Subarachnoid haemorrhage due to upper cervical trauma

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SUMMARY Seventeen cases are reported in which fatal subarachnoid haemorrhage was associated with injury to the upper cervical region. Most of these cases were alcohol-intoxicated, most had sustained their injuries in an altercation, and death was usually but not invariably rapid.

It is proposed that trauma to the upper cervical region can cause subarachnoid haemorrhage, by a mechanism involving tracking of blood into the subarachnoid space from a damaged vertebral artery or one of its branches.

Although the condition of spontaneous subarachnoid haemorrhage is well known, little attention has previously been given in the literature to the distinct condition of massive subarachnoid haemorrhage due to trauma.

The forensic implication of this condition is obvious: for example it can be difficult to state with certainty whether subarachnoid haemorrhage found at necropsy was the result of trauma, or coincidental with it. Moreover, a clinical application is suggested by the fact that three of the cases survived long enough to be admitted to hospital for treatment.

Material and methods

A retrospective study was conducted on seventeen cases of death due to subarachnoid haemorrhage, following immediately on trauma, seen between 1972 and 1983. Post-mortem examination was carried out on these cases for legal purposes, and involved the staff of the Department of Forensic Medicine and Science at The University of Glasgow. Case histories were obtained from police reports.

In addition to routine post-mortem examination procedures, the upper cervical region was investigated in the following manner: the skin on the back of the neck was reflected from the level of the upper part of the scapulae to the occipital region. This skin flap revealed the muscles on the posterior and lateral aspects of the neck, and these were examined for bruising. The muscle layers were then reflected to reveal the deep muscles of the neck, such that

contusional injury could be followed through the cervical structures, and a line of force inferred. Dissection proceeded to reveal the upper cervical vertebrae and the extracranial portion of the vertebral arteries. The atlas vertebra was removed, fixed in formalin and x-rayed. The vertebral arteries were traced as far as the foramen magnum, and examined for surrounding haemorrhage. The vertebral and basilar arteries were then removed for histological examination. The site of maximal subarachnoid haemorrhage was noted and the arteries constituting the Circle of Willis were carefully examined for lesions including aneurysms.

Case reports

CASE 4 (died 5.4.75)

This 38-year-old man had been drinking since the early evening. At 2315 h he became involved in a fight outside the public house. He was punched and fell to the ground face down, when he was kicked twice on the head, and lost consciousness. He was moribund on arrival at hospital at 2325 h but responded to resuscitation and commenced regular spontaneous breathing at 0005 h. He remained stable until 0130 h when seizures developed and he was transferred to a neurosurgical unit. He showed frequent left-sided seizures. The left pupil was larger than the right, and did not react to light. Cheyne-Stokes respiration was present. An EMI scan was negative. Life was pronounced extinct at 1035 h. Post-mortem examination showed bruising on the left eyelid, abrasions and lacerations on the left upper lip, three dislodged teeth and a bruise behind

the left ear. There was bruising of the scalp behind the left ear, extending down 7 cm into the muscles behind the angle of the left lower jaw. There was haemorrhage into the adventitia of the left vertebral artery, where it crossed the posterior arch of the atlas, which was not fractured. Extensive subarachnoid haemorrhage was found at the base of the brain, extending over the vault bilaterally. The arteries at the base of the brain showed no lesions. Blood was present in the ventricular system. Blood and urine were negative for alcohol, but some eleven hours had elapsed since alcohol consumption ceased.

CASE 10 (died 14.8.78)

This 45-year-old woman had been drinking throughout the day and she returned home very drunk. At 2330 h her husband struck her once with his right hand, and she hit her head as she fell. The police arrived at 2340 h, noted a shallow pulse and heard the victim make gurgling sounds. She was pronounced dead at 2350 h. Post-mortem examination showed a bruise on the forehead, an abrasion on the left side of the chin and an area of reddening on the right side of the neck, measuring 2.5 cm × 0.5 cm. There was bruising on the occiput and left upper neck, measuring 2.5 cm × 2.0 cm. There was recent haemorrhage around the intact left transverse process of the atlas and around the left vertebral artery. There was a large subarachnoid haemorrhage at the base of the brain with thin staining over the rest of the brain. The vessels of the Circle of Willis were thin-walled and healthy, and search revealed no lesions. There was blood in the ventricular system. Alcohol was present in a concentration of 290 mg per 100 ml of blood, and 287 mg per 100 ml of urine.

