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Delayed Post-Traumatic Haemorrhage of the Brain

N.O. AMELI, Ch.M. (B'ham), F.R.C.S. (E). F.I.C.S.*

The problem of intracerebral haematoma has received great deal of attention in recent years. (Jewsebury 1947, Margolis et al 1951, Falconer 1952, Ferey et al 1952, Barbé 1953, Werner 1954, Crawford & Russel 1956). There is no doubt that the condition is not rare and is often amenable to surgical treatment.

Signs of raised intracranial pressure due to an intracerebral haemorrhage may appear after a varying interval following a head injury. Böllinger(1891) under the title «Traumatische Spatapoplexie» described a condition of intracerebral haemorrhage occuring many months after an injury. This condition which is also called late delayed type is not dealt with here.

In this paper the accounts of fifteen cases are given in which the latent interval varied from two hours to one month.

At least in seven cases a predisposing factor was present.

These were: 4 angiomata, one cerebral tumour and two cases with arterial hypertension. Four cases had closed head injuries with presumably no underlying condition, two perforating wounds of the brain; and two cases of open depressed fractures of the skull.

*) Professor of Neurosurgery, University of Tehran.
Director of Neurosurgical Department, Pahlavi Hospital.

In 14 cases haemorrhage was of cerebral origin, and in only one case it was intracerebellar.

CASE REPORTS

Case 1.

A man of 32 was admitted to the Queen Elizabeth Hospital, Birmingham, in October 1947.

Eleven days before admission, after heading a football he collapsed. He soon recovered, and was able to walk home. He stayed at home the next day. Although he had some headache, he went to work the day after, and worked for four days. His headache continued and gradually his speech became unintelligible. His right hand became weak and he could not hold a cup or a cigarette. He was admitted to a local hospital, where a lumbar puncture was performed. This showed a xanthochromic fluid. On the 9th day bilateral papilloedema was observed.

On admission to Queen Elizabeth Hospital on the eleventh day he was deeply comatose with rapid and noisy breathing. Both pupils were moderately dilated, with no reaction to light. There was a twitching of the right side of the face. Tone was diminished in the right arm and leg with sluggish tendon reflexes and absent abdominals on the same side. Both plantar reflexes were extensor. Pulse rate was 64 per minute. X-ray of the skull showed no fracture.

Two exploratory burr-holes were made in the left temporal and frontal regions. No extradural haematoma was found. On inserting a cannula into the left temporal lobe some resistance was felt and œdematous brain tissue was aspirated.

Patient died a few hours later. Post-mortem examination of the brain showed a localised haematoma in the left temporal lobe.

Case 2.

A girl of 10 years was admitted to the Queen Elizabeth Hospital in October 1948. The day before admission she fell on her head whilst

playing at school. She did not lose consciousness, but when questioned by her father at home she appeared to be confused. During the night she was very restless and in the morning was semicomatose. In the afternoon she was admitted to the Queen Elizabeth Hospital.

On examination she was restless and could not be roused. The left pupil was slightly larger than right. Pulse was 80 per minute. Right limbs were less frequently moved than the left. Reflexes were all sluggish and both plantars were extensor.

During the next six hours she improved and could be roused, but died suddenly at 3.15 a.m.

Post-mortem examination of the brain.

The ventricles were filled with solid blood clot. There was a haematoma in the left frontal lobe. On carefully washing the clot a small angioma was found situated near the origin of the left middle cerebral artery. The haemorrhage must have occured from the rupture of one of the vessels of this angioma.

Case 3.

A boy of 12 was admitted to the Queen Elizabeth Hospital in November 1948. He had sustained a slight head injury at the swimming pool on the day before admission. He was able to walk home but there he complained of headache and vomited.

On admission to hospital the next day he was drowsy. There was a right homonymous hemianopsia to confrontation, and a right hemiparesis.

The arm was affected more than the leg. Pupils were equal. Pulse rate was 82 per minute. Ventriculography indicated a mass in the left temporal lobe. A left temporal burr-hole was made. No extradural haematoma was seen. On inserting a cannula into the left temporal lobe only few drops of dark blood escaped. Two days later his condition was worse. A subtemporal decompression was performed, and on

inserting a cannula into the brain substance 30cc. of old blood was aspirated. Patient's condition rapidly improved.

An angiography performed later showed a small arteriovenous angioma. (fig. 1.)

