EXTRADURAL HAEMATOMA AT THE VERTEX*

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The wider use of angiography in the investigation of cases of head injury in Bristol has revealed several cases of extradural haematoma at the vertex associated with raised intracranial pressure. It is submitted that a median extradural effusion at or near the vertex compresses the sagittal sinus, and, probably more importantly, the venous lacunae, resulting in a degree of engorgement of the hemispheres.

Case Reports

Case 1.—A man, aged 36 years, was found unconscious an uncertain time after a motor-cycle skid. He recovered during the next hour sufficiently to be able to give some response to questions. The left pupil did not react to light. All limbs moved well. Later the right pupil dilated and also failed to react to light. In a few hours the pulse rate dropped from 80 on admission to 66/min. Paraldehyde was given. The patient was unconscious and incontinent by 12 hours after injury, and the plantar responses became extensor in type.

When he came under neurosurgical care an hour later a flaccid left hemiplegia was evident. The limbs on the right side showed a tendency to decerebrate posturing in response to stimuli. The most striking finding was a quite exceptional engorgement of retinal veins bilaterally, with early papilloedema. There were no retinal haemorrhages in spite of the gross turgidity of the veins. The pulse was at times below 60/min. The right temporal region exhibited a haematoma under the scalp in relation to extensive fissuring of the calvarium. One fissure extended in a coronal plane across the vertex.

Angiography (Fig. 1) revealed a depression of the sagittal sinus away from the calvarium in the frontoparietal region, and some narrowing of its lumen. It is significant that to obtain this picture the radiologist (Dr. J. L. G. Thomson) had to make a further injection and an exposure fully two seconds later than customary for this phase. The anterior cerebral artery was but slightly deviated to the left of the midline in the antero-posterior view.

At operation a quadrangular plate of bone, 6 × 8 cm. in size, centred with a bias to the right side, was removed at the vertex, its posterior limit being the transverse

FIG. 1.—Case 1: late-exposure venogram.

fissure fracture. This exposed the whole of a localized extradural clot of 2 cm. maximal thickness. When the clot was removed the dura mater was seen to be intact and under nearly normal tension. The plate of bone was replaced.

Next morning, about 12 hours after operation, the hemiplegia had vanished completely. The retinal engorgement had subsided. The patient was able in a limited way to obey commands. Tracheostomy became necessary on the second post-operative day. For the first 12 days both pupils remained fixed and dilated, yet he was able to comprehend and soon to speak. The eyes were divergent, and the plantar responses remained extensor. On the twelfth day the left pupil began to react feebly to light, and simultaneously the plantar responses became normal. On discharge home after five weeks in hospital the right pupil was still inactive and the eye remained deviated outwards. The fundi were normal, and in all other respects the patient was very well. The third nerve palsy has since recovered.

The clot in this case was not of large volume but it was strategically situated so as to compromise venous drainage from the hemispheres superiorly, and give rise to a diffuse congestion of the hemispheres, just the conditions in which a complete or 'ring' herniation would occur at the tentorial aperture, with dorsal compression of the midbrain. The early return of consciousness and in contrast

*Based on a communication given to the Society of British Neurological Surgeons in Copenhagen on May 12, 1961.

FIG. 2.—A 'ring herniation' and grooving of each third nerve by the petro-clinoid ligament. (By courtesy of Messrs. Butterworth and the author.)

the long persistence of third nerve dysfunction indicate that the midbrain escaped structural damage, and that the third nerves were compressed against the petro-clinoid ligaments. Johnson (1957) figures well the morbid anatomy envisaged in this case (Fig. 2).

Case 2 (by courtesy of Mr. D. G. Phillips).—A man, aged 23 years, who was a known diabetic fell from his horse while riding alone and was rendered unconscious. He recovered after an uncertain interval and walked some distance home. Frontal headache and vomiting persisted for five days, then he became drowsy and was admitted to hospital. The only important neurological findings were papilloedema, and rigidity of neck muscles, with a positive Kernig sign. The clear and colourless lumbar fluid was under a pressure of 300 mm., containing 264 red cells/c.mm. The blood sugar was 214 mg. per 100 ml.

