Benign post-traumatic intracranial hypertension¹

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Intracranial hypertension in closed head injuries, whether of slow or rapid development, is usually attributed to extravasation of blood into one of the intracranial spaces or anatomical structures. Epidural haemorrhage, subdural haematoma, subarachnoid bleeding, or an intracerebral clot are well-recognized entities. General cerebral oedema, a syndrome still not understood, must also be taken into consideration in the differential diagnosis of post-traumatic intracranial hypertension. However, another group of patients exists, with increased intracranial pressure following relatively mild head injuries, which so far has not been sufficiently recognized and has received little attention in the medical literature. In 1955, Foley, in an extensive review of a clinical syndrome characterized by increased intracranial pressure with a benign clinical course, suggested a new term, 'benign form of intracranial hypertension'. Among other factors in the aetiology of this syndrome he considered head injury, which occurred in four cases of his series. In the same year, Martin described five cases of what he called 'traumatic hydrocephalus' because of their clinical similarity to 'otitic hydrocephalus' and he attributed the increased intracranial pressure, which followed some time after a relatively mild head injury, to interference with the venous blood return following obstruction of the superior longitudinal sinus. At the first International Congress of Neurological Surgery in 1959, Kinal reported four additional cases of traumatic thrombosis of the dural venous sinuses in closed head injuries, proven by sinusography or surgery.

This paper is concerned with this syndrome, of which seven cases have been seen, four in the last year. The important data in these cases are summarized in Table I. Two cases are reported in detail, those with the shortest and longest interval between injury and the appearance of signs of increased intracranial pressure, respectively.

CASE REPORTS

CASE 1 A girl, aged 10, was knocked down by an automobile while crossing the road on 5 May 1951 and

¹Presented at the Second European Congress of Neurological Surgery, 18-20 April 1963, Rome.

lost consciousness. She was taken home where she regained consciousness shortly after and vomited several times. During the following two hours she complained of severe headache and pain in her left knee and was therefore brought to hospital. On examination she was found to be conscious and there were superficial abrasions of the scalp in the occipital region and the left knee. The only neurological findings were bilateral extensor responses. A radiograph of the skull revealed a linear fracture crossing the right lateral sinus. The course of her illness in the ward during the following four days was complicated by occasional vomiting only. On the fifth day of her hospital stay her pulse rate dropped to 50/min. and a convergent squint appeared. On examination papilloedema of 2 to 3 diopters in each eye was discovered. The other neurological findings consisted of bilateral sixth nerve paralysis and extensor plantar responses. The same day she became disorientated and incontinent. A lumbar puncture revealed clear cerebrospinal fluid under pressure of 600 mm. of water. There were no cells in the cerebrospinal fluid and its chemical composition was normal. Intracranial bleeding was suspected but a ventriculogram disclosed the ventricular system to be of normal size and shape without any shift. Considering the fracture in the skull, crossing the right lateral sinus, a diagnosis of sinus thrombosis was entertained. The treatment consisted of repeated drainage of cerebrospinal fluid. The girl's condition improved steadily and on discharge from the hospital after three weeks slight blurring of disc margins was the only abnormal finding. She was followed-up in the out-patient department for some time and last re-examined after 12 years, at which time there were no neurological sequelae.

CASE 2 A married woman, aged 22, was knocked down by an automobile on 30 September 1962. She was taken to a hospital in town and from there transferred to our department. She had been unconscious for less than one hour. On admission she was conscious but dazed, complained of headache, and vomited. Examination revealed a right parietal scalp haematoma and mild meningeal irritation. A radiograph of the skull showed a linear fracture in the right parietal and temporal bones extending to the base. The meningeal signs disappeared after four days and she was discharged from hospital on the tenth day after injury to be followed in the out-patient department. She was re-examined routinely twice, and except for occasional attacks of headache there were no other symptoms or signs. On the third visit papilloedema of 6 diopters with multiple haemor-