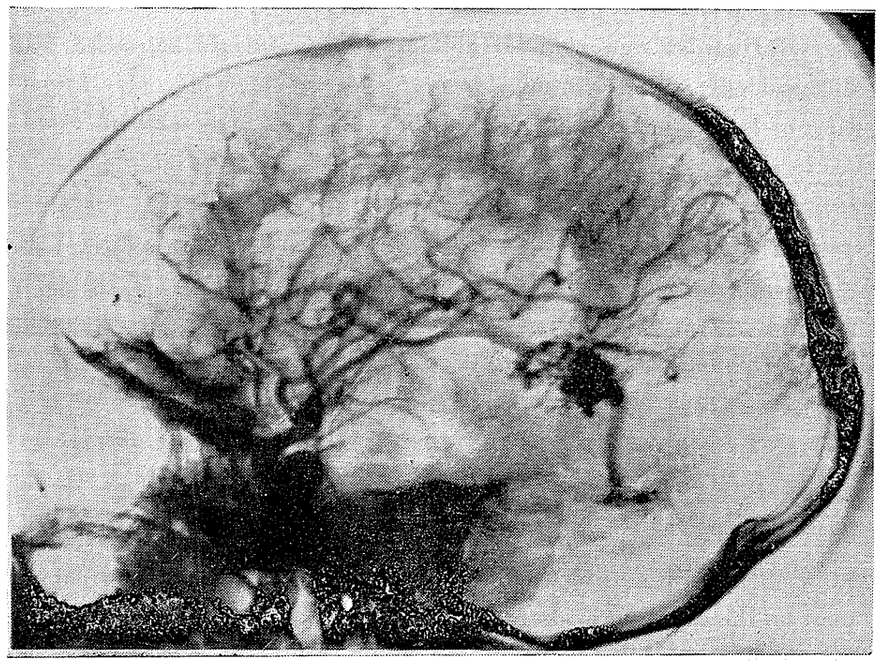

Fig. 1. Small arterio-venous angioma in the temporal region.

Case 4.

A boy of 18 years was admitted to Pahlavi Hospital in March 1951. For one week before admission he had complained of headache, and gradually became drowsy. On admission to hospital he was coma-

tose but could be roused. Pupils were equal and reacted to light. Pulse rate was 50 per minute. There was bilateral papilloedema. He had a left hemiplegia affecting the leg more than the arm and face. No sign of injury was visible on the surface of the skull.

As the patient's condition was rapidly deteriorating, it was decided to do an exploratory burr-hole. This was made 3cm. from the midline in the right anterior parietal region. A cannula was inserted into the substance of the brain in the hope of encountering a cyst or an abcess. At a depth of 2cm. a cavity was entered and 20c.c. of dark old blood were aspirated.

Patient regained consciousness whilst still on the table. His hemiplegia gradually recovered. He has remained free of symptoms since.

On questioning him later he stated that about 10 days before admission he had been boxing without gloves at the school and had received a hit on the head. As boxing without gloves was not permitted he had not mentioned this at home or at the school.

In this patient angiography performed after the operation did not reveal any abnormality. We were expecting to find an angioma.

Case 5.

A police officer aged 55 was admitted to Pahlavi Hospital in December 1952. For many years he had been under treatment for arterial hypertension.

Eight hours before admission, whilst dispersing a mob, he was hit on the head by a brick. This gave him a momentary dizziness and caused a bruise in the left parietal region. As his home was near he went there for rest and told his wife about the incident. After resting for half an hour he went to the police station to give in his report. An hour and a half after the injury he returned home complaining of headache. Half an hour later was semicomatose.

On admission to Hospital he was comatose. There was severe neck rigidity. Pupils were equal and moderately dilated. Pulse was 100 per minute. Temperature 38°. 5 C. Blood pressure was 220/150. There was

a complete right hemiplegia.

A lumbar puncture gave practically pure dark blood under pressure. Patient died next morning. Post-mortem was not performed.

Case 6.

A boy of 17 years was admitted to Pahlavi Hospital in March 1953. He had been well until eight days before admission. Then at a wrestling match his head was hit on the floor. At once he felt his right foot go numb. The referee stopped the match. He was able to walk home but had a slight headache.

Gradually in the next few days, his headache increased, his speech became difficult and became drowsy.

On admission to hospital he was comatose but could be roused, He appeared to be aphasic. He had bilateral papilloedema. He had a hemiparesis affecting the leg more than the arm and face.

This case was so similar to case 4 that a pre-operative diagnosis of intracerebral haematoma in the left parietal lobe near the midline was made.

Through a burr-hole in the left anterior parietal region a cannula was inserted into the brain substance. At a depth of 2cms. a cavity was entered and 30c.c. of dark blood were aspirated.

Patient at once regained consciousness. His hemiparesis gradually improved, but did not completely disappear, and remained slightly dysphasic. One week after the operation an angiography was performed. This showed gross displacements of the arteries suggestive of a parasagital mass. As the patient's condition was improving it was thought that this mass was blood clot in the process of absorption. He was discharged 14 days after admission.

He was readmitted three months later. The hemiparesis had gradually become worse, and lately he had been suffering from headache and vomiting. The dysphasia was more marked.

A new angiogram demonstrated further displacement of the arteries. Through left parietal osteoplastic flap a large solid infiltrating

tumour was removed. There was some old organised clot in the centre of the mass.

Histological report was a protoplasmic astrocytoma.

A course of deep X-ray therapy was given after the operation. Two weeks after the operation he left the hospital still dysphasic and hemiparetic.

He was last seen two years after the operation and there had not been any sign of recurrence.

Case 7.

A man of 32 years was admitted to Sina Hospital in April 1952 after a car accident. On admission he was unconscious, but regained consciousness after a few hours. Three days later he became drowsy, dysphasic and developed a right hemiparesis. He was transferred to Pahlavi Hospital five days after the injury.

On examination he was comatose, with right hemiplegia. Pupils were equal, and pulse rate 85 per minute.

With diagnosis of a left temporal lobe haematoma, a burr-hole was made in the left temporal region. Through a cannula 30cc. dark blood were aspirated from the left temporal lobe. Patient regained consciousness but had motor aphasia. His condition improved for two days. On the third night he rapidly went into coma and died within two hours.

