

Accepted Manuscript

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PII: S1752-928X(14)00185-1

DOI: [10.1016/j.jflm.2014.09.012](https://doi.org/10.1016/j.jflm.2014.09.012)

Reference: YJFLM 1082

To appear in: *Journal of Forensic and Legal Medicine*

Received Date: 3 June 2014

Revised Date: 27 August 2014

Accepted Date: 17 September 2014

Please cite this article as: Wong B, Ong BB, Milne N, The source of haemorrhage in traumatic basal subarachnoid haemorrhage, *Journal of Forensic and Legal Medicine* (2014), doi: 10.1016/j.jflm.2014.09.012.

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The Source of Haemorrhage in Traumatic Basal Subarachnoid Haemorrhage

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Abstract

Traumatic basal subarachnoid haemorrhage (TBSH) following trauma to the head, face or neck is well-established as a cause of death, however it remains a heavily disputed topic due to the fact that the site of vascular injury is difficult to identify. This has been attributed to variable methods of examination. Whilst many regions within the vasculature of the head and neck have been proposed as more susceptible to rupture, the vertebral artery remains the focal point of many investigations. In this paper, we present cases with TBSH in our forensic centre at Forensic and Scientific Services in Brisbane, Queensland (QLD), Australia over an eight-year period. A retrospective case review was conducted of autopsy cases from 2003 – 2011. Thirteen cases of TBSH were found, one case excluded due to vasculopathy. All decedents were male, the majority of which were involved in an altercation receiving blows to the head, face, or neck and were unconscious at the scene. All victims were either under the influence of alcohol, drugs, or a combination thereof. External examination revealed injuries to the head, face, and neck in all cases. Various combinations of further examination techniques were used during the post-mortem examination including brain and/or cervical spine retention, CT imaging, and angiography. Vascular injury was identified in eight of the twelve cases, all of which occurred intracranially and with seven involving the vertebral artery. In our study, histological analysis was most reliable in identifying the rupture site and angiography failed to reveal a rupture site. The added benefit of histological analysis over angiography is the ability to identify the microscopic architecture of the tear and also to diagnose a vasculopathy that may have rendered the individual more susceptible to TBSH.

Keywords

Subarachnoid haemorrhage; Trauma; Vertebral Artery; post-mortem examination

The Source of Haemorrhage in Traumatic Basal Subarachnoid Haemorrhage

Introduction

Traumatic basal subarachnoid haemorrhage (TBSH) following trauma to the head, face or neck is well-established as a cause of death⁽¹⁻⁴⁾. However, it remains a heavily disputed topic due to the fact that the site of vascular injury is difficult to identify^(2, 4-9). The diverging views that have emerged within the literature can be described as those in favour of an intracranial rupture site such as the intracranial portion of the vertebral artery or vessels of the posterior cerebral circulation^(6, 8-10) and those in favour of an extracranial rupture site (mainly in the vertebral arteries of the proximal cervical spine)^(1-4, 11). It has even been postulated that vascular injury and haemorrhage are not responsible for causing death and death is caused by disruption of brain stem and/or upper cervical spinal cord⁽⁶⁾.

This debate is largely propagated by lack of supporting evidence of vascular injury. This has been attributed to variable methods of examination. The basal cerebral circulation is often subject to tearing during brain evisceration at autopsy and specialised dissection techniques are required^(9, 10). If injury to the head, face, neck, or underlying structures is not visible or suspected, then such techniques necessary for brain and cervical spine retention are not often employed⁽²⁾. Comprehensive examinations, including radiological, macro- and microscopic analyses of the vasculature are not commonly reported in earlier published reports, thus prompting concerns over the validity of reported rupture site^(5, 6). Additional concerns regarding complete examination of the posterior circulation including both intra- and extracranial regions have been raised leading to inferences that this may not be completed in full when an injury site is provisionally identified thereby precluding comprehensive assessment of the remaining vascular branches^(5, 6).

