Int J Leg Med (1993) 105:243–245

International Journal of
Legal Medicine

Cerebral intraventricular haemorrhage in a young adult

R. C. Chapman and M. L. Rossi

Department of Forensic Medicine & Toxicology, Charing Cross & Westminster Medical School, University of London, 116, Fulham Palace Road, London W6 8RF, UK

Received August 18, 1992

Summary. A case of a 26-year-old man who suffered a fatal intraventricular cerebral haemorrhage following an episode of trauma is described. The initial appearance at necropsy suggested a traumatic subarachnoid haemorrhage and initial investigation was directed towards the anterior neck structures and the vertebral arteries with negative results. Dissection of the fixed brain showed a massive intraventricular bleed with secondary involvement of the subarachnoid space and dissection into the cerebral parenchyma. No bleeding points or natural disease of the cerebral vessels could be identified. The practical aspects of diagnosis and the cautious approach necessary in interpreting subarachnoid bleeding is emphasised. The significance of intraventricular haemorrhage following trauma has become more apparent with the advent of computed tomographic scanning. The implications for this and similar cases are considered.

Key words: Intraventricular haemorrhage – Head injury – Traumatic subarachnoid haemorrhage

Zusammenfassung. Der Fall eines 26 Jahre alten Mannes wird beschrieben, welcher nach einer Episode von Traumen eine tödliche intraventrikuläre Hirnblutung erlitt. Bei der Obduktion wurde nach dem ersten Eindruck eine traumatische Subarachnoidalblutung vermutet, und die anfängliche Untersuchung war daher auf die Strukturen des Halses und auf die Vertebralarterien gerichtet, jedoch mit negativen Resultaten. Die Sektion des fixierten Gehirns zeigte eine massive intraventrikuläre Blutung mit sekundärer Beteiligung des Subarachnoidalraums und Ausbreitung in das Hirnparenchym. Keine Blutungsquellen oder natürliche Erkrankungen der Hirngefäße konnten gefunden werden. Die praktischen Aspekte der Diagnose und der vorsichtige Ansatz, welcher notwendig ist, um Subarachnoidalblutungen zu interpretieren, werden betont. Die Bedeutung der intraventrikulären Blutung nach Trauma ist mit der Einführung der Computertomographie offensichtlicher geworden. Die Implikationen dieses und ähnlicher Fälle werden bedacht.

Correspondence to: R. C. Chapman

Schlüsselwörter: Intraventrikuläre Blutung – Kopftrauma – Traumatische Subarachnoidalblutung

Introduction

Adult intraventricular haemorrhage may result from a primary intraventricular cause such as an arteriovenous malformation within the choroid plexus or, more commonly, from rupture of a haematoma of the parenchyma into the ventricles. The parenchymal haematoma may result from a hypertensive bleed or from a ruptured arterial aneurysm excavating the cerebral parenchyma. The relationship with trauma is uncertain in many cases. Isolated intraventricular haemorrhage in which there is no parenchymal haematoma appears rare. We present such a case.

Case presentation

A 26-year-old man had been drinking alcohol at home with a group of friends on the evening prior to his death. At about 2100 hours he fell heavily down a flight of stairs but was apparently unharmed. At about midnight, following a verbal argument he was punched in the face by one member of the party. This subdued him but whether he was rendered unconscious is unclear. He was left, apparently asleep, on a sofa when the rest of the party departed 1 h later. He was found dead the next morning. A necropsy was carried out later the same afternoon.

Necropsy findings

The body was that of a fully dressed and well built young male. The body temperature was 20°C. Rigor mortis was established in all muscle groups.

There was a 2.5×1.5 cm bruise above the outer canthus of the left eye, bruising of the left side of the upper lip and right lower lip. There was also a bruise on the left side of the mandible measuring 1×0.8 cm. There was abrasion and bruising of both elbows.

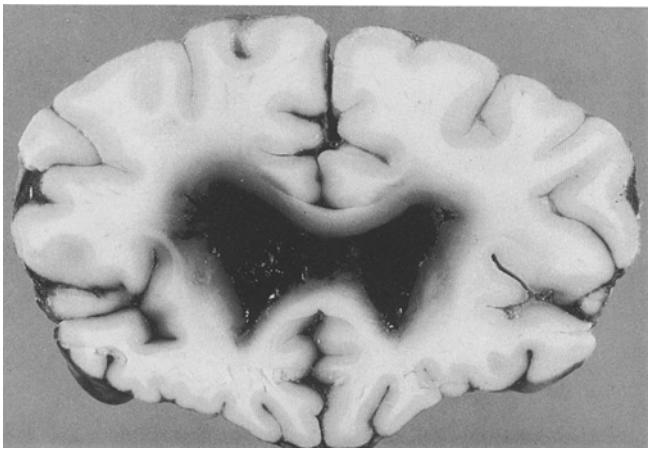

Fig. 1. Coronal section of brain in region of the head of the caudate showing blood in the lateral ventricles and infiltrating the periventricular parenchyma