Post-mortem showed a large solid haematoma in the left temporal lobe with much brain oedema in the surrounding area.

Case 8.

A man of 60 was admitted to Pahlavi Hospital in June 1952, five hours before admission had been hit by a taxi. He did not lose consciousness but soon became confused. He was able to give his name to the police, but could not remember his address. Until half an hour before admission he could walk normally and talk to those around him, but was disorientated and irritable.

On admission he was comatose but could be roused with diffi-

culty. Pupils were equal and reacted to light. Pulse was 90 per minute. Blood pressure was 190/140. He had left hemiparesis. It was known that the patient had been under treatment for arterial hypertension for many years.

A burr-hole was made in the right temporal region. There was no extradural haemorrhage. On opening the dura large quantity of fluid blood escaped. The incision and opening of the skull were enlarged. There was a large cavity full of fluid blood in the temporal lobe, which had opened into the ventricle. With difficulty hemostasis was achieved. Patients condition improved for a few hours, but died 18 hours after the injury. No post-mortem.

Case 9.

A man of 25 was admitted in May 1953 five days after receiving a stab wound in the right frontal region. After receiving the injury, the patient who had not lost consciousness was taken to the local hospital where the wound was dressed and he was sent home.

The next day he complained of headache. On the third day his headache became worse and he became drowsy. He was admitted to the hospital where he was first seen. He was then found to have a left hemiparesis. On the 4th day his condition was worse, but could still walk. On the 5th day he went into deep coma. He was then moved to Tehran, a distance of 100 miles. On admission he was deeply comatose, respiration irregular pulse 40 per minute, pupils equal and slightly dilated. There was bilateral papilloedema, and slight right hemiplegia.

Operation.

The original wound was only half an inch in length and clean cut, in process of healing. On opening the wound widely a small linear bony defect 1/2 cm. in length was seen where the tip of the knife had entered the external plate. On making a burr-hole near this defect and removing some bone, it was found that a small piece of internal plate had been separated by the force of the knife, and pushed into the brain substance. This piece of bone was removed and the dural tear excised and extended. On further incising the frontal lobe a large solid haematoma in the substance of the frontal lob was exposed. This was completely removed and bleeding points coagulated. The dural defect was repaired and wound closed. Patient's condition gradually improved, and regained full consciousness two days after the operation. His hemiplegia very slowly recovered. One month after the injury there was no trace of hemiplegia, and he has remained well ever since.

Case 10.

A man of 35 was admitted to Pahlavi Hospital in August 1954. He had been a known epileptic, and six days before admission he received a head injury during a fit, falling from a height of 9 feet and lost consciousness at once and was taken to Loghman Adham Hospital under Dr. Najm Abadi. Soon after admission he regained consciousness. X-Ray of the skull showed a fracture in the left temporal and parietal bones. For the next two days he complained of headache, but otherwise was normal.

On the third day he became drowsy and developed a right hemiparesis. He was then transferred to the Neurosurgical Department, Pahlavi Hospital.

On examination he was found to be comatose but could be roused with difficulty. There was bilateral papilloedema. Pupils were equal and reacted to light. Pulse was 52 per minute.

There was some difficulty in speech when roused. A right hemiparesis was present, face and arm more affected than the leg. Visual fields could not be examined. A left carotid angiogram showed a mass in the left temporal lobe. There was no angiomatous malformation.

Operation.

Under local anaesthesia a left temporal burr-hole was made and extended. Dura was widely opened. The lower temporal gyrus had a

greenish tinge. On inserting a cannula into the brain few drops of old blood escaped.

A horizontal incision was made in the left lower temporal gyrus.

At a depth of 1cm a cavity filled with solid clot was found. This was completely removed, and the few bleeding points coagulated.

Patient made an uninterrupted recovery with no residual disability. He was put on medication for control of his epilepsy.

Case 11.

A girl of 6 was admitted to hospital in May 1955. Seven days before admission she was accidentally shot at. (She had been playing in a field in a colourful dress, and she was taken for a pheasant.) She had received a small wound in the left frontal region. She did not lose consciousness, and her wound was dressed by her doctor. Four days later she began complaining of headache and vomited. She gradually became drowsy, and was sent to Tehran for treatment. On admission, she was semicomatose. Pupils were equal. Pulse 100 per minute. There was early bilateral papilloedema. She had slight right hemiparesis. The wound in the left frontal region had healed. X-ray of skull showed comminuted fracture of the left frontal bone, with minute fragments in the frontal lobe. There was a piece of metal in the occipital area. (Fig. 2.)

Under general anaesthesia the wound was reopened and enlarged. There was a small bone defect with irregular edges. This was enlarged with nibbling forceps. The dura was incised. A fairly large haematoma was removed from the frontal lobe. The piece of metal in the occipital area was not interfered with. She developed a C.S.F. leak for 7 or 8 days, after which the wound healed satisfactorily. She made complete recovery, and has remained well ever since.

Case 12.