Whilst many regions within the vasculature of the head and neck have been proposed as more susceptible to rupture, the vertebral artery remains the focal point of many investigations. The sites of rupture include the region through the foramen transversarium, the region between the atlas and axis, between the atlas and the base of the skull, its emergence from the foramen magnum (intracranially) and the vertebrobasilar junction^(1, 2). These vulnerabilities have been related to a number of factors ranging from anatomical structure of the vessel wall to its anatomical course as it runs through the foramen transversarium and into the foramen magnum^(2, 3, 9, 11). Currently, the mechanism of rupture is thought to be due to multiple factors, but primarily due to direct impact or movement of the head upon the neck leading to stretching, shearing, or compressive forces on the vessel externally^(2-6, 9, 10). However, it has also been proposed that sudden impacts or abrupt movements may lead to a significant increase in intravascular pressure with internal forces further contributing to rupture^(4, 8, 9). Alcohol, a frequent association with TBSH, is thought to exaggerate these effects on the vessels possibly due to local vascular dilation. Intoxication may also contribute to injury behaviourally and by decreasing reaction time to blows^(4, 9, 10).

In this paper, we present cases with TBSH in our forensic centre at Forensic and Scientific Services in Brisbane, Queensland (QLD), Australia over an eight-year period. The aim is to assess if the sites of ruptured vessels were identified and if identified, by which methods (visual, angiography or histology). Three typical case reports will be elaborated further in detail.

Materials and Methods

The Forensic Pathology section of the Forensic Science and Services in Brisbane performs all Coroner's post-mortem examinations in South East Queensland with the exception of the cases from the Gold Coast. In average, about 1200 post-mortems are performed annually.

A retrospective case review was conducted of autopsy cases from 2003 – 2011. Using cause of death 'subarachnoid haemorrhage' as a search parameter, all autopsy cases were filtered. All cases of subarachnoid causes due to natural cases (e.g. ruptured saccular aneurysm) were excluded. This included a case of ruptured saccular aneurysm that was due to trauma. Due to its relative rarity, individual forensic pathologists within the department were also interviewed for any cases that may not have been retrieved through the database search. Retrieved autopsy reports were de-identified and reviewed for the following information:

- Age
- Sex
- Mechanism of injury
- Survival time
- Toxicology – alcohol and other drugs
- Relevant autopsy findings – external/internal injury and cause of death
- Site of haemorrhage
- Methods used for examining the neck and posterior circulation of the brain

For the purpose of this study, TBSH was defined as haemorrhage in the base of the brain within the subarachnoid space caused by the rupture of an artery, not affected by aneurysm, in the posterior circulation of the brain and/or extracranial vertebral artery as a result of trauma to the head, face, or neck.

Results

Thirteen cases of TBSH were found, twelve through the database search and one through interview. In one case, neuropathological examination revealed abnormalities in the cerebral vasculature which may have been responsible for the haemorrhage, thus this case was excluded. All victims were male, age range 22 – 48 years (mean 32 ± 8.8). All cases but one were involved in an altercation receiving blows to the head,

face, or neck. One sustained injuries in a motor vehicle crash. In all cases, the decedents were unconscious at the scene with 10 of them either dying at the scene or some hours later. Of the remaining two, one died one day later and the other, 28 days later. Three were declared dead at the scene. All victims were either under the influence of alcohol (7 cases, mean 170.6mg/ml, range 33 – 277mg/ml), drugs (2 cases, prescription and/or illicit) or a combination thereof (3 cases) (Table 1).

On external examination, all cases exhibited injuries to the head, face, and neck. Various combinations of examination techniques were used during the post-mortem examination. These included brain and/or cervical spine retention, CT imaging, and angiography (Table 1).

Vascular injury was identified in eight of the 12 cases. In all these eight cases, the site of vascular injury was intracranial: seven at the intracranial segment of the vertebral artery and one at the left posterior communicating artery. A rupture site was not identified in remaining four cases (Table 2).

