the arterial anatomy and venous drainage and is highly accurate. One gets an idea of the flow dynamics which aids in planning for the treatment. The presence of the hard signs following injuries to the neck warrants angiography. [28] Disadvantages of this test is that it is invasive, technically demanding and expensive.

Computed Tomographic Angiography (CTA):

Since the onset of Multidetector Computed Tomography (MDCT), there has been an increase in its use to screen for injuries of the head, neck, spine, chest, abdomen and pelvic injuries. A number of studies have shown that the accuracy of CTA approaches that of DSA. [32,33,34,35,36] The investigation is quick and non-invasive. Also, the CTA can be done in the same sitting with the other routine trauma investigations.

Magnetic Resonance Angiography (MRA):

While MRA has a sensitivity of 50–75% and specificity of 67–100% for cranio-cervical vascular injuries. [37,38] Inspite of the good anatomical detail it provides, it is not a practical option in a trauma setting. This is because some patients come in with retained shrapnel or blades which may cause further vessel or organ damage when imaged. MRI suites are usually located remote from the trauma units making the imaging of patients even more difficult. MRA offers the advantage that no contrast is needed and no ionising radiation is used.

Duplex Ultrasound Scan:

Studies to evaluate the use of Duplex Ultrasound scan to identify traumatic vascular injuries have found that it has a low sensitivity and specificity when compared to DSA.[39,40] Duplex Ultrasound scan has limited use in vascular injuries not associated with disruption of flow and vasculature at the base of the skull. The results are operator dependent.

The challenge still remains on how to best investigate cranio-cervical trauma. MRI while providing good anatomical detail is impossible to use in an acute trauma setting and in

patients with retained shrapnel. It can however give useful information on the arterial blood flow. Putting aside, its great expense to maintain, it might play a much bigger role in future investigation of the subacute and chronic presenting lesions. The use of CT use is in ascendency. The multidetector CTs provide good anatomical detail. CTA falls short when it comes to providing flow characteristics across the lesion.

Treatment

Medical Management:

Some traumatic cranio-cervical lesions can be managed conservatively with a benign follow up course. This is when the lesions are asymptomatic. In patients with demonstrable intracranial emboli, anticoagulation and antiplatelet therapy may be appropriate provided there is no intracranial haemorrhage or active bleeding elsewhere. [6] Anticoagulation is usually initiated with systemic Heparin followed by Warfarin for about six weeks. Alternatively, antiplatelet therapy with Aspirin or Clopidogrel is used or a combination of antiplatelets and anticoagulants. [41,42,43,44]

Surgical Treatment:

Patients with active bleeding require urgent surgical intervention.
Pemberton and Livermore in their study as reported by Little and colleagues in the Lancet (1969) found that ligation of the internal carotid artery carried a 15.7% death rate, with cerebral complications in nearly 25% of survivors. [1]
Karlin and Marks in their retrospective review of 50 patients with extracranial traumatic carotid artery injuries surgically treated over a period of 27 years found a 7.8% mortality in patients undergoing repair and 50% mortality in those undergoing ligation. 38 patients underwent repair and 12 had carotid artery ligation. There were no new neurological deficits after repair of the carotid artery injuries. Ligation of the carotid artery was performed when there was difficulty in achieving haemostasis. [55]
Kieck and De Villiers in their 1984 study showed good outcome in the trapping of CCFs in their earlier series, direct excision of 3 AVFs and the clipping, trapping or excision of 6 post traumatic aneurysms. They did not report any surgical related mortality. [45]

Lee and colleagues in their 2007 study of 6 traumatic vertebral artery injuries treated endovascularly showed no procedure related morbidity or mortality with a follow up period of 36.5 months. [11] Bell and colleagues in their 2010 retrospective study demonstrated successful endovascular treatment of 64 penetrating cranio-cervical vascular trauma during the Iraq war. [46] Ahn and colleagues in a 2004 retrospective study successfully treated 13 traumatic carotid and vertebral artery injuries. [47] Diaz-Daza and colleagues also showed successful endovascular treatment of cranio-cervical vascular injuries in 8 patients in their 2003 retrospective study. Though most of the evidence for endovascular treatment of traumatic cranio-cervical vascular injuries is Class III studies, they have all shown good results with the treatment with minimal morbidity and mortality. [48,49,50,51,52,56]

Endovascular treatment modalities are now preferred to the open surgical methods when treating penetrating cervical vascular injuries in zones III and I, [53] vertebral artery arteriovenous fistulas [54] and carotid-cavernous fistulas. [22]

Cranio-cervical vascular injuries lead to a diversity of lesions. Some lesions, though lumped under one pathological name like dissections, have different natural histories. The challenge remains on how to best select the lesions that will best respond to a specific treatment.

