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The source of haemorrhage in traumatic basal subarachnoid haemorrhage

Brittany Wong [a, b], Beng Beng Ong [a, c, *], Nathan Milne [a, c, d]

[a] Queensland Health Forensic and Scientific Services, 39 Kessels Rd Coopers Plains, QLD 4108, Australia
[b] RCPA Scholarship in Pathology Recipient, Australia
[c] The University of Queensland, School of Medicine, QLD 4006, Australia
[d] Bond University, Faculty of Health Sciences and Medicine, QLD 4229, Australia

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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 as the site of vascular injury is difficult to identify. 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. We present a retrospective case review of TBSH in our forensic centre at Forensic and Scientific Services in Brisbane, Australia, from 2003 to 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 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, with seven involving the vertebral artery. Histology was most reliable in identifying the rupture site and angiography failed to reveal a rupture site. The added benefits of histology over angiography are the ability to identify the microscopic architecture of the tear and to diagnose vasculopathy that may have rendered the individual more susceptible to TBSH.

1. 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

* Corresponding author. Queensland Health Forensic and Scientific Services, 39 Kessels Rd Coopers Plains, QLD 4108, Australia. Tel.: +61 7 32749200; fax: +61 7 32749201.
E-mail addresses: Brittany_Wong@health.qld.gov.au (B. Wong), Beng_Ong@health.qld.gov.au (B.B. Ong), Nathan_Milne@health.qld.gov.au (N. Milne).

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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.

2. 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 to 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.

3. 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 170.6 mg/ml, range 33–277 mg/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.

4. Case reports

4.1. 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 (Fig. 1). The rest of the internal examination was unremarkable. His blood alcohol level was 0.203%.

4.2. 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.