Interestingly in four of these eight cases, the rupture site was only identified microscopically. In these cases, antemortem rupture was confirmed, as there was fibrin formation or reactive change at the rupture sites. Absence of reactive changes including presence of stretched muscle fibres is not considered as antemortem rupture as the possibility of artefactual tears cannot be discounted.

Table 1: Case Information

Case	Mechanism & Survival Time	Relevant Autopsy Findings	Method of Examination	Site of rupture	Toxicology
(1) 22 M ^a	<ul style="list-style-type: none"> Impact to the head and fell Unconscious at scene; brain dead 7hrs later 	<ul style="list-style-type: none"> Numerous injuries to the head and face including bruise behind the left ear Basal subarachnoid haemorrhage (BSAH) Frontal pole contusion 	<ul style="list-style-type: none"> Brain and Cervical spine retained Both X-ray and CT Angiography 	No site identified	Alcohol: 0.115%
(2) 22 M	<ul style="list-style-type: none"> Altercation Unconscious at scene, dead 1hr later 	<ul style="list-style-type: none"> Numerous injuries to the head, face, and neck including bruise behind the right ear BSAH 	<ul style="list-style-type: none"> Brain and Cervical spine retained X-ray Angiography 	No site identified	Alcohol: 0.079% MDMA Cannabis metabolite
(3) 23 M	<ul style="list-style-type: none"> Impact to the head and fell Unconscious at scene, brain dead 1hr later 	<ul style="list-style-type: none"> Numerous injuries to the head and face including bruise behind the left ear BSAH 	<ul style="list-style-type: none"> Brain retained X-Ray Angiography 	Intracranial vertebral artery identified microscopically	Alcohol: 0.203%
(4) 26 M	<ul style="list-style-type: none"> Altercation Unconscious, brain dead 1 day later 	<ul style="list-style-type: none"> Injury to the face and neck including bruise on left side of neck BSAH 	<ul style="list-style-type: none"> Brain and Cervical spine retained X-Ray Angiography 	Intracranial left vertebral artery identified microscopically	Alcohol: 0.277%
(5) 27 M	<ul style="list-style-type: none"> Impact to the head, and fell Unconscious at scene and died en route to hospital 	<ul style="list-style-type: none"> Main injury to the face (forehead) BSAH 	<ul style="list-style-type: none"> Brain and Cervical spine retained X-Ray Angiography 	Possible left posterior communicating artery rupture identified macroscopically	Alcohol: 0.295% Fluvoxamine
(6) 29 M	<ul style="list-style-type: none"> Impact to head and fell down Unconscious at scene, died while in hospital 28days later 	<ul style="list-style-type: none"> Injury to head and face BSAH 	<ul style="list-style-type: none"> Brain and Cervical spine retained X-Ray Angiography 	Intracranial vertebral artery identified microscopically (side not specified)	Alcohol: 0.150% (taken 6hrs after incident)
(7) 35 M	<ul style="list-style-type: none"> Altercation Unconscious at scene, brain dead 8hrs later 	<ul style="list-style-type: none"> Injury to head and face including bruise to the right lower occipital region Avulsion fracture of right transverse process of C1 BSAH 	<ul style="list-style-type: none"> Brain retained Full body CT scan 	Intracranial right vertebral artery identified macro- and microscopically	Alcohol: 0.158% Cannabis metabolites Methylamphetamine Low diazepam and metabolite
(8) 36 M	<ul style="list-style-type: none"> Found dead at scene 	<ul style="list-style-type: none"> Injury to head and neck including lacerations behind the left ear BSAH 	<ul style="list-style-type: none"> Brain and Cervical spine retained 	No site Identified	Methylamphetamine Low Morphine Oxycodone
(9) 36 M	<ul style="list-style-type: none"> Altercation Unconscious at scene, dead less than one day later 	<ul style="list-style-type: none"> Injury to the head and face including bruise behind left ear BSAH 	<ul style="list-style-type: none"> Brain retained 	Intracranial left vertebral artery identified macro- and microscopically	Low level of amphetamine derivatives Cannabis