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PART C: MANUSCRIPT *(4538 words)*

THE ENDOVASCULAR TREATMENT OF TRAUMATIC CRANIO-CERVICAL VASCULAR INJURIES

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ABSTRACT

Background

Cranio-cervical vascular injuries comprise a heterogeneous group with differing mechanisms of injury and pathological lesions. Endovascular intervention has changed the way that these lesions can be approached, however little information is available aside from case reports as to the role that endovascular treatment may have.

Aim

To assess if endovascular treatment of cranio-cervical vascular trauma is an effective treatment strategy which lowers morbidity and mortality.

Method

A retrospective review of all patients undergoing a Digital Subtracted Angiogram (DSA) following traumatic injury to the head or the cervical region at Groote Schuur Hospital from 1st January, 2006 to 31st December, 2010.

Results

633 patients had Digital Subtraction Angiograms for trauma to the Cranio-cervical region at Groote Schuur Hospital for the period from 1st January 2006 to 31st December 2010.

Of these, 74 patients had lesions on DSA that could be treated by endovascular intervention. 82 injuries were diagnosed and 79 injuries were treated.

On angiography, patients were either classified as having vessel lacerations, false aneurysms (FAs), Fistulas or vessel occlusions.

Differing technical approaches were required depending on the diagnosis.

All patients were successfully treated.

There were 3 procedure related technical complications and 1 neurological complication as a result of the endovascular treatment.

Conclusion

Traumatic cranio-cervical injuries are associated with severe morbidity and mortality if left untreated. They need to be screened for most especially in the setting of blunt trauma where the clinical presentation is delayed. Though the injuries have traditionally been treated surgically, endovascular treatment offers a viable and safe treatment alternative.

KEY WORDS: Cranio-cervical vascular trauma Traumatic aneurysms ,Fistulas Dissections, Endovascular treatment

INTRODUCTION

Cranio-cervical vascular injuries comprise a heterogeneous group with differing mechanisms of injury and pathological lesions. Endovascular intervention has changed the way that these lesions can be approached, however little information is available aside from case reports as to the role that endovascular treatment may have.

Surgical approaches to head and neck vascular injuries are difficult due to restricted access and surrounding structures such as cranial nerves which may be difficult to identify and preserve especially after trauma. Endovascular approaches offer rapid access to vascular lesions with little risk to surrounding structures.

Penetrating injuries in this region may present acutely with extensive loss of blood or an expanding haematoma and compression of surrounding structures requiring rapid treatment. This is often associated with vessel wall disruption and blood extravasation or a false aneurysm. Symptoms may also develop later after an injury particularly with post-traumatic fistulas.

Blunt cranial injuries can result in carotid injury and carotid cavernous fistula because the vessel is vulnerable to injury where it is fixed within the cavernous sinus. Distal cerebral vessel injury is also possible where vessels may be stretched or compressed against dural edges of the falx or tentorium. Blunt cervical injuries tend to result in dissecting injuries of the carotid or vertebral arteries with associated distal embolic events and the challenge is early detection and institution of appropriate treatment.[1,2,3]

The ideal treatment of the injured vessel wall is repair while maintaining the integrity of the lumen. However, many traumatic lesions are life threatening and vessel closure may represent the most effective option.

We sought to review our experience with the endovascular treatment of cranio-cervical vascular trauma in order to characterize the lesions seen, tools used in their treatment and outcomes.

SUBJECTS AND METHODS

Patients were selected from the record of all patients undergoing a Digital Subtracted Angiogram (DSA) following traumatic injury to the head or the cervical region at Groote Schuur Hospital from 1st January, 2006 to 31st December, 2010. All patients who underwent endovascular treatment of a vascular injury were captured.
Thereafter, a retrospective folder review was conducted to determine the demographic and clinical data of the selected subjects.
The preliminary investigation in some patients was a CT angiogram or an Ultrasound scan. However, all subjects eventually had a DSA that identified a traumatic vascular injury in the cranio-cervical region.
The angiograms were reviewed in order to describe the location of vascular injury, the nature of lesion, modality of treatment and angiographic outcome.