Results

The 17 cases have been tabulated with regard to age, sex, trauma, estimated maximum survival time, relevant necropsy findings, and toxicology (Table 1). There was a male to female preponderance of 14:3. The mean age was 34 yr, with a range of 19–58 yr. The type of violence is shown in Table 2.

Seven of the cases were struck by hand. In case 15 the husband of the deceased was witnessed slapping her about the head several times and in case 16 the victim was punched twice. However, in the other five cases there was a clear account of a single blow being struck, ranging in severity from the punch of an ex-boxer to the blow of a drunken woman. Case 14 had injuries about the left ear suggestive of a blow from a fist, and a skin mark consistent with her husband's ring; she was found to have a tear of the

left vertebral artery close to the origin of the basilar artery.

In seven of the other cases death followed an assault by kicking. In case 6, bruising suggested that the victim was kicked in the frontal region, and no neck bruising was evident. Haemorrhage was seen around both vertebral arteries in conjunction with basal subarachnoid haemorrhage. Cases 3, 4, 9, 11 and 17 were punched to the ground, whereupon they were kicked several times about the head and neck. All five showed bruising to the upper neck and/or parotid region, and in one case this was in the form of a rectangular patterned bruise suggestive of a shoe sole. Four showed haemorrhage around the ipsilateral vertebral artery, but case 3 was found to have a 2 mm transverse tear of the basilar artery, just beyond the origin of the right superior cerebellar artery. Fig. 1 shows the surface injuries sustained in case 12 who was kicked once in the upper neck, was found to have an abrasion behind the left ear, and was found on histology to have haemorrhage around a moderately atheromatous left vertebral artery. This is the only one of our cases to show pre-existing pathology in the cerebral arteries on histology.

Two cases had been struck with blunt instruments in the parotid region (cases 1 and 8). A 1.22 length of wood of section 2.5 cm × 2.5 cm and poker had been used.

Finally, one of our cases, number 13, was knocked over a fence after being struck by a motor vehicle. This was the only one of our cases to have sustained a fracture to the transverse process of the atlas. A post-mortem radiograph (Fig. 2) demonstrated a bilateral congenital absence of the anterior arch of the foramen transversarium and a fracture of the posterior arch of the foramen on the right.

SURVIVAL TIME

All estimates of the time elapsing between the assault and death in the series are maxima. For example, in case 5 the victim was left overnight and was found to be dead the following morning, some nine hours later. In only one of our cases (No 8) has no attempt been made to estimate the survival time, as the body of the deceased was only found 7 days after her death. The results for the remaining 16 cases are given in Table 1 and summarised in Table 3.

The longest survivor was case 9, who lost consciousness six hours after the assault and was maintained on artificial ventilation for 39 h thereafter. However, the patient showed signs of brain death shortly after losing consciousness.

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Table 1 Trauma, survival time, necropsy findings and toxicological results in 17 cases of subarachnoid haemorrhage due to upper cervical trauma