(10) 36 M	<ul style="list-style-type: none"> Motor vehicle crash Dead at scene 	<ul style="list-style-type: none"> Injury to the head, face, and neck Superficial lacerations to frontal and temporal lobes. BSAH Subarachnoid haemorrhage involving cerebral hemispheres and brainstem 	<ul style="list-style-type: none"> Macroscopic examination of brain and cervical spine 	Intracranial right vertebral artery identified macroscopically	Alcohol: 0.117%
(11) 46 M	<ul style="list-style-type: none"> Altercation Unconscious at scene, dead on arrival at hospital 	<ul style="list-style-type: none"> No obvious external Subcutaneous dissection revealed injury to the head, face, and neck BSAH 	<ul style="list-style-type: none"> Brain and Cervical spine retained Full body CT scan 	Intracranial right vertebral artery identified microscopically	Alcohol: 0.206%
(12) 48 M	<ul style="list-style-type: none"> Altercation Dead 5 mins later 	<ul style="list-style-type: none"> Injury to the head, face, and neck BSAH 	<ul style="list-style-type: none"> Brain retained X-Ray Angiography 	No Site Identified	Alcohol: 0.223%

^a: (Case Number), Age, Sex; M= Male

Table 2: Site of Haemorrhage

	Total	Site	Total
Not Identified	4 (33.3%)	-----	-----
Identified	8 (66.7%)	Intracranial	8
		Extracranial	Nil

Case Reports:**Case 3**

A 23-year-old male was struck in the head and fell over. He was unconscious at the scene, resuscitated and brought to the hospital. Tests at the hospital showed subarachnoid haemorrhage. He was declared brain dead shortly afterwards. The post-mortem examination showed three sites of injury to the head: a bruise behind the left ear, an abrasion over the left forehead, and bruising and laceration to the lips. There were also minor injuries to the limbs. Neuropathology showed subarachnoid haemorrhage greatest at the base of the brain. The circle of Willis was intact. Acute rupture of one of the vertebral arteries was identified on microscopic examination but not on gross examination or with the use of vertebral artery x-ray angiography (Figure 1). The rest of the internal examination was unremarkable. His blood alcohol level was 0.203%.

Case 4

A 26-year-old male was involved in an altercation resulting in unconsciousness at the scene. A CT scan performed after admission to hospital showed extensive subarachnoid and intra-ventricular haemorrhage with obstructive hydrocephalus. He remained in ICU for one day after which he was declared brain dead by brainstem assessment and cerebral angiography. The post-mortem examination revealed a bruise and swelling to the lower buccal mucosa extending to the lip and a bruise within the left splenius capitis muscle at the level of the angle of the mandible. There were also minor injuries to the chest, the left knee, and the left dorsum of the foot. The basal subarachnoid haemorrhage was confirmed. Duret haemorrhages were present in the midbrain and pons. Vertebral artery x-ray angiography did not identify a site of haemorrhage but rupture of the intracranial portion of the left vertebral artery was identified microscopically. All other posterior intracranial vessels were intact. The rest of the internal examination was unremarkable. His blood alcohol level was 0.277%.

Case 10

A 36-year-old male driver was involved in a head-on collision with a prime mover on a road with a speed limit of 80 km/hr. He was declared dead at the scene. He wore a seatbelt but the vehicle was not equipped with airbags. On external examination, there were multiple injuries over his entire body; in particular he sustained large abrasions and lacerations to the head, neck, chest, and abdomen that could have been caused by the seatbelt. There were also multiple fractures to the head, face, ribs, pelvis, and limbs not attributable to the seatbelt. Internal examination showed multiple injuries to the brain (laceration to the frontal and temporal lobes, subarachnoid haemorrhage involving the cerebral hemispheres, base of the brain and brainstem and basal subarachnoid haemorrhage), lungs, sigmoid colon, liver, bladder, and testes. A 22mm longitudinal tear of the intracranial portion of the right vertebral artery extending to the vertebrobasilar junction was identified macroscopically and confirmed histologically (Figure 2). There was no evidence of underlying abnormality of the vessel wall that could have contributed to this tear. His blood alcohol level was 0.117%.

