THE DIAGNOSIS OF CHRONIC SUBDURAL HÆMATOMA OF TRAUMATIC ORIGIN

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INTRODUCTION

Within recent time there has been a considerable increase of the literature on chronic subdural hæmatoma. The personal observation of several cases recently within the space of a year is evidence of its clinical importance and is perhaps sufficient justification for redrawing attention to this not uncommon condition. Examination of the literature suggests that certain aspects of the symptomatology are not receiving the recognition they deserve; the cases here recorded are important in that they illustrate those aspects to which it is desired to draw attention. Failure to bear in mind the possibility of the condition may result not only in valuable time being lost before the correct treatment is instituted, but may prove fatal to the patient.

No useful purpose is served by distinguishing between the more acute and the more chronic types of subdural hæmatoma. There is, however, a type of case in which severe and sometimes rapidly fatal symptoms of intracranial hæmorrhage develop immediately after trauma. Operation or autopsy shows that the lesion in some of these cases is an acute subdural hæmorrhage. In the author's opinion such cases should not be regarded as examples of chronic subdural hæmatoma. There is sometimes a tendency to confuse these two states; it is felt that case 6 reported by Kaplan is essentially an example of an acute subdural hæmorrhage. In chronic subdural hæmatoma the latent interval which elapses between the occurrence of trauma and the onset of symptoms varies considerably, and in those cases where symptoms develop immediately after trauma the initial lesion is presumably a small subdural hæmorrhage which passes unrecognized as such and gradually develops into a true hæmatoma; in cases where the latent period is long, it seems probable that the slowly leaking blood collects and does not give rise to symptoms until after a definite hæmatoma is formed.

It is proposed in this paper to give a clinical description of the cases observed and to limit subsequent discussion to a consideration of the occurrence of raised intracranial pressure.

CASE REPORTS

Case 1.—M.B., male, age 5 months.

The history is incomplete as the patient had only been adopted by the foster
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Parents one week previous to the onset of symptoms and had seemed well at that time. As far as could be ascertained the infant had been previously healthy and there was no history of trauma; details of the birth were unknown.

State on Admission.—The infant was deeply comatose, groaning occasionally and moving all limbs; there was some vomiting; the temperature was 100° F.; meningeal signs were absent. The head was large and there was faint bruising of the skin of the right frontoparietal region; the fontanelles were tense. The eyes were bulging and there was an external divergent strabismus. There was bilateral ptosis and the pupils were fully dilated and inactive to light. There was marked bilateral papilloedema of the haemorrhagic type.

The diagnosis made was that of severely raised intracranial pressure from an undetermined cause. In view of the almost moribund state of the patient it was decided not to investigate the condition further and the infant died within a few hours of admission.

Autopsy.—There was faint external bruising of the subcutaneous tissue of the right frontoparietal region, and an effusion of blood into the pericranial tissue of that area and into that of the left parieta temporal region. A subdural haematoma of considerable size lay over the convexity of the right cerebral hemisphere, particularly in the parietooccipital region, and a smaller one over the convexity of the left cerebral hemisphere. On both sides the lesion was more marked adjacent to the superior longitudinal sinus. The sites of origin of the haemorrhage were not determined. There was a considerable degree of internal hydrocephalus present, and the cerebrospinal fluid in the ventricles was free of blood. There was no evidence of any external hydrocephalus. The skull was normal apart from its hydrocephalic shape and abnormal separation of the sutures.

Case 2.—W. T., male, age 40 years.

History.—The patient was well until ten weeks previous to admission to hospital, when he developed a feeling of soreness at the back of the head and pain there on coughing. The pain was at first intermittent, but gradually became more constant and severe, and was associated with slight general headache. He continued to work as an engine driver, but after a period of six weeks developed blurring of vision and diplopia. During the month previous to which he came under personal observation the headaches became easier, but the soreness and pain at the back of the head and neck continued. Apart from vomiting on two occasions there has not been any other symptoms.

State on Admission.—The patient was fully conscious, cooperative, and in no severe pain. There was marked bilateral papilloedema of the haemorrhagic type. The visual fields were normal apart from some enlargement of the blind spots. The pupils were of medium size, and the left was slightly the larger; they reacted normally. The ocular movements were normal apart from some limitation of outward movement of the left eye which showed a slight downward and inward strabismus. There was a trace of weakness of the lower part of the left side of the face on voluntary movement. The motor system was normal apart from slight hypotonia of the left upper limb. The reflexes were normal, but on one occasion the tendon jerks of the left upper limb and the left knee jerk were increased. The blood pressure was 140/74. External examination of the skull did not reveal any abnormality. X-ray examination of the skull showed some local thinning of the cranium at the vertex on the right side, with a much enlarged venous marking extending from this point to the base; the sella turcica was enlarged. The cerebrospinal fluid was clear and colourless and had an initial pressure of 400 mm. of water; the cells were normal; the protein content was 0·08 per cent., and there was a trace of globulin present.