Case	Age (yr)	Sex	Traumatic history	Survival time	Necropsy findings	Blood alcohol [urine alcohol] (mg/100 ml)
1	38	M	Four kicks to head. Blow to neck with length of wood	40 min	Abrasions and bruising around R ear. Haem around R TP of C1 and R VA Basal SAH	280
2	43	M	Punched on side of chin	40 min	Swelling angle of R jaw and neck. Haem around R TP of C1 and R VA Basal SAH	200 [255]
3	23	M	Kicked and punched on face and head	15 min	Bruise of helix L ear. Laceration to mouth. 2 mm tear in basilar artery at R superior cerebellar artery. Basal SAH	250 [315]
4	38	M	Punched to ground. Two kicks to head	11 h 20 min	Bruise behind L ear. Haem into and around L VA over C1 Basal SAH	Negative
5	36	M	Struck on face by hand	<9 h	Abrasions L ear. Bruising muscles of L posterolateral neck. Haem around L TP of C1 and L VA Basal SAH	238 [417]
6	23	M	Struck above left ear	40 min	Bruise L forehead. No neck bruising. Blood around both VA's Basal SAH	142 [194]
7	20	M	Single punch to right side of neck	12 min	Bruising R neck. Haem around R TP of C1 and R VA Basal SAH	Negative
8	50	F	Struck with poker	?	Bruising behind L ear. Haem in soft tissues of left side of neck up to foramen magnum Basal SAH	Negative
9	19	M	Punched to ground. Kicked on head.	45 h	Bruise behind L ear. Haem around L TP C1 and L VA. Basal SAH	Negative
10	45	F	Struck once by hand	20 min	Reddening R side of neck. Haem around L TP of C1 and L VA. Basal SAH	290 [287]
11	31	M	Punched to ground: kicked at least twice	10 min	Bruising L neck, and muscles down to spine. Haem around L TP of C1 and VA. Basal SAH	256 [337]
12	58	M	Kicked on neck once	30 min	Abrasion behind L ear. Haem around L TP of C1 and L VA. VAs + BA atheromatous Basal SAH	193 [294]
13	26	M	Struck by vehicle and knocked over fence	35 min	Bruise and swelling below R ear. Haem into muscle on R side of neck. Fracture R TP of C1 Basal SAH. Fracture R sphenoid bone	Negative
14	53	M	Struck once on face	30 min	Bruising & abrasion around L ear. Torn R VA at BA Basal SAH	214 [278]
15	29	F	Struck by hand	15 min	Three bruises L upper neck. Bruising in deep muscle over L TP of C1. Haem around L VA. Basal SAH	162 [197]
16	19	M	Punched twice on head	4 h	Bruising and abrasion of R ear. Haem around R TP of C1 and R VA. Basal SAH	76 [133]
17	31	M	Punched and kicked repeatedly	1 h 15 min	Multiple superficial injuries to face bruise anterior to R ear. Haem around R TP of C1 and R VA. Basal SAH	262 [300]

TP = transverse process
C1 = first cervical vertebra
VA = vertebral artery
BA = basilar artery
Haem = haemorrhage
SAH = subarachnoid haemorrhage

Table 2 Type of violence in 17 cases of subarachnoid haemorrhage due to upper cervical trauma

No of cases	Trauma
7	Kicked
7	Struck by hand
2	Struck by blunt instrument
1	Struck by motor vehicle

Table 3 Survival time in 16 cases of subarachnoid haemorrhage due to upper cervical trauma

Survival time	No of cases
0-30 min	7
30-60 min	4
60 min-12 h	4
12-48 h	1

TOXICOLOGY

All 17 cases were assayed for alcohol in blood, and 16 for alcohol in urine. These were negative in five cases, two of whom were known to have been drinking, but survived long enough to metabolise large amounts of alcohol. Three of our cases had not been drinking. For the remaining cases the mean values were: blood alcohol = 213 mg/100 ml (range = 76-290 mg/100 ml) and urine alcohol = 275 mg/100 ml (range = 133-417 mg/100 ml).

DISCUSSION

The condition described in this paper has generally been referred to as "traumatic subarachnoid haemorrhage". Although well established, we feel that this term is somewhat misleading, as it is com-

Fig. 1 Photograph of the left upper neck in case 12, showing the typical external injury seen in cases of subarachnoid haemorrhage due to upper cervical trauma.

Fig. 2 Post-mortem radiograph of the first cervical vertebra from case 13, demonstrating a congenital defect in each transverse process and a fracture in the right transverse process.

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mon knowledge that subarachnoid haemorrhage is frequently seen following head injuries such as are sustained in road traffic accidents. In these cases the haemorrhage is diffuse and thin, arising from superficial cortical vessels, and is often associated with cerebral contusions. There may also be associated subdural haemorrhage or cerebral lacerations.¹ It is clear that we are discussing quite a different condition, in which fatal massive basal subarachnoid haemorrhage results from a discrete injury and is found in isolation from other meningeal haematomata and cerebral injuries. We suggest that the term "traumatic subarachnoid haemorrhage" be restricted to this entity, as opposed to the non-specific condition.