The angiographic appearance was described as either vessel laceration, false aneurysms (FAs), fistula or vessel occlusion.
Complications due to the procedure and retreatments were noted as was the length of follow-up. Patients were then telephonically contacted to find out their current clinical condition and if they had returned to work.

RESULTS

633 patients had Digital Subtraction Angiograms for trauma to the Cranio-cervical region at Groote Schuur Hospital for the period from 1st January 2006 to 31st December 2010.
Of these, 74 patients had lesions on DSA that could be treated by endovascular intervention. 82 injuries were diagnosed and 79 injuries were treated.
Of the treated patients, 68 (91.9%) were male and 6 (8.1%) were female.
The mean patient age was 29.54 years, with a median of 27 years and mode of 26 years. The minimum age was 16 years with a maximum age of 74 years and a range of 58 years.

The Mechanism of injury and location of the vascular injury profile is summarized in Table 1. Twenty three patients had vascular injuries due to blunt trauma with 24 vascular injuries. Of these, 18 injuries were intracranial and 5 were extracranial with only one cervical injury. Penetrating trauma caused 58 vascular injuries in fifty one patients. 28 vascular injuries were extracranial, 13 were intracranial and 17 were cervical. Gunshot Wounds accounted for 8 vascular injuries. The remaining 50 injuries were due to stabs. 3 patients with penetrating trauma had double injuries with one located in the cervical and the other in the extracranial compartment.

Table 1: Mechanism of injury and Location vascular injury.