Next day headache and drowsiness increased, and the left plantar response became extensor in type. The patient

FIG. 3.—Case 2: lateral angiogram.

was transferred to neurosurgical care. No new findings were elicited. Pulse and respiration rates were on the slow side. The pupils were normal. The papilloedema and the asymmetry of plantar reflexes were confirmed. The bladder was distended. The blood sugar had risen to 502 mg. per 100 ml.

Angiography showed an extracerebral effusion of some size, level with the parietal region (Fig. 3), and it is to be noted that the film shows evidence of a retarded cerebral circulation. The exposure was made at the interval of time after injection which usually shows cerebral veins and the sagittal sinus in part. The anterior cerebral artery was median in position, but in the lateral view was displaced considerably downwards in the posterior part of its course. It is important that the sagittal suture had been 'sprung' by the blow on the head.

Paired parietal burr holes were made not far from the midline, and a quantity of fluid blood and some clot was evacuated from the extradural space. Bleeding apparently ceased and the wounds were closed, with drainage.

Headache and vomiting continued, and catheterization of the bladder was necessary. Two days after the intervention the burr holes were re-exposed and a further accumulation of fluid blood and clot was evacuated. When saline was injected into the lumbar theca a small additional amount of old clot was extruded. Ventriculography showed nothing alarming. Thereafter improvement began, after an initial tendency to drowsiness, and urinary retention. The blood sugar was kept well under control. At the time of the patient's discharge home a month after admission the papilloedema had not quite subsided, and the left plantar reflex was still extensor in type. When seen two months later the patient was in excellent condition, with normal fundi.

In this case the sagittal sinus and drainage of blood into it was compromised more posteriorly than in the previous case, and the important Rolandic veins probably escaped severe compression. The syndrome was certainly much more benign and slower in its development than in Case 1.

Osteoplastic craniotomy would have been more effective, but lesser measures were preferred, and were successful, in this known diabetic case.

Case 3.—A man, aged 46 years, immediately lost consciousness lasting for about 20 minutes after a motor-cycle accident. Radiographs showed a fissure fracture of the vault disposed antero-posteriorly and crossing the midline. On admission to hospital a fairly large haematoma was evident under the scalp at the vertex. The pulse was 88/min. No abnormal neurological findings were apparent. Headache and vomiting continued after admission, and on the fourth day the pulse rate had fallen to 56/min. Headache increased and next day early papilloedema was seen. No new abnormal neurological signs were found on transfer to the neurosurgical unit: in particular, the plantar responses were normal. Carotid angiography showed separation of the sagittal sinus away from the vault of the skull over a considerable extent (Fig. 4). The pulse quickly returned to a normal

FIG. 4.—Case 3: lateral angiogram.

rate and headache ceased over the next two days. On that account operation was deferred. On the ninth day after injury lumbar puncture revealed a pressure of 270 mm.; the colourless fluid was normal. When the patient was transferred to the hospital of origin after 10 days of observation the ocular fundi were normal and he had remained free from symptoms.

In this case the clot was thin and perhaps extensive, so that there was no focal compression of the sinus and its tributaries.

The last case in this series is of interest because the extradural haemorrhage was larger than in any of the foregoing cases. It was located more anteriorly. The angiogram seems to show that the venous drainage from the hemisphere superiorly, though distorted, was little impeded. There was no papilloedema.

Case 4.—A man, aged 39 years, was involved in a motor-cycle accident, of which details are not available.

FIG. 5.—Case 4: lateral angiogram.

On admission to hospital soon afterwards the patient was conscious, with amnesia for the accident. Apart from a pulse rate of 48 to 60/min. and extensor plantar responses bilaterally, his condition remained good. On the second day after the accident a left hemiparesis was noted, and transfer to neurosurgical care was arranged. The blood pressure was 130/80 mm. The ocular fundi were still normal. The hemiparesis was of flaccid type, and only the left plantar response remained extensor. A fissure fracture crossed the vault transversely.