The diagnosis made was that of increased intracranial pressure from an extracerebral lesion, probably a menigioma, situated in the right posterior parietal region.
On admission the patient denied a history of trauma but later stated that five days before the onset of symptoms he had received a fist blow of moderate severity in the left temple, without any obvious ill-effect. In view of this history of trauma, it was felt that there was a possibility of the lesion being a subdural haematoma. During the month that elapsed whilst the patient was under observation, it was noticed that the retinal haemorrhages disappeared and the papilloedema largely subsided without the onset of any consecutive atrophy of the discs. During this time the patient was confined to bed and the suggestion was made that the supposed haematoma was shrinking; by this time the patient was free of all symptoms. In view of the sparsity of physical signs and the unusual subsidence of the papilloedema, ventriculography was attempted; neither lateral ventricle could be located.

Operation.—The right posterior parietal region was explored, and a large encysted subdural haematoma was found and evacuated. The patient made an excellent recovery.

Case 3.—C. W., male, age 47 years.

History.—The wife of the patient stated that he was well until six weeks previous to admission to hospital, when he was knocked down by a motor car whilst returning home from work. He sustained a small cut over the right temple and a contused right eye, but was not rendered unconscious. He was taken to the local hospital, and sent home without being detained. He seemed a little dazed on reaching home but slept well that night, and seemed normal on the following morning. During the next three weeks he seemed well and continued work as a builder. He then complained of frontal headaches, which recurred in increasing frequency and severity, and was observed to have trembling of both hands, more marked on the right side. There was slight vomiting, increasing unsteadiness of gait and progressive mental apathy. During the two days previous to coming under personal observation he had been very drowsy and occasionally comatose.

State on Admission.—The patient was in a condition of rousable coma; the pulse was slow (40–50 beats a minute) and there was frequent yawning. There was intense bilateral papilloedema with many old and recent haemorrhages. The pupils were of medium size, and the left was slightly the larger; both reacted to light. There was doubtful asymmetry and weakness of the lower part of the right side of the face. He could move all limbs, but there was a flaccid weakness on the right side, more marked in the upper limb. The tendon jerks were all present, but less easily elicited on the right side; the plantar responses were extensor; the right abdominal reflexes were absent. Pinprick appeared to be appreciated normally on both sides of the body; other sensory examination was impossible. Blood pressure was 130/70; temperature was normal. There was marked tenderness of the skull to palpation and percussion in the right frontal region. On the few occasions when he could be roused, there appeared to be marked difficulty in naming objects, but in view of the comatose condition of the patient this was not interpreted as evidence of a true dysphasia. The cerebrospinal fluid was clear and colourless, and appeared to be under increased pressure (no measurement recorded); protein content was 0·08 per cent., and there was a trace of globulin present; cells were normal.

The diagnosis made was that of a leftsided subdural haematoma, but in view of the marked tenderness of the skull in the right frontal region it was decided to explore both sides.

Operation.—The left prefrontal area was trephined, and no haematoma was found. Exploration on the right side revealed a large tense encysted subdural haematoma. This was evacuated with as little disturbance as possible and the patient made a complete recovery.

Case 4.—T. R., male, age 82 years.

No history was obtainable of the onset of symptoms.
State on Admission.—The patient was an old man, very collapsed, semiconscious, mumbling incoherently at times and resenting examination; occasionally he would try to get out of bed. There was almost continuous twitching of the left side of the face and of the right upper limb; there was a flaccid paralysis of the left upper limb. The fundi showed, as the only abnormality, some arteriosclerotic changes. Both kneejerk2 were absent, the anklejerk3s were present and there were bilateral extensor plantar responses. The blood pressure was 200/90, and there was marked thickening and tortuosity of the peripheral arteries. Meningeal signs were absent. External examination of the skull was normal. There was incontinence of urine and faeces; the temperature varied from 100° F. to 101° F., and the clinical picture remained unaltered until death, two days after admission. The cerebrospinal fluid was clear and colourless; no biochemical or cytological examination was made; the pressure was not raised.

The diagnosis made was that of diffuse cerebral vascular disease; it was assumed that there were multiple thrombotic lesions, and possibly a small rightsided cerebral haemorrhage in addition.

Autopsy.—There was a large encapsulated subdural hematoma covering the greater part of the right temporal and parietal lobes. Apart from flattening and senile rinkage the underlying brain was normal. There was generalized and severe atheroma of the cerebral arteries. The site of origin of the haemorrhage was not ascertainable. The skull was normal.

Case 5.—L. S., female, age 43 years.