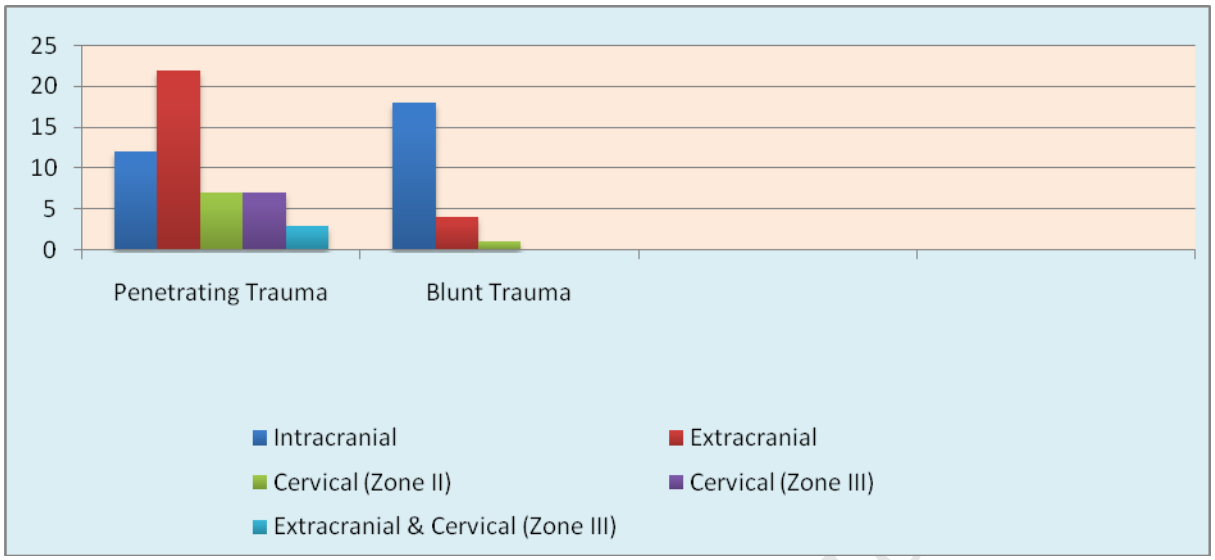
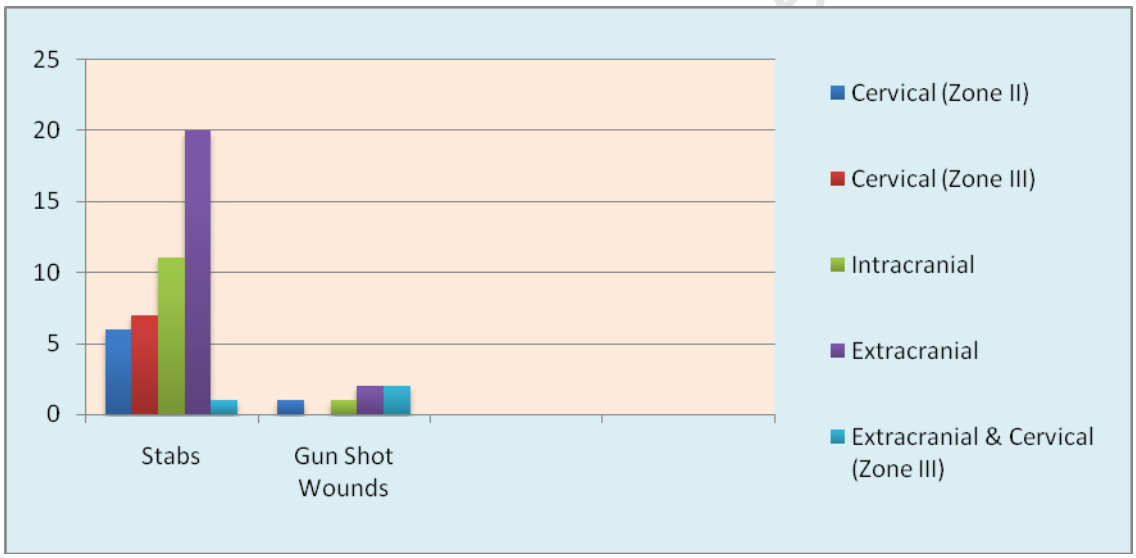
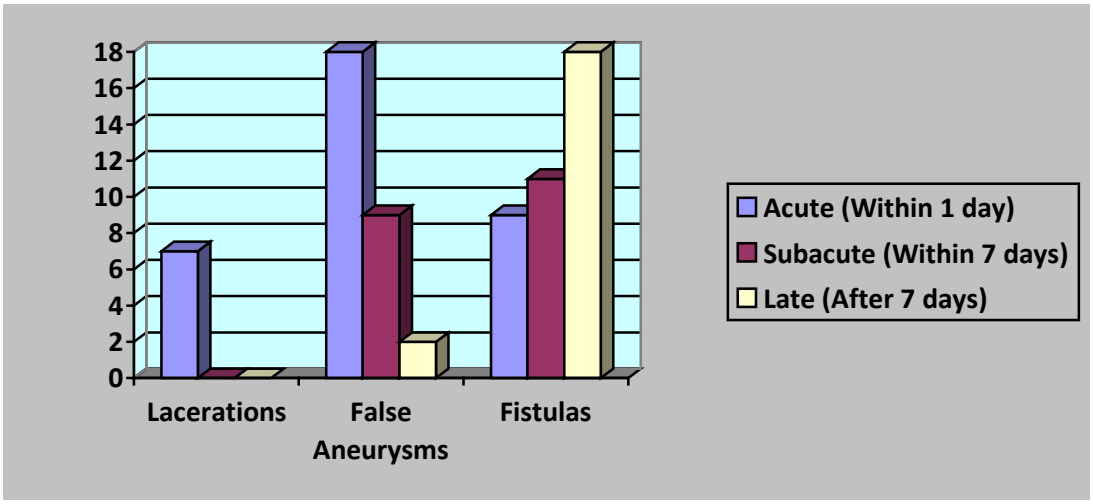


Table 2: Sub classification of the Penetrating Injuries depending on Aetiology



Thirty four of the patients presented acutely. Twenty patients presented in the sub acute phase and 20 had a delayed presentation. Table 3 shows the relationship between the time of presentation and the nature of the vascular lesions.