The angiogram is shown in Fig. 5, exposed at a normal timing. The preceding films of the series showed a free and active arterial to venous intracranial circulation.

This patient was operated upon because of the onset of a delayed post-traumatic hemiparesis. The large extradural clot, at most 3 cm. thick, was evacuated by right frontal craniotomy.* Next day some motor recovery was noted and was complete by the third day. On discharge from hospital two weeks after operation the plantar responses were almost symmetrical.

Discussion

In all of the cases recorded there was a fracture or diastasis of the vault near the vertex. In addition intracranial hypertension was manifest as a leading clinical feature in three of them. One patient had a lucid interval and the others had a latent period before disturbing neurological signs became evident.

With reference to the first case, the degree of retinal venous engorgement was remarkable. Angiography demonstrated a notable retardation of the circulation in the vessels draining into the sagittal sinus in the Rolandic region overlaid by clot. This was to a lesser extent true also in Case 2. The volume of the clot in Case 1 was small, comparable to that of many clots in the commoner sites, which declare their presence insidiously over many days and cause no alarming syndrome. The hemiplegia was of delayed but rapid onset: its complete disappearance overnight suggests that it may well have been due to cerebral venous obstruction, and particularly compression of thin-walled lacunae. The contrasting angiographic appearance in Case 4 with a voluminous clot is relevant to the argument.

Impairment of the efficiency of resorption of cerebrospinal fluid by the local pressure of extradural clot may perhaps be a lesser factor contributing to intracranial hypertension.

A survey of the literature has so far revealed only three illustrations of comparable angiograms in radiological reports without clinical information (Wickbom, 1949; Pecker, Javalet, and Stabert, 1959; Tiwisina and Stäcker, 1959). It has been learnt, in a personal communication from Professor Pecker, that his case had no papilloedema. He has also

*Operation by Mr. R. M. Varma, now neurosurgical consultant in Bangalore, India.

operated successfully on another patient with a syndrome very similar to that in Case 2.

Lindgren (1960) makes passing reference to a case in which operation was not required.

Petit-Dutaillis, Guiot, Pertuiset, and Le Besnerais (1956) describe a case of haematoma in the posterior fossa revealed by separation of the torcular Herophili from the skull. This patient had not developed papilloedema over a period of observation extending to several days (personal communication from Dr. Le Besnerais). The sinus was of course separated from the skull well posteriorly.

Concerning treatment it is evident that operation is not required in all cases. When the syndrome is florid and acute, or persistent in spite of lumbar puncture, craniotomy seems to be indicated.

These four cases have been encountered in the past four years, so that the condition is probably not rare.

Summary

Four cases of extradural haematoma at the vertex, revealed by angiography, are described; in all, the calvarium was fractured at or near the midline

superiorly. In three of the cases papilloedema developed and in two of those operation was necessary.

It is suggested that compression of the lacunae into which the Rolandic veins pour their blood superiorly may be the chief factor in the rise of intracranial pressure noted in the cases, and in the production of the hemiplegia which in one case vanished overnight after removal of a haematoma of relatively small size. In one case the cerebral venous outflow was demonstrably retarded.

I am much indebted to my colleague, Mr. D. G. Phillips of Bristol, for allowing me to include Case 2 in the series. Mr. D. O. Hancock, senior registrar, kindly drew my attention to some of the references, which he had obtained for another paper.

REFERENCES

- Johnson, R. T. (1957). In *Modern Trends in Neurology (Second Series)*, p. 277. ed. D. Williams. Butterworth, London.
 Lindgren, S. O. (1960). *Acta chir. scand.*, Suppl. 254, p. 20.
 Pecker, J., Javalet, A., and Stabert, C. (1959). *J. Radiol. (Paris)*, 40, 623.
 Petit-Dutaillis, D., Guiot, G., Pertuiset, B., and Le Besnerais, Y. (1956). *Presse méd.*, 64, 521.
 Tiwisina, T., and Stäcker, A. D. (1959). *Chirurg*, 30, 344.
 Wickbom, I. (1949). *Acta radiol. (Stockh.)*, 32, 249.