Discussion

It has been well documented that TBSH typically occurs in younger intoxicated males who have been involved in an altercation receiving blows to the head, neck, and or face^(3, 4, 9-11). Every case but one (motor vehicle crash) analysed in this study fits this profile.

Of the 12 cases in our study, 10 were intoxicated with alcohol while the other two were under the influence of drugs; interestingly both with amphetamine derivatives. All but one was involved in altercations. The influence of alcohol has been reviewed in the literature. Four main mechanisms have been proposed - the effects on blood vessels directly (decreased vasospasm and increased vasodilation), delayed reaction of voluntary muscles thereby leading to more vigorous movements of the head or neck, poor coordination and control of actions, and increased aggression leading to susceptibility to violent situations⁽⁹⁾.

All of our cases were unresponsive at the scene and a majority died within the day of incident. Only one of our cases survived more than one day. This rapid decline in consciousness and quick progression to death are well reported in the literature^(3-5, 9-11). However, it has been proposed that other mechanisms, such as disruption of the brainstem or upper cervical spine, may be causal where collapse and death follow rapidly after trauma and that TBSH may merely be a concomitant lesion⁽⁶⁾. Lindenberg and Freytag⁽¹²⁾ believed that hyperextension of the neck can result in tearing of the nerves of the brainstem (now known as diffuse traumatic axonal injury) and if this mechanism is suspected, lesions as such should be examined for. The theory would merit further analysis particularly with the availability of special immunohistochemical stains.

Approximately half of our cases exhibited numerous external injuries and the other half exhibited relatively few injuries isolated to specific regions of the head, neck, and face. This finding was not surprising as it has been shown previously, that vascular injury resulting in TBSH can result from relatively mild to moderate amounts of force^(2, 3, 9). Indeed, one may not have any external evidence of injury as the application of indirect force (as seen when avoiding a blow) has also been reported as a cause of TBSH⁽⁸⁾. As alluded to previously, methods of examination need to include specific head and neck dissection techniques to ensure the posterior circulation remains intact for gross inspection and histological analysis.

Vascular injury was identified in eight of the 12 cases reviewed. In all cases, the site of rupture was in the intracranial vessels with seven of the cases involving the vertebral artery. This is compatible with the overall consensus that intracranial rupture is the most common lesion. It is also proposed that in cases where an extracranial lesion is uncovered first, an extended search of the intracranial circulation may reveal additional rupture/s. This notion has been described by others^(5, 6) and relates directly to the method used in the examination of the head and neck. Some case reviews have revealed a preponderance of extracranial sites of injury, however these studies placed high levels of significance on upper cervical

vertebral malformations and fractures as a mechanism of rupture and thus may have overlooked intracranial sites^(1, 2, 11).

At present the aetiology of TBSH is somewhat unclear. Although TBSH often result from a blow to the head, neck, or face, the exact sequence of events leading to vascular disruption has not yet been revealed. A number of mechanisms have been proposed and they have been summarised below.

Early reports suggest that brain and meningeal movement caused directly by a blow or by avoiding a blow, can result in a shearing force causing disruption^(2, 3, 13-15). This effect may be exacerbated when the vessel is fixed to adjacent structures, thus decreasing its ability to deform and compensate for the additional forces that have been applied. Similarly, a fixed vessel may stretch excessively with applied force again increasing susceptibility to rupture^(3, 13, 16). Hyperextension, acceleration and deceleration movements have also been shown to cause disruption via both shearing force and excessive stretch^(14, 15, 17).