Table 3: Relationship of Identified Vascular Lesions to Time of Presentation



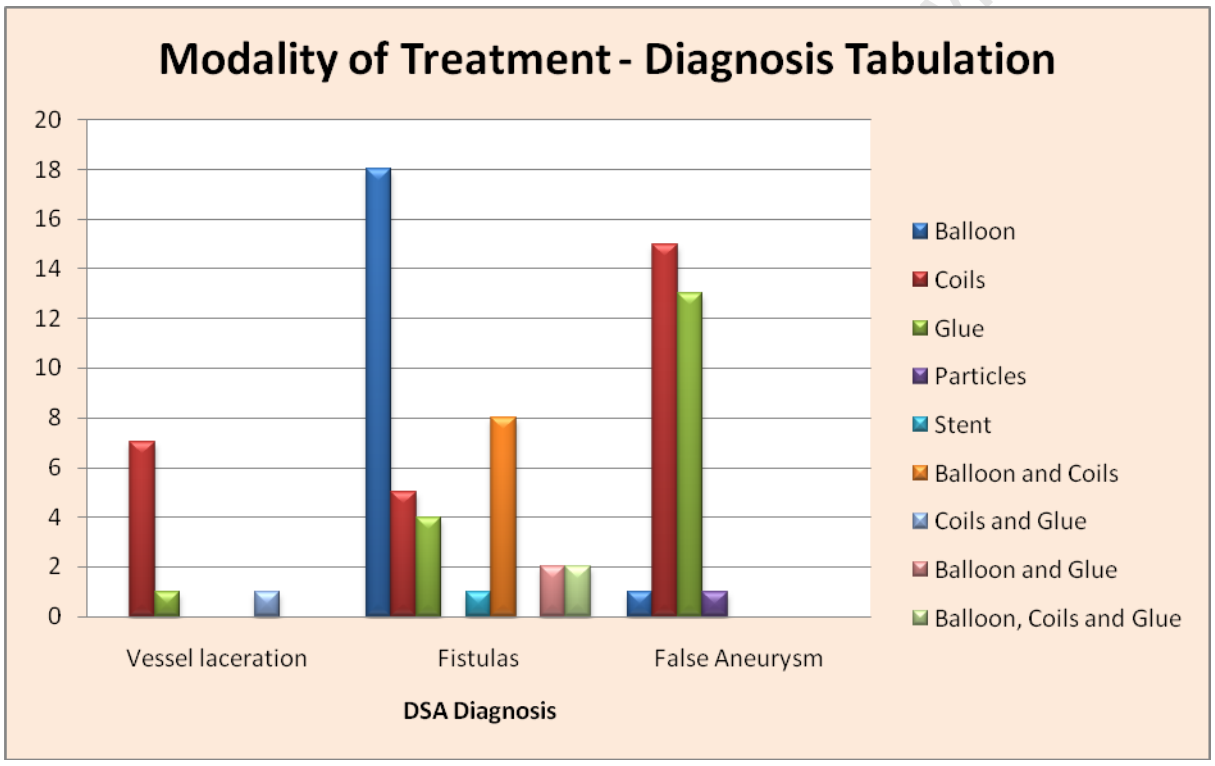
3 patients presented with focal neurological deficit due to ischaemia. They were all stabs to the neck with injury to the internal carotid artery. Two presented acutely and one presented in the subacute phase.
16 patients presented with active bleeding, 17 patients presented with expanding or pulsatile haematomas, 11 with local haematomas, 27 patients with symptoms and signs of CCF, 2 with retained blades and 1 with headaches and seizures.

79 injuries were treated endovascularly. The nature of the vascular lesions with the modality of treatment used is shown in the tables 4 and 5 below.
Vascular injuries identified after DSA included 33 FAs, 26 CCFs, 11 AVFs, 7 vessel lacerations and 2 VVFs.
8 patients had more than one vascular lesion attributable to the trauma. 5 of the lesions were treated endovascularly. The three lesions that were not treated were vessel occlusions due to penetrating trauma.

Table 4: Types of Vascular Injuries and How they were treated

		Balloon	Coils	Glue	Particles	Stent	Balloon and Coils	Coils and Glue	Balloon and Glue	Balloon, Coils and Glue
DSA Findings	Vessel laceration	0 (0.0%)	7 (8.9%)	1 (1.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (1.3%)	0 (0.0%)	0 (0.0%)
	Fistulas	18 (22.8%)	5 (6.3%)	4 (5.1%)	0 (0.0%)	1 (1.3%)	8 (10.1%)	0 (0.0%)	2 (2.5%)	2 (2.5%)
	False Aneurysm	1 (1.3%)	15 (19.0%)	13 (16.5%)	1 (1.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

Table 5: Graphical Representation of How Vessel Injuries were treated



Associated injuries to the cranio-cervical injuries included 22 traumatic brain injuries, 6 mandible fractures, 4 spine injuries, 4 base of skull fractures, 6 abdominal and chest injuries, 2 limb fractures, 2 pinna lacerations and 7 facial fractures. 35 patients did not present with associated injuries.

There were 3 procedure related technical complications and 1 neurological complication (Trigeminal Neuralgia) as a result of the endovascular treatment. 6 patients needed

retreatment. 4 of them were CCFs, 1 FA and 1 AVF. There was no procedure related mortality.