TABLE I
SUMMARY OF CASES IN THE PRESENT SERIES

No.	Age (yr.)	Sex	Head Injury	Time Between Injury and Syndrome (days)	Symptoms	Signs	Radiograph
1	20	M	Laceration of scalp (occipital), loss of consciousness for 1 hr.	7	Headache, disturbance of vision	Bilateral papilloedema 1-2 diopters + haemorrhages; bradycardia 50-56/min.	Linear fracture crossing left transverse sinus
2	10	F	Loss of consciousness for 1 hr.	5	Headache, vomiting, diplopia	Bradycardia 50/min.; bilateral papilloedema 2-3 diopters, bilateral sixth nerve palsy	Linear fracture crossing right transverse sinus
3	8	F	Mild, no loss of consciousness	14	Headache, vomiting, dizziness	Bilateral papilloedema 2 diopters + haemorrhages, nystagmus	Separation coronal suture
4	56	F	Mild, unconscious several minutes only	31	Headache, apathy	Bilateral papilloedema 2 diopters, small haemorrhages; sixth nerve palsy left	No fracture
5	27	M	Mild, unconscious short time	6	Headache	Bradycardia 54-64/min.; bilateral papilloedema 1 diopter; meningeal irritation	Linear fracture within sagittal sinus with depression internal table
6	22	F	Loss of consciousness for 1 hr., R. parietal scalp haematoma	52	Headache, blurred vision	Bilateral papilloedema 6 diopters +; haemorrhages, enlarged blind spots	Linear fracture right parietal extending to the
7	20	M	Unconscious for 1 hr.	21	Headache	Bilateral papilloedema 1-2 diopters	No fracture

rhages was discovered in both fundi and she was readmitted to the ward. Visual acuity was 5/5 in both eyes but the blind spots were enlarged. There was no other neurological deficit. The electroencephalogram was normal. A right carotid angiogram was considered normal. In view of the marked papilloedema with enlargement of the blind spots and in order to prevent deterioration of vision, a right subtemporal decompression was performed. During the days following operation Urevert was also administered intravenously. The bulge at the site of the decompression decreased slowly in size, the haemorrhages absorbed, and so did the swelling of the papillae. A lumbar puncture after three weeks revealed clear cerebrospinal fluid under pressure of 200 mm. of water. On discharge from the hospital 24 days after her second admission the swelling of the discs had decreased to about 1 diopter. Re-examination six weeks later disclosed normal discs. The patient had no complaints.

DISCUSSION

From an analysis of this material, together with that in the literature, a definite clinical syndrome can be described.

There is always a history of mild to moderate injury to the head. In only one of my cases, a girl of 8, was there no concomitant brain concussion; all the others had lost consciousness for a short time but never for more than one hour. None of Foley's cases was associated with brain concussion; three out of five of the patients reported by Martin and all four of Kinal's patients were rendered unconscious at the time of injury.

No patient exhibited signs of shock either immediately after the injury or thereafter.

In the majority of the cases, the impact to the head was in the occipito-parietal region. Scalp laceration and skull fractures in the posterior part of the head were, therefore, common findings in these patients. Of 17 reported x-ray examinations of the skull, only five showed no radiological evidence of fracture and in 10 cases the fracture was topographically related to the underlying venous sinus, six fracture lines crossing one of the transverse sinuses and four traversing or impinging upon the longitudinal sinus.

The time which elapsed between the head injury and the discovery of signs of intracranial hypertension ranged from several days to many months, the longest being 18 months, in one of Foley's cases. Although the symptoms date from the time of injury to the head, there was always an interval of relative freedom before re-aggravation of symptoms appeared. The most common complaint was headache, generalized in character and episodic. Blurring of vision and diplopia were next in frequency. Nausea and vomiting were common in children. The dominant clinical signs are bradycardia, papilloedema, sometimes accompanied by haemorrhage and exudate, and occasionally sixth nerve palsies on one or both sides. Meningeal irritation or extensor toe reflexes were occasionally present.