A boy of 14 was admitted to Pahlavi Hospital in June 1955, four hours after falling off his bicycle. There was no loss of consciousness until 3 hours after the accident, when it came on suddenly following on

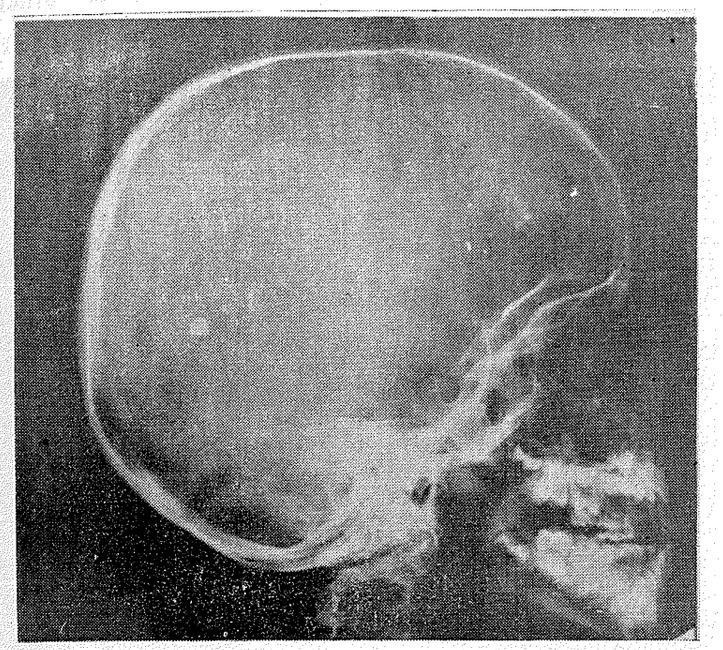

Fig. 2. In the frontal region bone destruction and small metallic particles, and in the occipital region the foreign body can be seen.

acute headache. On examination he was deeply comatose the pupils were equal but did not react to light. Breathing was noisy and laboured. Pulse was 120, temperature 38° 5 C. There was severe neck rigidity with head retraction. There was also a right hemiplegia. Lumbar puncture gave a heavily blood stained fluid under pressure. Relatives were given a very grave prognosis.

Next day patient's condition improved. He could be roused, and breathing was normal. He slowly improved, but it was soon obvious that he was aphasic.

On the 4th day a left carotid angiography was performed. This showed a mass in the left parietal region, surrounded by some abnormal

vessels presumably an angioma. (Fig. 3.)

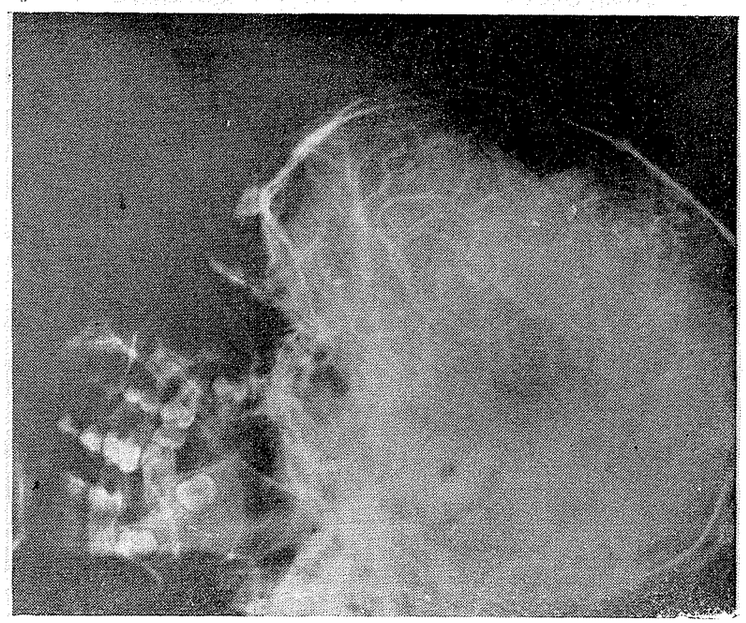

Fig. 3. Displacement of the vessels in the parietal region indicate an intracerebral mass. There is some suggestion of vascular deformity above this mass.

Patient's aphasia and hemiplegia gradually improved, and he was discharges 3 weeks after admission. Six months later he was readmitted. He still had a hemiparetic gait with some dysphasia. Angiography was repeated. This time a definite angiomatous malformation could be seen in the left parietal region (Fig. 4). Operation for removal of the angioma has so far been refused.

Case 13.

A man of 25 a bus conductor was admitted to Pahlavi Hospital in February 1955. He had been suffering from headache for one month.

In the last ten days his headache had been very severe and had vomited repeatedly. During the last 3-4 days his sight had rapidly diminished.

On examination it was obvious that he was suffering from severe headache. He was co-operative but not alert.