There was one patient, a stab to the cervical region (Zone II) with a left Vertebral artery - Internal Jugular Vein arteriovenous fistula that was initially treated surgically for injuries to the Common Carotid artery and Internal Jugular vein but he continued to bleed and was later treated endovascularly to stop the bleeding.
65 patients were independent on discharge, 8 were dependent and 1 died while in Intensive Care Unit (ICU) during the time of admission. He was a high cervical spinal cord injury patient.
35 patients followed up with Groote Schuur Hospital. The average follow up period for this cohort was 275 days with a range of 2 to 1951 days.

DISCUSSION

Mechanism of injury

The mechanism of traumatic injury in reported studies obviously differs between military series and civilian reports and this ultimately influences the types of injuries seen. Our series is dominated by low velocity penetrating injuries with blunt injury more likely to result in a cranial vascular lesion than a cervical lesion. In this series there was only a single cervical vascular injury from blunt trauma. He was a passenger in a motor vehicle accident who sustained a high cervical spinal cord injury with facet fractures to cervical spines 2,3 and 4 and a right sided Vertebro-vertebral fistula (VVF).
Blunt injury to the carotid or vertebral vessels is diagnosed in approximately 0.1% - 0.8% of patients hospitalized for trauma. [6,7] With the inclusion of asymptomatic patients screened for Blunt Carotid Vertebral Injuries (BCVIs), the incidence raises to 1% - 3% of all blunt trauma patients. [2,8,9,10] The overall mortality related to blunt injuries is 20% to 30%. In addition, 37% to 58% of patients develop permanent neurologic deficits attributable to loss of blood supply to the central nervous system. [10]

The overall incidence of traumatic intracranial vascular injury in penetrating head trauma is estimated to be between 3.8% and 5.7%. [11,12] When looking at severe penetrating head trauma alone, the incidence of aneurysm formation has been found to be 20% to 50% in some populations. [13]
Kieck and De Villiers reported that previous studies by Fleischer et al, 1975 and Laun et al, 1979 (survey) showed that blunt trauma was responsible for the majority of traumatic intracranial aneurysms. However, in their study, penetrating trauma was responsible for the majority of their intracranial aneurysms. [15]

In this study, the pickup rate of traumatic cranio-cervical injuries was 12%. Of that cohort, penetrating trauma contributed to 69% of the injuries. Eighteen percent of the injuries were intracranial. Blunt trauma to the head accounted for 30% and to the neck

accounted for 1% of the injuries. The fact that the majority of the injuries were caused by penetrating trauma collaborates the previous study by Kieck and De Villiers also done at Groote Schuur Hospital. [15]

The neck injuries showed in the study were all in zones II and III because all the zone I and some of the zone II vascular injuries are treated by the trauma and vascular surgeons.

Presentation and pathology

Ninety two percent of the patients were male which reflects the trend in the literature. This is due to increased risk behavior among young males and the social violence including assaults and gang wars. [15]

Cervical injury patients mainly presented with acute bleeding or an expanding hematoma. One was stabbed in the sub-occipital region with blade retained in right lateral mass of C1. They all presented early and on angiography were found to have vessel lacerations with contrast extravasating into a tract, an arteriovenous fistula or a false aneurysm.

Patients with fistulas tended to present over a varying time period from immediately post injury to years after the injury. Acute fistulas were more likely to be associated with active bleeding whereas later presenting patients had the classical features of bruit, pulsatile vessels and hyperdynamic circulation if the fistula was large.

In this study, traumatic brain injury was the commonest associated injury with both the blunt and penetrating cranio-cervical vascular trauma. It was the associated injury in 22.7% of the blunt trauma and 19.2% of the penetrating trauma. All patients with vertebral artery injury (VAI) had associated cervical spine injuries. However, 51.9% of patients with penetrating vascular trauma had no associated injuries. 36.4% of the blunt cranio-cervical patients presented with isolated vascular injuries.

Investigations

Penetrating vascular injuries are easier to suspect and screen for. Blunt carotid and vertebral arterial injuries are uncommon but have the potential for devastating consequences. Unfortunately, there are no reliable means to suspect these injuries before neurologic symptoms or abnormalities show on computed tomographic scan. [6,16,17] The cerebral ischaemia rate is 40-80% and mortality rate is 25-60% without adequate diagnosis and proper treatment. [18] Screening patients based on injury mechanisms and patterns allows for diagnosis and treatment of injuries while they are still asymptomatic, potentially improving neurologic outcomes. [7,19,20,21]

Digital Subtracted Angiography (DSA) is the most sensitive imaging study but due to its invasiveness, its role as a screening tool is questionable. [6] It is still considered the gold standard when diagnosing traumatic vascular injuries. It offers the advantage of being able to follow up with treatment of the vascular injury soon after making the diagnosis.