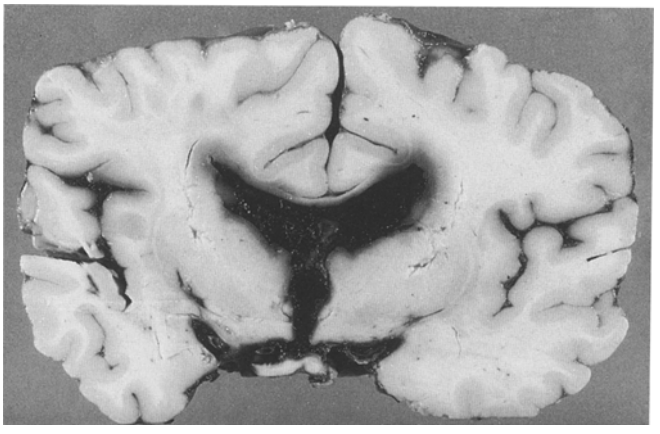

Fig. 2. Coronal section of brain showing blood cast within lateral and third ventricles and within subarachnoid space at base and over cortices

The internal examination demonstrated fresh bruising of the strap muscles beneath the left side of the chin and bruising at the angle of the jaw on the left side within the sternomastoid muscle. The skull was intact. There was extensive subarachnoid haemorrhage, particularly basally and around the upper cervical cord. The basal vessels were scrutinised for the presence of aneurysms or other abnormalities with negative results. The brain was placed in fixative for later dissection.

The initial impression was of a traumatic subarachnoid haemorrhage associated with trauma to the upper neck and cervical spine. Further investigations excluded this diagnosis. Plain x-rays of the cervical spine did not show a fracture at any level. Angiography of the vertebral arteries revealed no abnormality. Finally the entire cervical spine was excised with a rim of foramen magnum and the entire length of each vertebral artery explored by direct dissection. No deep bruising was demonstrable adjacent to the vessels, no vascular injury was present and there was no fracture of the cervical spine. Subsequent dissection of the fixed brain by serial slicing revealed a large intraventricular haemorrhage with a blood cast of the lateral and third ventricles. There was rupture of the ependymal layer and penetration of blood into the parenchyma surrounding the lateral ventricles at several points (Fig. 1 and 2). Histological sections stained with haematoxylin and eosin and LBCV stains revealed infarction of the periventricular white matter. Examination of the vessels revealed no evidence of hypertension or of any vascular pathology. The bleeding point could not be identified. The choroid plexus was examined histologically but revealed no abnormality or bleeding point. The remainder of the necropsy revealed no significant findings of natural disease or injury. There was no evidence of systemic hypertension.

Toxicological examination (Metropolitan Police Laboratory, London, U.K.) demonstrated a blood alcohol of 333 mg/100 ml. Screening for other drugs, including amphetamine, was negative.

Discussion

The neuropathological findings were interpreted as showing an intraventricular bleed with blood under considerable pressure. This had hampered the blood supply to the periventricular white matter resulting in infarction. There was continuity between intraventricular, intraparenchymal and subependymal blood, the blood dissecting from the ventricles into the surrounding white matter. The appearances suggested an arterial rather than a venous bleed.

Intraventricular haemorrhage in the adult may result from a primary intraventricular cause such as an arteriovenous malformation within the choroid plexus or, more commonly, from rupture of a haematoma of the parenchyma into the ventricles. Such a haematoma may itself result from a hypertensive bleed or from a ruptured berry aneurysm excavating the cerebral parenchyma. A study published in 1977 described a series of 54 patients with intraventricular haemorrhage diagnosed by computerised tomography (CT) [1]. The study confirmed the accuracy of the radiological diagnosis of the condition and showed that the majority of cases in the series resulted from natural diseases of which the most important were hypertension, arterial aneurysm and arteriovenous malformation. In only one of their cases was the origin within the choroid plexus (an arteriovenous malformation). The rest arose from the usual sites of hypertensive bleeding within the basal ganglia or the thalamus or from berry aneurysms of the cerebral vessels. In only one case was the bleeding regarded as post-traumatic. In this case a 40-year-old man suffered severe head injuries in a road traffic accident. The exact side of bleeding could not be determined. The authors commented that intraventricular haemorrhage was an uncommon finding in their cases of head injury, this case being the only one in a total of 196.

Later work involving C.T. scanning has shown that post-traumatic intraventricular haemorrhage is not as uncommon as the above reference would seem to indi-

cate. A series of papers in the late 1970's and 1980's suggested that intraventricular haemorrhage occurs in 2–3% of cases of head injury in which C.T. scans are deemed necessary [2–5]. The prognosis of these cases was grave and the explanation simple; in most cases intraventricular haemorrhage resulted from rupture of a large parenchymal bleed into the ventricles.