He had advanced bilateral papilloedema, nystagmus, adiodokokinesis and hypotonia of the limbs on the right side. He had a large angio-

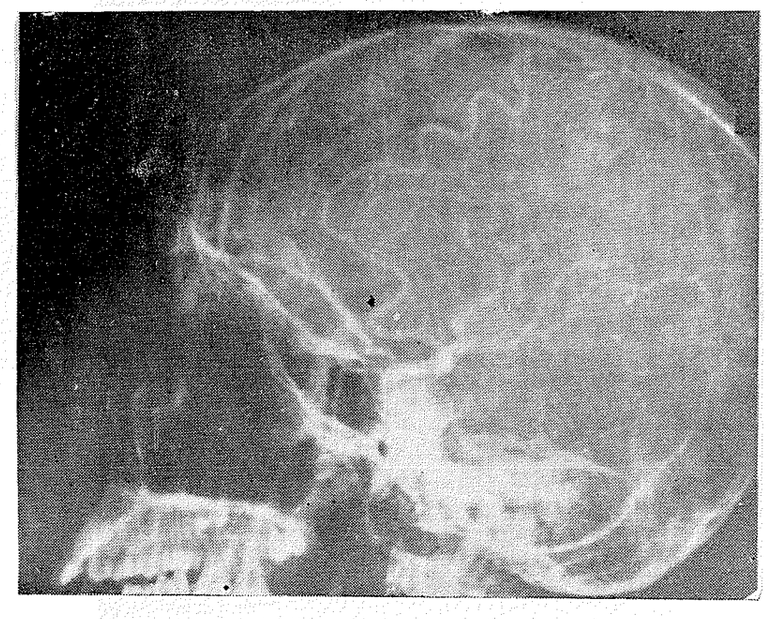

Fig. 4. An arterio-venous angioma can be seen in the anterior parietal region. (The same case as Fig. 3.)

ma occupying the anterior two thirds of the right side of his tongue. (Fig. 5.)

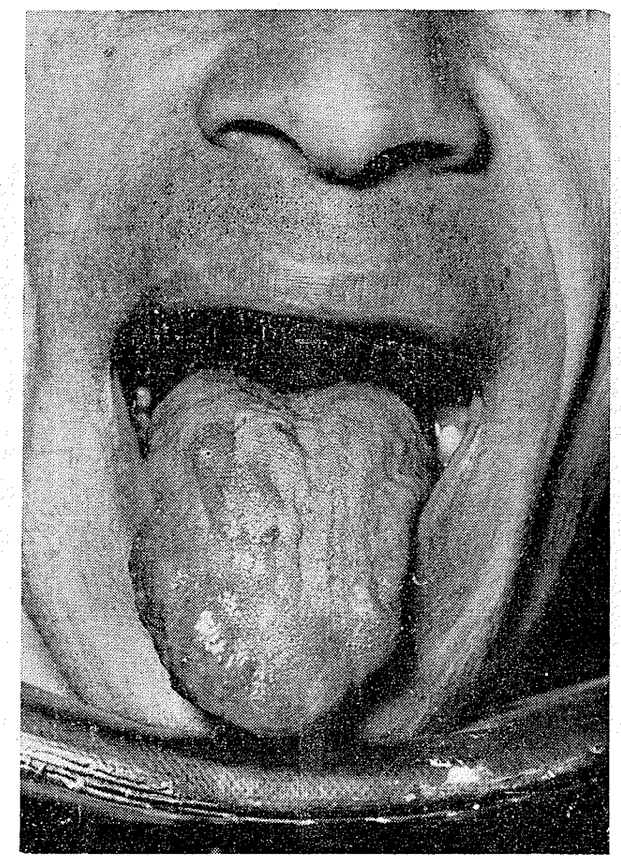

Fig. 5. Large angioma occupying the right side of the tongue.

Patient was carefully questioned regarding trauma. He stated that a week before the onset of the symptoms he had received a slight head injury in a brawl at a bus stop. A provisional diagnosis of right intracerebellar haematoma was made, with a possible congenital angiomatous condition as predisposing factor.

A posterior fossa decompression was performed. A cannula was

inserted into the right cerebellar lobe. Some resistance was felt but no cyst or liquefied haematoma was discovered. Following the decompression, patient's headache rapidly subsided. Cerebellar signs also improved, and he was discharged with only slight ataxia. Three weeks later he

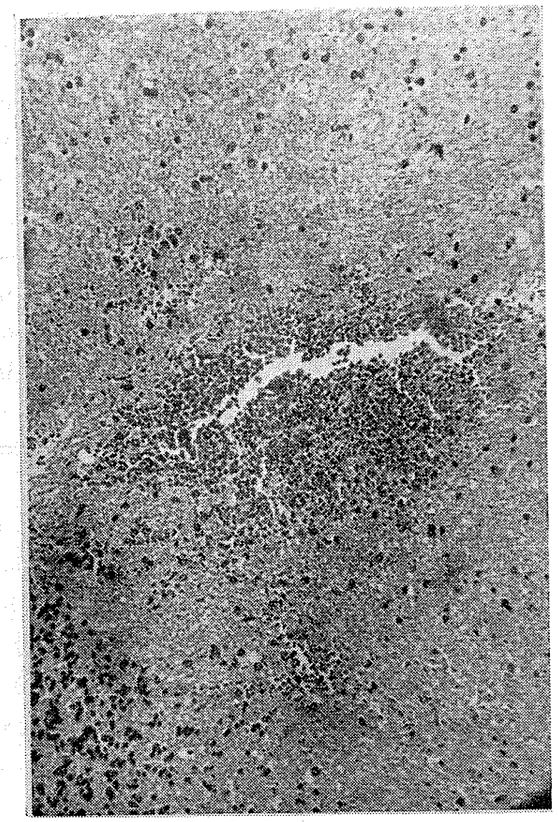

Fig. 6. Histological appearance of cerebellar tissue at the periphery of haematoma.

was readmitted with recurrence of his symptoms. He was reoperated, and this time an incision was made into the right lobe of the cerebellum.