Compression of a vessel may lead to an increased intra-arterial pressure subsequently causing tears at vessel branches or of the vessel itself^(3, 13, 15). Some reports have discounted this effect stating that an increase by a factor of 10 is needed⁽⁴⁾. However, Farag et al⁽¹⁸⁾ in vitro experimentation found rupture possible with pressures of 150 – 250 mmHg. In this paper, they assert that the interplay of multiple factors such as increased intra-arterial pressure and abnormal movements is more likely. This leads to transient occlusion of vessels, which alters the internal haemodynamic properties and causes disruption from within the vessel. Circumferential stretch increases the forces applied to the lumen of a vessel⁽¹⁶⁾ thus dissection and complete rupture may be on a spectrum of injury. Indeed, in all the cases where the rupture was identified macroscopically, the tears were longitudinal indicating the mechanism being due to the vessel being circumferentially stretched as observed during an increase in intra-arterial pressure.

In the instance of an extracranial injury, dissection allows blood to track up the vessel wall into the intracranial space. Direct injury caused by a fracture of the upper cervical vertebrae may also cause vascular rupture, however this is not a requisite feature leading to TBSH⁽⁴⁾.

A number of studies have focused on structural alterations and tensile strength of the vertebral artery as a possible reason for variable outcomes following head and neck injuries. Essentially, this structural variation in wall composition is believed to increase the susceptibility of the vertebral artery to injury^(14, 19-21).

Congenital intrinsic abnormalities of the vessel wall have been mentioned briefly over the last twenty years as possible contributors to TBSH^(4, 11, 19), however up until recently this had not been explored any further. Pickup and Pollanen⁽¹⁹⁾ revisited the existence of fatal outcomes despite the benign nature of the injury sustained. They highlighted that following minor impacts to the head and neck, fatality seemed disproportionate to the amount of force applied. They described a potential link between TBSH and Ehlers-Danlos syndrome, more specifically the vascular syndrome caused by mutations of the COL3A1 gene, the gene encoding type 3 procollagen. They conclude by suggesting genetic testing should be considered in all cases of traumatic vertebral artery rupture as subclinical connective tissue disorders may explain the susceptibility of some individuals to fatal outcomes - their underlying genetic make-up increasing their vulnerability to physical injury.

At present, comprehensive examinations including specialised imaging and dissection techniques for TBSH is recommended but are not performed routinely^(22, 23). The examination methods employed in our cases were varied. This is not surprising; as of yet there are no accepted good practice guidelines for the examination of TBSH⁽⁹⁾. Most but not all of the cases underwent comprehensive examinations consisting of brain and cervical spine retention as well as radiographic imaging. A survey of the literature revealed many proposed protocols all including a combination of angiography, en-bloc neck dissection, and careful brain dissection and removal^(4, 6, 8, 9, 22).

Interestingly in four cases, no site of vascular injury was identified. The difficulty in identifying rupture sites has been explained in previous works to be due to three factors. The first being due to 'blind areas' at the sites of entry into the intracranial space and in regions where the vessels are encased in bone^(4, 24). Secondly, artefactual tears created during the dissection process may obscure true tears^(5, 7, 9) and thirdly the absence of a thorough examination of both the intracranial and extracranial vasculature. As Contostavlos⁽⁵⁾ stated "if the cupboard has not been searched, it cannot be said to be bare". Suspicion must remain high when mechanisms of injury are suggestive of TBSH as some cases may present with minimal external injury and without clear indication of vascular damage^(3, 16, 17, 24).

In our study, histological analysis was most reliable in identifying rupture sites and angiography failed to reveal rupture sites even in cases where injury was ultimately identified via histological analysis. The added benefit to histological analysis over angiography is the ability to identify the microscopic architecture of the tear and also to diagnose a vasculopathy that may have rendered the individual more susceptible to TBSH.

Acknowledgement

We would like to thank Forensic and Scientific Services, Queensland Health, Office of the State Coroner and Forensic and Scientific Services Human Ethics Committee for giving us permission to publish the paper.

We would also like to thank The Royal College of Pathologists of Australasia for awarding the RCPA Scholarships in Pathology to BW to allow her to complete the paper.