However, a paper in 1988 [6] described 10 surviving patients in which intraventricular haemorrhage occurred after blunt head injury. The scans showed intraventricular haemorrhage as the sole abnormality in 6 of these cases and there was no intraparenchymal bleeding. All cases except one resulted from road traffic accidents. The other resulted from a fall from a high chair in a 20-month-old baby.

Isolated intraventricular haemorrhage has been described following injection of approximately 1000 mg of amphetamine in a 31-year-old male [7]. The authors regarded this as the result of acute hypertension. Cerebral vasculitis was not present. Three cases of isolated intraventricular haemorrhage have been described following rupture of aneurysms of the distal posterior inferior cerebellar artery into the lateral recess of the fourth ventricle [8]. In these cases there was no obvious parenchymal bleeding nor any blood in the basal cistern.

A single case of post-traumatic rupture of a choroid plexus angioma producing massive intraventricular haemorrhage has been described [9]. In this case, following a road traffic accident, there were severe multiple injuries but only a small scalp bruise to indicate direct head injury.

Against this background, the finding of a massive isolated intraventricular haemorrhage in a young man following a fight presented a number of problems. The first arose from the deep seated nature of the bleeding and the consequent delay in necropsy diagnosis. The initial inpression was of a traumatic sub-arachnoid bleed and this diagnosis was rigorously excluded. Following adequate brain fixation it became clear that the bleeding was within the ventricles. The second problem was to decide on the singificance of the bleed and its relation to the history of violence. The temporal association would appear strong. The condition of the body and the body temperature allowed a tentative estimate of time of death as occurring some 8–12 hours before the necropsy was carried out. This suggested a minimal survival period of 3 h following the blow to the face at 12 midnight the previous day. Many of the patients described by clinicians following severe head trauma involving intraventricular haemorrhage survived a number of hours following the episode of trauma. It is clear that trauma is a not infrequent cause of intraventricular haemorrhage. In addition there was no natural disease to explain the haemorrhage. However, to our knowledge, there are no reports of isolated intraventricular haemorrhage caused by blows to the head or face. There was no other evidence of trauma to the brain. The mechanism of traumatic intraventricular haemorrhage is far from clear. However, 2 cases are described of patients developing delayed intraventricular haemorrhage approximately 10 h following trauma [10]. The authors suggest that these may have resulted from distortion of the ventricular walls with damage to the subependymal veins. Other workers have attempted to draw analogies with the intraventricular haemorrhages that occur in neonates, suggesting that a combination of hypoxia and venous congestion post head injury exacerbates traumatic capillary damage and leads to haemorrhage [6].

In the case described, however, it was clear that the appearances favoured an arterial bleed and that such mechanisms are unlikely to explain the findings.

Finally, there was the complicating factor of alcohol intoxication to consider. Alcohol intoxication is associated epidemiologically with both cerebral infarction and spontaneous subarachnoid haemorrhage. It is clearly associated with traumatic subarachnoid haemorrhage. It is known to have complex effects on cerebral blood flow and autoregulation, it interferes with haemostasis and has effects on plasma osmolality, red cell aggregation and deformability. It may produce arteriolar spasm [11]. Such complex effects may be involved in the production of isolated traumatic intraventricular haemorrhage.

References

1. Little JR, Blomquist GA, Ethier RE (1977) Intraventricular haemorrhage in adults. Surg Neurol 8:143–149
2. Olif M, Fried AM, Young AB (1978) Intraventricular haemorrhage in blunt head trauma. J Comput Assist Tomogr 2:625–629
3. Merino-de Villasante J, Taveras JM (1976) Computerised tomography in acute head trauma. Am J Roentgenol 26:765–778
4. Zuccharello M (1981) Post-traumatic intraventricular haemorrhages. Acta Neurochir (Wien) 55:283–293
5. Cordobes F (1983) Intraventricular haemorrhage in severe head injury. J Neurosurg 58:217–222
6. Christie M, Marks P, Liddington M (1988) Post-traumatic intraventricular haemorrhage: a reappraisal. Br J Neurosurg 2:343–350
7. Imanse J, Vanneste J (1990) Intraventricular haemorrhage following amphetamine abuse. Neurology 40:1318–1319
8. Yeh H, Tomsick TA, Tew JM (1985) Intraventricular haemorrhage due to aneurysms of the distal posterior inferior cerebellar artery. J Neurosurg 62:772–775
9. Gross A, Prochnicka B (1989) Intraventricular hemorrhage originating from choroid plexus angioma in a road accident victim. Z Rechtsmed 102:409–413
10. Piek J, Bock WJ (1986) Secondary intraventricular haemorrhage in blunt head trauma. Acta Neurochir (Wien) 83:105–107
11. Editorial (1983) "Binge" drinking and stroke. Lancet 2:660–661