There was a solid haematoma infiltrating the brain substance. Macroscopically no angioma could be seen. All the blood clot, and also

a small piece of cerebellum adjacent to the clot were removed. Following the operation patient made excellent recovery and has remained well since. Histologically the clot was reported to be in process of organisation. The cerebellar fragments showed interstitial haemorrhage surrounding thin walled blood vessels. (Fig. 6 & 7.)

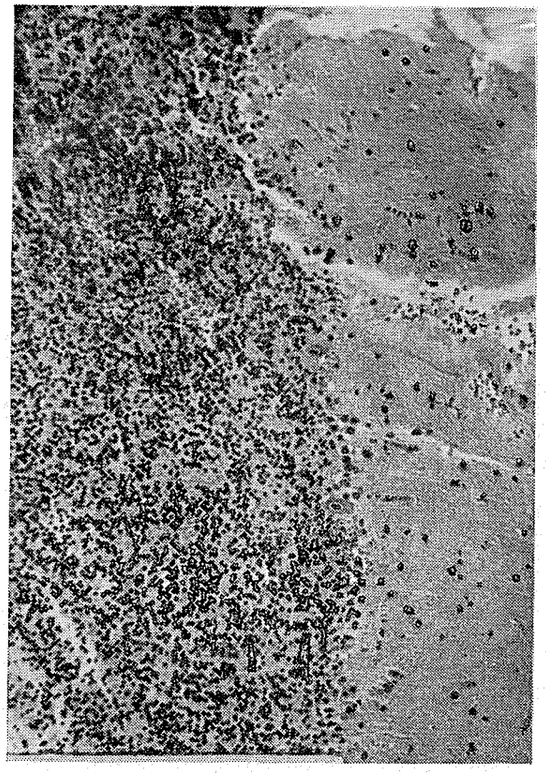

Fig. 7. Histological appearance of cerebellar tissue at the periphery of haematoma.

Case 14.

An eight year old boy was admitted to Pahlavi Hopital in May 1957, three days after an open depressed fracture of the left parietal bone caused by a brick falling on his head. He had not lost consciousness but apparently the right arm and leg had become paralysed. He had been admitted to a local hospital where his wound was excised and sutured. As the patient was becoming drowsy he was transferred to the Neurosurgical Department of Pahlavi Hospital.

On examination he was drowsy but co-operative. There was slight dysphasia. A right hemiplegia was present.

Straight X-ray of the skull showed a depressed fracture of the left parietal bone.

On the next day he was found to be more drowsy. The wound was reopened and the depressed pieces of bone were removed and the opening enlarged.

A small dural tear was present. The dural tear was excised and further opened to expose a bruised cortex under high pressure.

A small opening in the cortex revealed a fairly large subcortical haematoma. This was completely removed and the bleeding points were coagulated.

Post-operative progress was satisfactory, but his hemiplegia although improved slowly, it did not clear up completely.

Case 15.

A man of 22 was admitted to Pahlavi Hospital in June 1957; five days after an open fracture of the skull. He had been admitted to another hospital where his wound had been treated by excision of the scalp and removal of depressed fragments.

There had been no loss of consciousness. Three days later he began complaining of headache; and weakness of the limbs on the right side. On admission to Pahlavi Hospital he was alert and co-operative. There was a sight hemiparesis. The hand was severely affected. A carotid angiogram showed a mass in the left parietal region. (Fig. 8.)

At operation a large solid subcortical haematoma was removed. Post-operatively patient made rapid and ultimately complete recovery.

DISCUSSION

Of the 15 cases reported above 13 were males and two females. The number is too small for generalisation, but it may indicate the grea-

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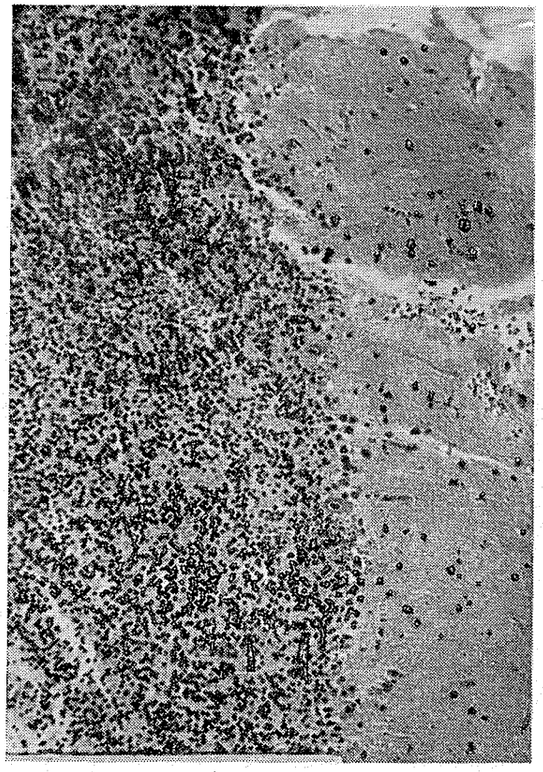

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ter liability of male to injury. If we exclude the two cases suffering from arterial hypertension the others were in the young age group (6-35).