(This case is recorded because of the unusual operative findings, and because the clinical picture was suggestive of a chronic subdural hematoma, which was the preoperative diagnosis. It is not included in the subsequent discussion.)

History.—The patient was well until one week previous to the onset of symptoms when, one night, she got out of bed and pulled the washstand on top of her. A week later she awoke during the night with a severe headache, and vomited; she got out of bed and walked into an adjacent room, on reaching which she apparently fell and struck one side of her head, and as a result of this blow she lost consciousness. Later in the morning she was found lying semiconscious on the floor, and was eventually taken in that condition to hospital, where she stayed for one month. So far as can be ascertained she remained semiconscious for four days, after which her general condition improved. When she came under personal observation, which was one month after the onset of symptoms, she stated that for one week after her collapse on the floor she had had frequent severe pains in the right side of her head, and had been unable to open the right eye. During this time she had paralysis of the left upper limb, and had complained of a sense of uselessness of the right limbs when the headaches were particularly severe. She also had difficulty in speaking, and fluids dribbled from the right angle of her mouth. On admission she was drowsy and apathetic, on account of which it was difficult to ascertain her symptoms with accuracy. For about three weeks she had had frequently occurring visual hallucinations; whether these occurred only on one side of her field of vision was never ascertained beyond all doubt. They appeared as fully formed figures and objects, such as vague whitish figures approaching her from round a distant corner, a fireplace with people seated round it, brightly coloured headgear worn by imaginary patients, black and white dogs. For about two weeks she had noticed a tendency to fail to see objects on her right side, and she had frequent diplopia.

State on Admission.—The patient was plethoric and obese, drowsy in appearance and pale in manner. There was marked bilateral papilledema. Bedside examination of the visual fields revealed a left homonymous hemianopic defect with some constriction of the right lateral fields. The pupils were of moderate size and the left was slightly the larger; they reacted normally. There was a slight internal strabismus of
the left eye and diplopia on looking to the left. The lower part of the right side of the face was asymmetrical and weak. The outstretched right upper limb tended to droop a little, and the handgrip was a little less strong on that side. The motor power of the lower limbs was impaired. All tendon jerks were exaggerated, more so on the left side; the abdominal reflexes were absent; the plantar responses were extensor but within a short time of admission they became equally flexor. Sensation was normal apart from slight impairment of the sense of position in the left digits. The blood pressure was 124/70. External examination of the skull revealed as the only abnormality faint bruising of the left periorbital region. X-ray examination of the skull was normal. The cerebrospinal fluid had an initial pressure of 190 mm. water; it was clear and colourless; the cell content was 6 per c.mm.; the protein content was 0.085 per cent., and there was a trace of globulin present.

The diagnosis made was that of a subdural haematoma situated in the right temporal region.

Operation.—A right temporal exploration was made. Through the unopened dura mater the brain was observed to be darker and more yellow than normal, and on reflecting the dura it was seen to be unusually vascular. Just above the Sylvian fissure there was a soft area in the brain which on being needled yielded about 10 c.cm. of old blood-stained fluid. Incision into this area revealed a large haematoma extending into the temporosphenoidal region. The haematoma was evacuated and the patient made an uneventful recovery.

DISCUSSION

It is apparent that there was an increase of the general intracranial pressure in three out of the four cases of subdural haematoma here recorded; this increase was most severe in the infantile case and only absent in the senile case. In another instance, not here recorded, that of a boy, age 15 years, who had a large subdural haematoma in the left frontotemporal region, for one month there had been persistent headache and vomiting, and here also there was intense papilloedema, indicating a marked increase of the general intracranial pressure. It is of importance to determine why raised intracranial pressure occurs in some but not all cases of chronic subdural haematoma, for it would seem that the symptomatology of the condition is determined largely by its presence or absence. Its presence results in a much more definite clinical picture, which may have to be differentiated from other common conditions such as cerebral tumour; in its absence, the clinical features of the condition are vague and difficult of recognition. It is suggested that its occurrence is determined largely by the age of the patient. Little attention appears to have been given to this point by the many authors who have contributed to our knowledge of the condition. The usual practice of considering separately the condition as it occurs during infancy and during adult life is probably partly responsible for this neglect of the age factor; neurologists see all but the infantile cases, which tend to be referred to the pediatrician. An examination of the condition, as it occurs throughout life, suggests that there is a sequence of symptomatology which at present is not receiving due recognition.