DEGREE OF TRAUMA

From our results, it is clear that subarachnoid haemorrhage can result from a relatively minor degree of trauma. In the present series, only one showed a fracture of transverse process of the atlas (case 13). This victim was subjected to the severe trauma of collision with a motor vehicle, and was predisposed to fracture by a congenital defect in the transverse processes of his atlas. In the remaining cases fractures were not demonstrated, indicating that fracture of the transverse process of the first cervical vertebra is by no means an essential feature of the syndrome. The degree of trauma in 14 of our 17 cases was only moderate, in that it was the result of the impulse generated by an unarmed person in an assault. In our view, it is the accuracy rather than the sheer magnitude of the traumatic force that is critical. In short, subarachnoid haemorrhage may result when a relatively minor force is "on target" at the correct angle in the region of the transverse process of the atlas.

Simonsen² states that the trauma need not be particularly great, and he considers injuries at the level of the base of the skull particularly apt to produce this condition—such a site was found in 72% of his cases. All of the cases described by Contostavlos³ had been involved in physical encounters with an unarmed person, which were severe enough to fracture the tips of their transverse processes by direct injury; in addition, two of them had suffered mandibular fractures. Cameron and Mant⁴ are in accord that trauma need only be minor: of their four cases, two were punched in the neck, one was struck with the open hand, and one kicked. In Simonsen's two later cases blows were struck with the fist and, after defleshing the atlas, fissures were found in one transverse process.⁵

MECHANISM OF HAEMORRHAGE

It can be seen from our data (Table 3) that death from subarachnoid haemorrhage due to trauma is usually very rapid. 69% of our cases died within one hour of injury, and not one case survived longer than 48 h. The range of survival times was 10 min to 45 h. Death was almost immediate in most reported cases,^{3,4} with the longest survival time being 18 days.⁵

Nevertheless, the mechanism whereby trauma to the upper neck produces subarachnoid haemorrhage is unclear. There are several hypotheses that may explain this condition:

- (a) The blunt force incident on the neck may be transmitted to the vertebral artery where it is firmly fixed by the foramen transversarium of the atlas, resulting in a shearing force causing rupture of the vessel wall, either within or outside the skull.
- (b) Alternatively, the same effect may result from compression of the vertebral artery against the crescentic lower border of the posterior atlanto-occipital membrane, with tearing of the artery or one or more of its small branches.

When the tear is extracranial, there are two routes by which blood may then reach the subarachnoid space:

- (i) By dissecting upwards in and around the arterial wall to reach the subarachnoid space where the vertebral artery passes through the foramen magnum. In 15 of our 17 cases (88%) there was haemorrhage around the vertebral artery, which clearly extended upwards from the transverse process of the atlas to the foramen magnum. These necropsy findings tend to strongly support Contostavlos' view that blunt trauma to the vertebral artery causes disruption of the tunica, with consequent dissection into the subarachnoid space.³
- (ii) By tracking from the site of injury through the intervertebral foramina into the subarachnoid space of the upper cervical spine. The extracranial portion of the vertebral artery does in fact give off some small branches which pass through the intervertebral foramina to anastomose with the anterior spinal arteries in the spinal canal. In the longer surviving cases the haemorrhage may have occurred from one of these spinal branches rather than the main arterial trunk. Another possible explanation is that in these cases the site of haemorrhage is the vertebral venous plexus, which runs with the artery throughout the foramina transversaria of the upper six cervical vertebrae.
- (c) The trauma applied to the upper neck may

somehow cause rupture of the intracranial part of the vertebrobasilar arterial system. In two of our 17 cases (12%) there was no haemorrhage around the extracranial part of the vertebral artery, but the sites of rupture were positively identified within the subarachnoid space. These were a tear in the basilar artery in case 3, and a tear in the intracranial portion of the vertebral artery in case 14. Simonsen concentrated attention on the basilar artery. In 75 cases of traumatic subarachnoid haemorrhage he found 28% associated with pathological changes in the artery, compared with 73% in the comparison group of 341 cases of spontaneous subarachnoid haemorrhage.² Only one of our cases showed pre-existing arterial pathology, in the form of moderate atheroma, despite a careful search for lesions such as aneurysms by both gross examination and histology.