The lack of focal neurological signs and the general well-being of the patients, when relieved of

TABLE I—continued

SUMMARY OF CASES IN THE PRESENT SERIES

Lumbar Puncture		E.E.G.		Contrast Studies		Treatment	Results	Follow-up (yr.)
Cerebrospinal Fluid Pressure (mm. H ₂ O)	Cells	Protein (mg.-%)	Wassermann Reaction					
310	0	25	Negative		—	Repeated C.S.F. drainage	Recovery	19
600	0	25	Negative	Diffuse abnormal activity	Normal ventriculogram	Repeated C.S.F. drainage	Recovery	12
300	0	15	Negative	Diffuse slow waves	Normal ventriculogram	Repeated C.S.F. drainage	Recovery	8
				Diffuse abnormal activity	—	Diamox	Recovery	1
				Normal	Bilateral carotid angiogram normal		Recovery	1
200	0		Negative	Normal	Rt. carotid angiogram normal	Rt. subtemporal decompression Urevert	Recovery	1
220	0	53	Negative	Normal	—	Diamox	Recovery	1

headache, in spite of the raised intracranial pressure, were striking features.

The E.E.G. was not contributory. Normal records were obtained in about half of the cases and the others showed diffuse abnormal slow activity. Lumbar puncture revealed high manometric pressures but the cerebrospinal fluid usually showed a normal cytological and biochemical composition.

With such a clinical picture, a diagnosis of 'benign form of traumatic intracranial hypertension' may be entertained. Contrast studies in the form of ventriculography or angiography will definitely rule out any space-occupying lesion. Once aware of this syndrome, an expectant attitude can be undertaken and the benign nature of the process will prove itself by spontaneous clinical improvement. This may be accelerated by the administration of dehydrating agents such as Diamox or urea preparations. Only occasionally, when long-standing papilloedema may endanger vision, may surgical decompression be indicated. Such danger can be appreciated if the visual acuity and blind spots are repeatedly examined.

The mechanism involved in this form of intracranial hypertension is most probably partial or complete thrombosis of one of the major sinuses, such as the sagittal, the torcular Herophili, or the dominant transverse sinus. Thrombosis of the venous sinuses in closed head injuries, found at necropsy, was described by Bagley (1934) and by Carrie and Jaffé (1954). They found that the wall of the superior

sagittal sinus contains small sinusoids which represent the continuation of the contributory veins. Owing to their fine structure, these sinusoids are very vulnerable, and their rupture may result in haemorrhages within the wall of the sinus, possibly leading to injury of the lining endothelium with subsequent thrombosis. Skull fractures crossing or impinging upon the sagittal or one of the transverse sinuses, noted in 10 cases, could well injure the wall of these venous channels with ensuing thrombosis. In three of Kinal's patients this was shown to be the case by direct sinusography. In his fourth case, laceration of the wall of the sagittal sinus by a bone fragment was proven at operation. The obstruction in the lateral sinuses was complete in two and partial in the third. In four of our patients skull fractures at the site of sinuses probably caused injuries to their walls and thus could have given rise to the thrombosis of the sinus. In the fifth patient, a linear fracture extending from the right parietal through the temporal bone to the base of the skull might well have injured the sphenoid sinus. In the remaining two patients in whom no fracture was diagnosed, the deformation of the skull at the time of impact to the head could, as suggested by Martin, well result in damage to the wall of the sinus. In two of my patients ventriculography was performed to rule out a space-occupying lesion. The ventricles were of normal size and shape. Carotid angiography, carried out uni- or bilaterally, was of no help in outlining the sinuses. Sinusography was

considered as too major an intervention for a benign disease.

That this syndrome is self-limiting is confirmed by its benign clinical course. It is assumed that recanalization of the thrombosed sinus or the establishment of a venous collateral circulation reduces the venous congestion so that cerebrospinal fluid may be normally absorbed. Treatment should be supportive only. Dehydration, particularly with urea preparations, will give the patient temporary relief from headache. In only one case, with severe papilloedema up to 6 diopters and increasing blind spots, was subtemporal decompression indicated in order to avoid secondary optic atrophy. The outcome of the disease in all cases was complete recovery with no deficit in visual acuity or visual fields.

SUMMARY

In summary, seven cases of so-called benign intracranial hypertension following mild to moderate head injury have been presented. A review of the literature revealed 13 other cases. It is suggested that partial or complete thrombosis of the sagittal sinus, the torcular Herophili, or the dominant lateral sinus, with subsequent interference in venous blood flow and normal absorption of cerebrospinal fluid is the mechanism of this clinical syndrome.

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