Trotter's account of the probable source of the haemorrhage and of the causation of symptoms in cerebral compression of this type in part explains
why there may be such variety and variability of the clinical pictures with so few symptoms and signs; the more recent work of Gardner and others offers additional explanation of their late onset and variability. It seems probable, however, that the indefinite clinical picture on which emphasis is usually laid in describing this condition only occurs with any degree of constancy during the later decades of life. The present writer suggests that during these years, and especially in the presence of such conditions as atheroma of the cerebral arteries, chronic alcoholism and dementia, the degree of shrinkage of the brain which is present probably prevents the occurrence of any increase of the general intracranial pressure, with the result that the clinical features of the condition are correspondingly indefinite. It has proved difficult to obtain information concerning the ages at which the normal human brain attains final adult dimensions and at which it normally commences to undergo shrinkage. According to Berry, the former occurs between the eighth and eleventh years of life; it seems not improbable that shrinkage also commences at a correspondingly early age. During the period in which the brain is enlarging and moulding the skull, rapid and severe increase of intracranial pressure is likely to be of frequent occurrence in this condition, with the result that the clinical picture is correspondingly more sharply defined; if the fontanelles are still open, the cranial bones will tend to yield to their fullest extent before the rising pressure. Once shrinkage of the brain has commenced, it is suggested that raised intracranial pressure gradually ceases to occur with such severity and frequency, until in the senile period, where the cerebral volume is minimal, it probably never occurs.

A comprehensive review of the records of cases reported in the literature fully supports the view that the clinical picture of the condition and the occurrence of raised intracranial pressure are both alike determined by the age period in which the lesion develops. During the infantile period, so constant and so marked is the increase of intracranial pressure which occurs that the clinical picture closely simulates that of 'idiopathic' hydrocephalus, as has been emphasized by Peet and Kahn; case 1 is an example. During the remainder of the growing period and during the early and middle periods of adult life, clinical evidence of raised intracranial pressure is commonly present: cases 2 and 3 are examples. Thus papilledema is of common occurrence at these periods of life, being present in approximately 60–70 per cent. of cases; where it is absent, the frequent occurrence of such a group of symptoms as progressive headache, vomiting, drowsiness and coma indicates the presence of raised intracranial pressure. During senescence, evidence of raised intracranial pressure is very uncommon in this condition and the symptomatology is more constantly vague and difficult of recognition; case 4 is an example.

References in the literature concerning the occurrence of raised intracranial pressure are variable and in some cases, it is felt, in need of amplification. Trotter states that definite optic neuritis is a rarity. Martin remarks
that there is little rise of the general intracranial pressure and that signs of
this are usually absent; he says, however, that papilloedema has been
recorded quite frequently, especially in the rather acute cases. Brain states
that papilloedema is often absent, though with reference to intracranial
birth injuries he mentions that papilloedema and retinal haemorrhages may
be present and the fontanelles bulging and non-pulsating. Grinker refers
to the paper by Peet and Kahn in connexion with the condition in infants;
in discussing the adult form he states that very slowly signs of increased
intracranial pressure develop, such as headache, choked disc, slow pulse and,
often, vomiting. Osler and McCrae mention that papilloedema is unusual.
Critchley, in a recent review, writes that symptoms directly referable to the
haematoma are vague and usually most difficult to evaluate, that mental
changes are the most characteristic symptoms and are very rarely absent,
and that papilloedema is sometimes found. Munro, in a review of 62 cases,
reports changes in the fundi (presumably papilloedema) 10 times, and in the
21 cases in which the blood pressure was raised this was attributed to the
increased intracranial pressure which was present; unfortunately he only
gives details of a few cases and it is difficult to accept all of these as examples
of the condition under review. In a recent paper in which he reports and
discusses the symptomatology of 16 cases, Furlow writes that headache is
the most common complaint and choked disc the most common finding.
The author is in agreement with this statement, though it is difficult to agree
with him that the symptomatology of the condition is vague, for in all but
the elderly and senile it tends to be one of raised intracranial pressure, most
characteristically so during the infantile period.

Note on Case 5.—Cases of traumatic intracerebral haemorrhage of this
type are uncommon. Oppenheim mentions that trephining for a traumatic
haematoma of the dura mater has frequently been without result because
the haemorrhage has had its site, not in the meninges, but deep in the substance
of the brain. Moulonguet refers to a few similar cases reported in the literature.
Craig and Adson have recently described nine cases of spontaneous
intracerebral haemorrhage, in some of which trauma or strenuous exercise
were contributing factors.

**SUMMARY AND CONCLUSIONS**

1. Four cases of chronic subdural haematoma and one case of intra-
cerebral haematoma of traumatic origin are described.

2. The frequency of the occurrence of raised intracranial pressure in
chronic subdural haematoma is stressed and discussed.

3. Emphasis is laid upon the relationship which appears to exist between
the age of the patient and the occurrence of raised intracranial pressure
in this condition.

4. The earlier in life the subdural haematoma develops, the more frequent
and severe does the raised intracranial pressure tend to become.
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5. It is only during the later years of life, when raised intracranial pressure is uncommon in this condition, that the clinical picture is so constantly vague.

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