This can partly be explained by the factor of vascular malformations present in four of the cases. As Dandy (1928) has remarked, the intracerebral hemorrhage occuring in youth or middle age is usually due to presence of a tumour or vascular abnormality. It seems that this is even true in the post-traumatic group.

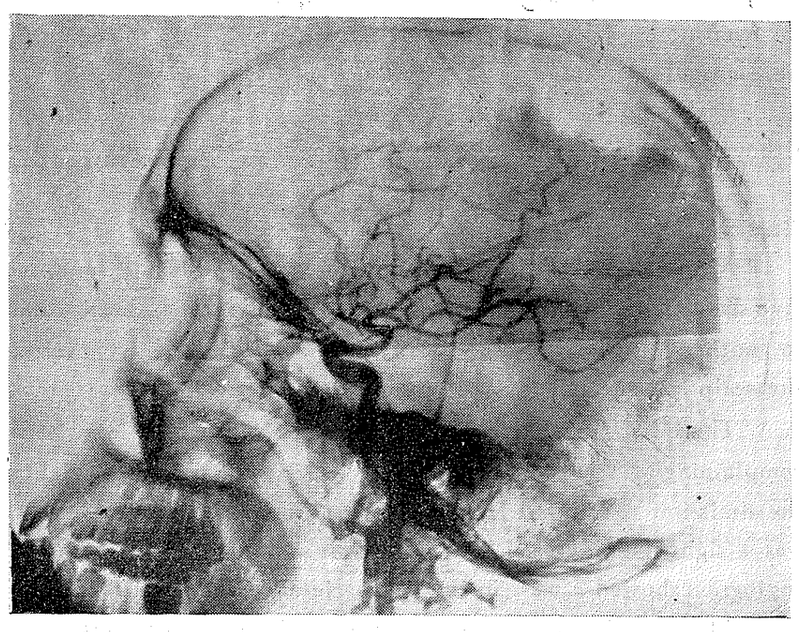

Fig. 8. Displacement of the posterior part of Sylvian vessels suggestion of a mass in the parietal region, under the bone defect.

The injuries in all the 7 cases that had a predisposing factor were slight. It is possible that in many cases described in the past as spontaneous haemorrhage slight trauma had been present, but either had been forgotten by the patient or the patient never recovered conciousness to recall the slight injury which probably had not been noticed by others.

In case 4 if the patient had not recovered it could have been classified as spontaneous.

In 4 cases the predisposing factor was a small angioma. This is the most interesting group. In many cases of spontaneous haematoma this condition has been found. Hawkins & Rewell (1946) Margolis et al (1951) Crawford & Russsel (1956).

There is no doubt that many cases are missed not only by clinicians but also by pathologists. Case 2 is an example. If the brain and the intracerebral clot had not been carefully examined the angioma would have been missed. Angiography is a valuable aid in diagnosis, but even this may be misleading. In case 12 the first angiogram taken soon after the haemorrhage was not very conclusive, but in the second angiogram taken six months later the angioma is clearly seen. The haematoma caused collaspe of the vessels and they did not show up in the first angiogram.

Presence of a congenital vascular malformation should be suspected if in a young person intracerebral haemorrhage develops after a slight head injury.

Cerebral Tumour.

According to Oldberg (1933) who analysed 832 cases of cerebral tumours in only 3.7% of the cases gross haemorrhage was seen, and in only 0.84% of the cases the symptoms were directly attributable to haemorrhage.

Post-traumatic haemorrhage in symptomless tumour (case 6) is rare. I have seen one other case in a post-mortem examination of a soldier in Tehran in 1938. After a fist fight in a camp he lost consciousness and died soon afterwards. At post-mortem there was extensive haemorrhage from a parasagital meningioma.

Trauma.

In cases of closed head injury, the early delayed haematoma is usually found in the centre of the temporal or frontal lobes (Courville & Blomquist 1940). The haemorrhage may be directly beneath the point of injury, in which case there are signs of brain contusion (Case 7), or it may be on the opposite side, in the centre of the lobe without any evidence of surface lesion : *contre coup*.

In penetrating wounds of the cranium intracerebral haematoma is not rare. Schorstein (1947) found 87 intracerebral haematomata in 316 cases of penetrating wound of the skull (23%). He had also observed that in 75% of the cases when there had been a small wound of entry, intracerebral haematoma had occured.

Case 2 is of interest that although the missile had passed from the frontal to the occipital lobe haematoma was produced near the site of entry. In case 9 there was no true penetration of the knife into the brain substance, but it was the broken piece of the inner table which had been pushed into the frontal lobe.

Arterial hypertension.

In both cases described (5 & 8) the injury had been slight and they did not lose consciousness for some hours after the injury. In these cases it is difficult to say what role the trauma actually played in production of haemorrhage. One can only presume that it had caused some weakness in vascular wall, and the hypertension did the rest. It can be argued that the emotion due to injury would be sufficient cause for cerebral hemorrhage in a hypertensive patient.