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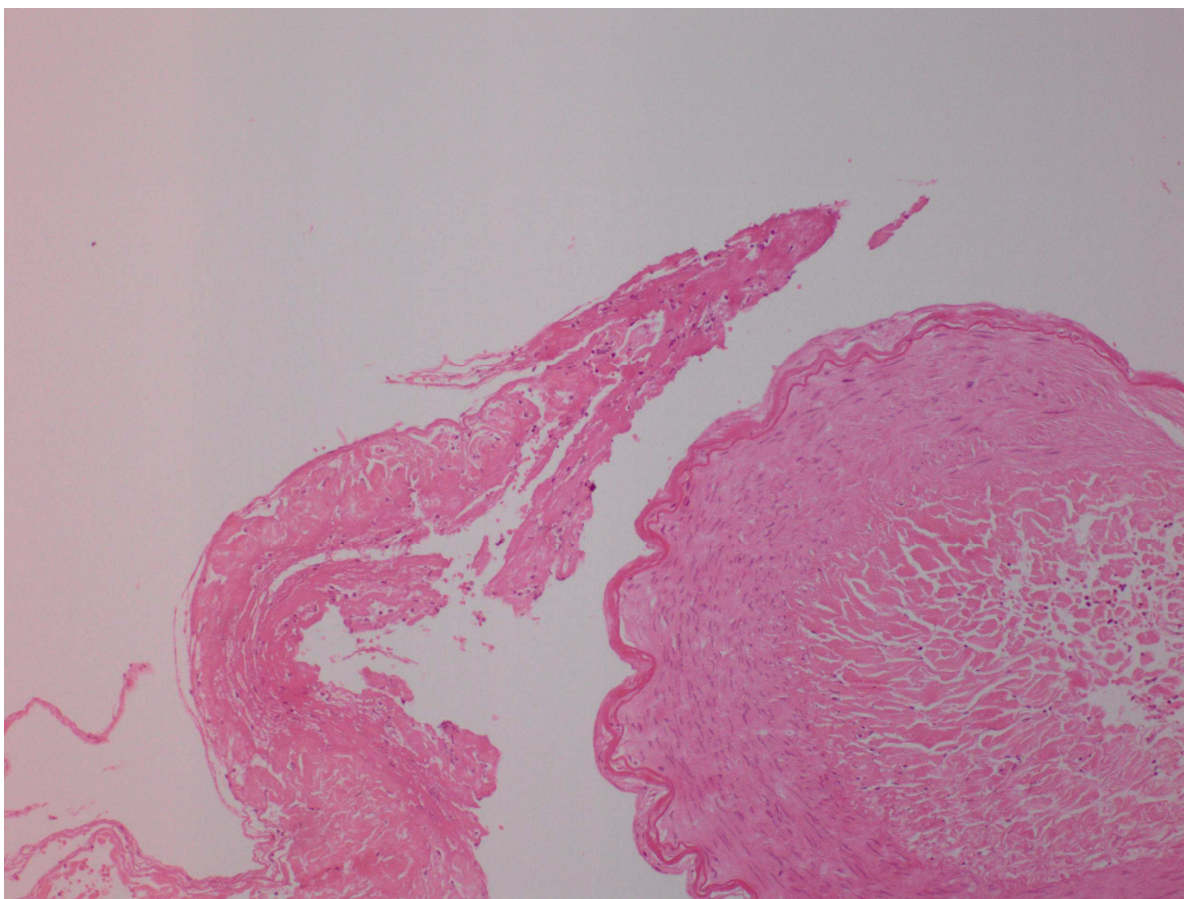


Figure 1: Photomicrograph of the rupture site of vertebral artery showing inflammatory cells and fibrin (case 3)