In summary, the evidence in our series favours the view that, in the majority of cases, haemorrhage originates from the extracranial portion of the vertebral artery. However, the actual site of rupture is difficult to demonstrate. It is likely that in a minority of cases the site of rupture is intracranial. The available evidence indicates that such haemorrhage can occur in previously healthy arteries.

TOXICOLOGY

In our series, the deceased was not intoxicated in only three cases. The remaining 14 were known to have been drinking, and the mean blood alcohol in those cases where this is applicable was 213 mg per 100 ml.

Simonsen² found that 87% of his 75 cases of traumatic subarachnoid haemorrhage were under the influence of alcohol, compared with only 9.7% of 145 cases of spontaneous haemorrhage where blood alcohol was measured. He considers alcohol to have a dual role: primarily the local dilating effect on the cerebral vessels, rendering them more likely to rupture, and also "as alcohol-intoxicated persons fall more heavily and have less appropriate avoiding reactions than have sober persons. This increases the possibility of head injuries during fights". All three of Contostavlos' cases were heavily intoxicated,³ but the author makes no comment on the role of alcohol. Cameron and Mant⁴ found high blood alcohol concentrations in two of their four cases, and stated that alcohol is an important contributory factor. They do not comment on the fact that two of their cases were not drinking at the time of their haemorrhages.

While recognising the role of alcohol at an anecdotal level, we regard this as the most controversial

area in the syndrome. Firstly, Simonsen² omits to point out that alcohol is significantly associated with homicide in general. Persons intoxicated with alcohol are far more readily involved in fights, and it may well be that the "less appropriate avoiding reactions" are relevant not only to the fact that drunken persons do not avoid the fight in the first place. We have seen cases in the literature and in our own experience where subarachnoid haemorrhage as a result of upper cervical trauma occurred entirely in the absence of alcohol. This is not to say that alcohol does not predispose, only to suggest that the predisposition is largely due to its effect in evoking aggressive tendencies in human behaviour. Nevertheless, it may well be that alcohol does have a pharmacological role in the production of the subarachnoid haemorrhage. Hillborne and Kaste⁶ carried out a study on 75 cases of aneurysmal subarachnoid haemorrhage in patients aged 15–55 yr, and found that the bleeding was preceded within 24 h by a bout of alcohol drinking in 25% of cases. They concluded that occasional alcohol intoxication carries an increased risk of aneurysmal subarachnoid haemorrhage. Unfortunately for our purpose, patients who died within the first 24 h without regaining consciousness were excluded from that study. A possible mechanism is suggested by the work of Barry and Scott,⁷ who found that vasospasm in the cat basilar artery, produced by administration of the animal's fresh blood to the artery, was of markedly reduced intensity and duration after intravenous infusion of 5% ethanol. These effects were unrelated to changes in blood pressure. This suggests that the *rapidity of death* in our cases may have been contributed to by alcoholic inhibition of the spasm after damage to the artery.

Conclusion

Fatal subarachnoid haemorrhage should be suspected to be traumatic in aetiology when injuries are seen on the ear, in the parotid region or on the upper neck. However, we have seen cases in which the external signs of injury are atypical; in such cases the true cause of the subarachnoid haemorrhage will only become apparent if the pathologist has a high index of suspicion and dissects the upper extracranial portion of the vertebral arteries. Such dissection usually reveals bruising in the deep muscles of the neck, with haemorrhage around the vertebral artery and the transverse process of the first cervical vertebra. In our experience, fracture of the process is an unusual finding, indicating a particularly severe degree of trauma. Alcoholic intoxication is a frequent, but not an essential part of the syndrome. The mechanism of haemorrhage is still obscure, and

in our opinion there may well be more than one mechanism involved. It is obviously important to establish the exact source of haemorrhage in future cases, and vertebral artery angiography probably holds the greatest promise for research.

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