Epidural Haematoma Following a Shunt Revision

By

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Summary

A case of epidural haematoma developing after shunt revision is presented. Change from the prone to the standing position was thought to be the causative factor.

Occurrence of epidural haematoma after ventriculo-atrial or ventriculo-peritoneal shunt operations, has only seldom been described. Due to its rarity, potential dangers, and mortality rate, we feel justified in presenting a case of epidural haematoma after shunt revision.

Case History

Our patient was born as twin A. Her twin brother, B, died at the age of three months from severe hydrocephalus. Because of moderate communicating hydrocephalus our patient was operated on at the age of four months when a right medium Holter ventriculo-atrial shunt was inserted. She developed normally, and the shunt was not revised in the next few years. Because of intermittent headache and double vision she was admitted to our department when 12 years old. Physically she appeared normal except for a slightly hydrocephalic cranial vault, with a head circumference of 56.5 cm. Ophthalmoscopy revealed no signs of increased intracranial pressure. The Holter valve did not appear to be functioning. Under general anaesthesia positive contrast ventriculography through a right frontal burr hole revealed aqueductstenosis with uniform dilatation of the lateral ventricles, and a brain mantle of 2 cm. Intracranial pressure during controlled N₂O/Halothane®/O₂-anaesthesia was 7–11 mm Hg. The Holter valve was replaced with a medium pressure Hakim valve connected to a Raimondi low pressure peritoneal catheter.

She was kept in bed for the next 24 hours during which time progress was uneventful. She was then allowed to stand after which over the course of the next few hours she became somnolent and developed left hemiparesis. She was treated with dexamethasone but, as the hemiparesis progressed, right carotid angiography was carried out revealing a huge frontal extracerebral haematoma. An epidural clot with a volume of 150 ml was removed through a frontotemporal craniotomy. The dura was adherent to the frontal burr hole used for ventriculo-
graphy and no source was found for the bleeding. The shunt system was occluded by a temporary clip for 17 days during which time the patient's condition was good. The clip was then removed and after a few days in bed she was again allowed to stand up. She was discharged in good condition with a functioning valve. Seen in our outpatient clinic one year later she was doing well and had no headache or double vision. The shunt was still functioning.

Discussion

Reviewing the literature we have found 31 cases of epidural haematomas occurring as complications following intraventricular pressure releasing operations. Most of these patients had greatly increased intracranial pressure and severe hydrocephalus. All the operations were followed by sudden drops in intracranial pressure. The complication appeared in only one patient after a ventriculo-atrial shunt. In this case the shunt operation was preceded by negative contrast ventriculography. Most patients developed symptoms during or immediately after operation. In one case the epidural haematoma was found by chance 15 months postoperatively. In two cases leukaemia and a low prothrombin index were thought to be major reasons for the bleeding. In another two cases bleeding dural vessels was seen during reoperation. Eighteen patients (58%) died as a consequence of their epidural haematomas, and only seven (23%) recovered completely.

Previous authors seem to agree that it may be the sudden lowering of the intracranial pressure that causes the bleeding. The intracranial pressure measured at the foramen of Monro is about — 5 mm Hg in the erect position. Following insertion of a differential pressure valve (Holter, Hakim, Pudenz) the intracranial pressure may be recorded as low as — 15 to — 30 mm Hg in the erect position. This is due to a siphoning effect in the distal tubing of the shunt system. The high frequency (21%) of subdural haematomas following shunting in patients with normal pressure hydrocephalus is probably also to some degree related to the extremely low intraventricular pressures. The reason why our patient developed an epidural haematoma is unknown. We think the fact that she developed her symptoms immediately after changing position from supine to erect more than 24 hours after the operation supports the theory that the sudden development of a negative intracranial pressure is dangerous. To neutralize these severe negative intracranial pressures in the standing position Portnoy produced a so called antisiphon device, and Hakim is working with a subdural pressure transducer which, when negative pressures occur, will close the differential valve. However, until these technically difficult problems have been