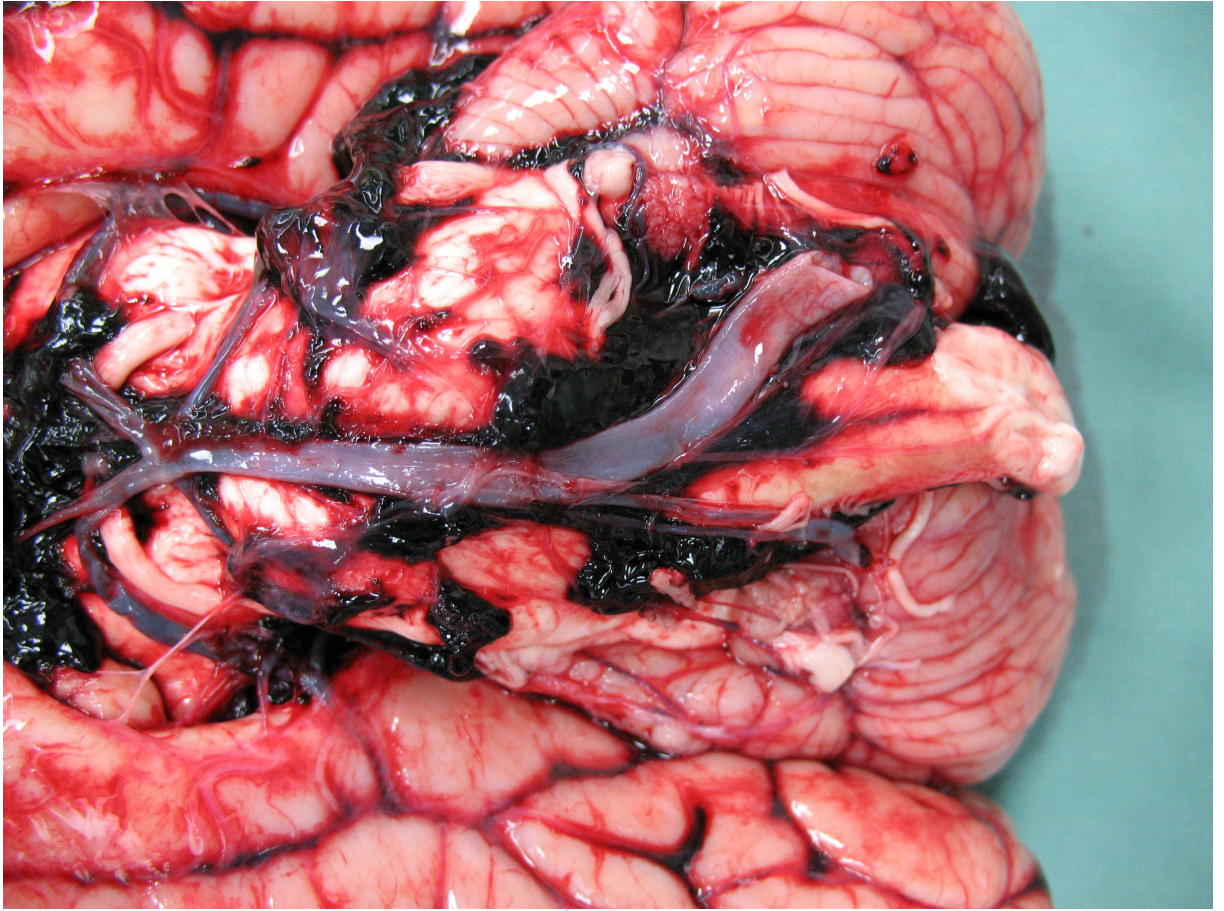


Figure 2: Rupture of the vertebral artery seen grossly (case 10)

Highlights

1. A study was performed to look for the site of vascular rupture in cases of traumatic subarachnoid haemorrhage excluding those due to berry aneurysms.
2. There were 12 cases in our department from years 2003 to 2011. Vascular injury was found in eight of these cases. All the sites of rupture were intracranial in location. In four of the locations, the rupture was visualised grossly with confirmation by histology in two of these cases. In another four cases, the location was only visualised in the histological sections of the artery.
3. It is recommended that in these cases all investigations including histology be performed to locate the site of the rupture.