PATHOLOGY

On exposure of the surface, if the haematoma is subcortical a slight green discouloration is visible (Penfield 1936). In some cases (3, 4 & 7) the cavity contained fluid blood whilst in others solid blood

clot. There is no doubt that in the first group some clot formation had been present, and in those that recovered was ultimately absorbed. Browder & Turney (1942) found clotted blood in the brains of patients dying a few hours after injury, but 12-15 hours later the clot was syrupy.

The cause of raised intracranial pressure may be due to continued bleeding into the brain substance as in case 9 where solid haematoma was found 5 days after the injury, or as in case 4 may be due to break down of its molliculear content and rise of osmotic pressure giving rise to further increase in size by absorption of fluid. (Rowbotham & Ogilvie 1945).

There may occasionally be a natural recovery. According to Buzzard & Greenfield (1921) the final result may be either a large cavity containing a slightly blood stained fluid or linear scar. Calcification of the haematoma or its wall may develop (Pilcher 1941).

DIAGNOSIS

Many cases of intracerebral haematoma are diagnosed as extradural haematoma. Lucid or latent interval is so characteristic of an extradural haematoma that the mistake is understandable. There are few points that may help in differential diagnosis. In intracerebral haematoma the usual pupillary changes seen in the extradural type are often absent. The patient's condition may slightly fluctuate, unlike that of extradural haematoma which steadily deteriorates. The most important aid in diagnosis is angiography. (Webser et al (1951) Rousseaux et al (1952) Ameli 1956).

The angiography not only shows the site of the haematoma but may show a vascular abnormality as a predisposing cause. This safe procedure should not be withheld in the diagnosis and treatment of such a dangerous condition.

Case	Sex	Age	Latent interval	Nature of injury	Predisposing factor	Treatment	Results
1	M.	32	11 days	slight	?	Exploratory burr-holes	Died
2	F.	10	12–14 hours	slight	Angioma	——	Died
3	M.	10	2 days	slight	Angioma	Burr-hole & aspiration	Recovered
4	M.	18	7 days	fairly severe	——	Aspiration	Recovered
5	M.	55	2 hours	slight	Arterial hypertension	——	Died
6	M.	17	8 days	slight	Cerebral tumour	Aspiration & removal of tumour	Recovered
7	M.	32	3 days	severe	?	Aspiration	Died
8	M.	65	4 hours	? slight	Arterial hypertension	Exploration	Died
9	M.	25	5 days	penetrating wound	——	Removal of clot	Recovered
10	M.	35	3 days	severe	——	Removal of clot	Recovered
11	F.	6	7 days	penetrating wound	——	Removal of clot	Recovered
12	M.	14	3 hours	slight	Angioma	Repeated L.P. Decompression	Recovered
13	M.	25	one month	slight	Angioma	Later Removal of the clot	Recovered
14	M.	8	3 days	Compound fracture	——	Removal of clot	Recovered
15	M.	22	5 days	Compound fracture	——	Removal of clot	Recovered

TREATMENT

One cannot do better than quote Harvey Cushing (1903) on this subject. «I do not see any reason why we should exclude these cases from possibilities of surgical relief simply because the hemorrhage lies beneath the cortex, any more than that intracranial hemorrhage in other situations should be allowed to run its course.»

Penfield (1933), Craig & Adson (1936), Hanby (1935), David & Hécan (1954) Werner (1954) and other have reported cases of intracerebral haematoma successfully treated by surgical intervention. Three procedures are possible.

1- Making a burr-hole over the haematoma, and aspiration by insertion of a canula.

2- Decompression, and then two weeks later when liquefaction of the clot is completed, aspiration.

3- Full craniotomy with evacuation of the clot. In the cases reported in this paper all three procedures have been practised.

1. Cases 3, 4 & 6 were successfully treated by simple aspiration. In case 7 aspiration was temporarily effective but patient died few days later. It is possible that the patient could have been saved if the clot had been completely evacuated.

2. Case 13 was treated by decompression. Second operation was performed two months after the accident and still there was no liquefaction. Whether the removal of the haematoma at the first operation would have been successful is debatable.

3. In cases 9, 10, 11 & 13 haematoma was completely evacuated and with excellent results.

In patients with rapidly increasing intracranial pressure occurring within few days of injury complete evacuation of haematoma should be attempted. In cases where the severe symptoms develop two to three weeks after an injury simple aspiration may be sufficient. But in any case that aspiration has been practised, if the symptoms and signs do not disappear at once, (return of consciousness, hemiparesis aphasia, etc.) then

it is likely that a large clot is present, and one should do a full operation, preferably after a second angiography.

SUMMARY

15 cases of delayed post-traumatic haemorrhage of the brain are described. The lucid interval varied from 2 hours to one month. In at least seven cases a predisposing factor had been present. The importance of small angioma as a factor in young people has been emphasized. Clinical and pathological aspects and treatment of the condition have been discussed.

RÉSUMÉ

15 cas d'hémorragie cérébrale post-traumatique tardive sont rapportés. L'intervalle libre de ces cas ont été entre 2 heures jusqu'à un mois. Dans 7 cas, l'auteur a trouvé un facteur prédisposant. L'importance de l'angiome de petite taille pourra être considérée chez les jeunes sujets.

L'auteur a discuté l'aspect pathologique et le traitement de l'hémorragie cérébrale post-traumatique.

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