All this can be done in the same setting. Also, the diagnosis and treatment can be done under local anasthaesia.

CT Angiography (CTA) of the cranio-cervical vessels is an important tool for rapid detection of vessel injuries in trauma patients. It can serve as a screening tool for vascular injuries in this region. Depending on the different types of associated fractures, a DSA may be indicated for focused attention to the vessels for injuries. That said, dissection of internal carotid and vertebral arteries can occur without fractures. [8] Múnera and colleagues in their 2000 study found the sensitivity and specificity of helical CT angiography to be high for detection of major carotid and vertebral arterial injuries resulting from penetrating trauma. Sensitivity of helical CT angiography was 90%, specificity was 100%, positive predictive value was 100%, and negative predictive value was 98%. [22] Gracias and colleagues in their study in 2001 that compared the accuracy of helical CT angiography with conventional angiography in patients with penetrating neck injuries, helical CT angiography was found to have sensitivity of 90%, specificity of 100%, positive predictive value of 100%, and the negative predictive value of 98%. [41] Utter and colleagues in 2006 found CT scan to have a high sensitivity as a screening tool for Blunt Vertebral and Carotid Artery injuries with a Negative Predictive Value of 92% and a Positive Predictive Value of 19%. Base of skull fractures and Fractures of the lateral elements of the cervical spine vertebrae were the most predictive indicators of vessel injury. [14] All studies were with later generation CT scans with more than 8 multi-slice CT scans.
CT angiography requires use of contrast and is poor in detection of vascular injuries in the periphery.

MRI has been used with some success in diagnosing traumatic cerebro-cervical injuries and it provides great anatomic detail of the vessels. [24] However, there are limitations in using it in an acute trauma setting. The patient will need to be transported to the MRI suite with MRI compatible equipment. Such patients require very close monitoring. If the patient has penetrating particles, they may move during the study or cause a lot of artifact. It is also an expensive procedure.

Kraus and colleagues in their 1999 study and Martin and colleagues in their 1991 study both showed Duplex Ultrasound scan to be useful in diagnosis of cranio-cervical vascular injuries due to blunt trauma. However, it was not recommended as a screening tool. [25,26] The study is operator dependent. The results in the trauma setting may be hampered by local pain, local subcutaneous emphysemas or hematomas and open wounds. It is unable to detect vascular injuries that do not cause disturbances in blood flow.

Management

The majority of penetrating neck injuries can be managed with selective non-operative management [27] because the risk of vascular injury is low. However if patients have a significant wound haematoma, particularly if it is pulsatile, a pulse deficit, a bruit or active bleeding requiring tamponade, then further vascular imaging is required. These clinical features also apply to facial and scalp injuries.

Various surgical techniques have been used in the treatment of cranio-cervical vascular injuries. Included among these are vessel ligation, primary resection and repair, vessel by-pass, trapping and clipping of the false aneurysms.

The technical difficulties encountered for direct repair of the internal carotid in zone III injuries can be substantial. Approaches to zone III injuries require extensive surgical exposure of the distal cervical ICA and adjacent structures in the infratemporal fossa. Distal control of the ICA is a potential problem if the injury extends to the cartilaginous ring at the entrance to the carotid canal. Mandibular osteotomy or zygomatic arch osteotomy can improve access but do not address back bleeding from the petrous carotid artery. [28]

EC-IC bypass avoids the problems of direct repair while preserving the cerebral circulation, but requires a craniotomy and exposure of either the petrous or supraclinoid ICA or the MCA as the distal recipient vessel for the bypass. [28,29,30]

Surgical series have reported mortality of between 0 and 15.7% and morbidity of 0 to 25%. [15,31,32] Given that there are difficulties in obtaining surgical exposure of these lesions and little limitation to endovascular access, it seems logical to manage these injuries in an angiography suite. Several endovascular series dealing with cranio-cervical vascular trauma have been reported, they have all shown good results with minimal morbidity and mortality. [4,5,13,23,33,34,35,36,37,38,40]

As far as possible patients should be resuscitated before transfer to an angiography suite. This would include adequate airway protection, tamponading the bleeding and blood replacement. Rapid hemostasis in bleeding patients with penetrating injuries can often be achieved with a Foley catheter passage down the injury tract, balloon inflation and skin suture around the catheter. [27] Where control of bleeding cannot be obtained as with blunt injury and multiple facial fractures time should not be wasted and patients should be taken directly for angiography as vessel closure may be the quickest way to stop the hemorrhage. [39]

Our angiography protocol is to start with a common carotid angiogram and vertebral angiogram on the side of the injury focusing on the area of injury and remembering that penetrating injuries may extend deep and across the midline. Injuries may be subtle and without obvious contrast extravasation even in actively bleeding patients or they may be easily identifiable as with large false aneurysms. If no injury is detected then ascending cervical and deep cervical injections are performed. Where cerebral circulation may be compromised contralateral vascular studies are performed to assess collateral circulation.

Once an injury is visualized, the damaged vessel is selectively catheterized and further angiography performed using gentle hand injections so as not to precipitate further bleeding. With vessel lacerations and false aneurysms our approach is to perform trapping of the injured segment or proximal vessel occlusion as close to the lesion as possible. Proximal occlusion is effective in smaller vessels of the external carotid circulation but less likely to work in the vertebral or internal carotid arteries where trapping is preferred. Because trauma patients are usually young, closure of the cerebral vessel is well tolerated. In this series, there was only a single patient with carotid injury and inadequate collateral circulation who required a covered stent reconstruction of the cervical internal carotid. None of the patients with a closed cerebral vessel has presented with a flow related aneurysm. We avoid stent reconstruction in these young trauma patients. This is because of the antiplatelet requirement post treatment in patients who may have other associated injuries and the unknown risks of stent fracture and vessel stenosis.

Fistula closure is often complex requiring multiple endovascular tools to obtain cure by trapping or embolization onto the venous side of the lesion. Fistulas seen in the first few days of injury may not have a secure wall and we favor trapping over direct fistula embolization.

More than 50% of our patients had an associated injury and it is particularly important to exclude tracheal, esophageal and mediastinal injuries in patients with penetrating and blunt neck injuries.

Despite these other injuries and many patients presenting in a shocked state, 86% of patients were independent at follow up. All lesions were successfully endovascularly treated with the only technical problems being balloon deflation in CCF treatment and a single patient having trigeminal pain after treatment for CCF.

CONCLUSION

Traumatic cranio-cervical injuries are associated with severe morbidity and mortality if left untreated. They need to be screened for most especially in the setting of blunt trauma where the clinical presentation is delayed. Though the injuries have traditionally been treated surgically, endovascular treatment offers a viable and safe treatment alternative.

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PART D: APPENDIX

DRC approval letter

UNIVERSITY OF CAPE TOWN

Department of Surgery

Departmental Research Committee
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1st November 2011

Dr Peter Kato Ssenyonga
Department of Surgery
Division of Neurosurgery
Groote Schuur Hospital
University of Cape Town

Dear Dr Ssenyonga

RE: PROJECT 2011/075

PROJECT TITLE: The Endovascular Treatment of Traumatic Cranio-Cervical Vascular Injuries

The above proposal was reviewed by the Department of Surgery Research Committee and I am pleased to inform you that the committee approved the study.

Please use the above project number in all future correspondence.

Yours sincerely

signature removed

PROFESSOR ANWAR S MALL
CHAIRMAN: RESEARCH COMMITTEE

Ethics Committee Approval letter

UNIVERSITY OF CAPE TOWN

Faculty of Health Sciences
Faculty of Health Sciences Research Ethics Committee
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22 November 2011

HREC REF: 543/2011

Dr P SSenyongo
c/o Prof A Taylor
Neurosurgery
H-53 OMB

Dear Dr Ssenyonga

PROJECT TITLE: THE ENDOVASCULAR TREATMENT OF TRAUMATIC CRANIO-CERVICAL VASCULAR INJURIES

Thank you for submitting your study to the Faculty of Health Sciences Human Research Ethics Committee for review.

It is a pleasure to inform you that the Ethics Committee has **formally approved** the above-mentioned study.

Approval is granted for one year till the 28 November 2012.

Please submit a progress form, using the standardised Annual Report Form (FHS016), if the study continues beyond the approval period. Please submit a Standard Closure form (FHS010)if the study is completed within the approval period.

The HREC are curious why patients <13 are being excluded in this retrospective review, we presume that it is due to the fact that GSH deals mainly with adult and adolescents.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please quote the REC. REF in all your correspondence.

Yours sincerely

signature removed

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS

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Federal Wide Assurance Number: FWA00001637.
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