SPONTANEOUS SUBARACHNOID HAEMORRHAGE OF INTRASPINAL ORIGIN

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LEEDS

The clinical picture of spontaneous subarachnoid haemorrhage of cerebral origin is now definitely established, and cases occur with moderate frequency. The onset is usually sudden, generally with loss of consciousness, which passes off gradually. There usually follows severe headache—often in the occipital region—recurrent vomiting, and more or less confusion. As a rule there is no paralysis and no sensory changes, though such may occasionally be found. The fundi may show retinal haemorrhage, or there may be only a papillitis. On lumbar puncture the cerebro-spinal fluid is usually found under slightly increased pressure, and contains blood. The blood is intimately mixed with the cerebro-spinal fluid, thus differentiating the condition from the trauma of lumbar puncture. The causation is usually ascribed to a leak or burst of a miliary aneurysm on one of the superficial cerebral arteries. Though the condition is fatal in a considerable number of cases, on the whole the recoveries more than outnumber the deaths. If the patient survives there are, as a rule, no sequelae.

Spontaneous subarachnoid haemorrhage of intraspinal origin is a much rarer condition, and hence the justification for publishing the clinical and laboratory notes of the following case.

CASE HISTORY

A married man, aged 40, consulted one of us (H. D.-W.) on October 4th, 1932, because of feeling "off-colour" for the previous few days. He gave a history of marked constipation—an unusual feature for him—slight headache, and malaise. No abdominal tenderness. Sent to bed, given bowel wash-outs.

On October 6th the condition was worse, and he was admitted to the Harrogate Investigation Clinic. Tongue dirty, breath foul, slight abdominal tenderness, knee-jerks and abdominal reflexes present. Temperature 100.6° F., vague pains in back. Blood culture negative, blood count showed a very slight leucocytosis, blood urea normal, Wassermann reaction negative, moderate albuminuria, urine sterile on culture. Six days after admission to the clinic the blood gave a positive agglutination to typhoid (1 in 250 dilution). The spleen was now palpable, abdomen doughy, and there were a few rose spots. The condition was now considered to be due to a typhoid infection, and the patient was treated accordingly. The pains in the lumbar region became progressively worse in the course of the next few days, being almost girdle-like in type. Rigidity of the spine appeared. Blood pressure now became definitely raised: systolic 155 mm., diastolic 105 mm. Pulse rate 60.

October 16th.—The pains in the back were so severe and so persistent that it was thought that some other causative factor must be present. Fundi were still negative. Knee-jerks had gone, but ankle-jerks were active. Plantar response flexor, superficial abdominal reflexes absent; except in right lower quadrant. No sensory changes. Bowels very obstinate. Lumbar puncture was considered.

October 20th.—Seen for the first time by G. W. W. Blood pressure 160/110. No fundal changes; central nervous system as before. Report of lumbar puncture (S. M.): no increase of pressure, blood present, 7,000 red cells per c.mm., white cells 240 per c.mm., supernatant fluid distinctly yellow, Wassermann reaction negative, Lange sol curve almost normal,

sterile on culture. After lumbar puncture the temperature fell and the general condition of the patient improved.

October 23rd.—Patient much as before, abdominal reflexes returned. Knee-jerks could just be elicited.

October 24th.—Relapse. Patient vomited. Return of severe pain in lumbar region. Spinal puncture repeated. Slight increase of pressure, fluid redder than previously, 43,000 red blood cells per c.mm. Obviously a fresh haemorrhage had occurred. Patient much more comfortable after lumbar puncture.

October 25th.—Return of vomiting. Patient seen in consultation by Dr. James Collier and G. W. W. Fundi examined under atropine—no ocular haemorrhage, no fundal changes. Lumbar puncture repeated. Pressure markedly increased, red blood cells 45,000 per c.mm. Supernatant fluid after centrifugalization was bright yellow in colour, and gave a very faint haemoglobin spectrum.

October 26th.—Condition more satisfactory.

October 27th.—Return of severe pain in back. Lumbar puncture repeated. Fluid as before. For the next five days following this the condition slightly improved.

November 2nd.—Patient had disturbed night, recurrence of severe pain in back, vomiting, abdomen tense.

November 3rd and 4th.—Slight improvement. Pains now extending to quadriceps extensors.

November 5th.—Rigidity in the back much more marked, nausea and vomiting, lumbar puncture—increase of blood in cerebro-spinal fluid, 86,000 red blood cells per c.mm. Pressure much increased.

November 6th to 9th.—Condition unsatisfactory. Patient not improving.

November 10th.—Disturbed night. Twitching of muscle bundles. Lumbar puncture. Put on calcium by the mouth and magnesium sulphate per rectum (G. W. W.).

November 11th to 16th.—Condition not satisfactory. Patient complained of generalized pains, neuralgic in type, all over the body.

November 17th.—Return of vomiting. Pain intense at times.

November 18th.—Patient worse, pains more severe. Hyperaesthesia marked. Lumbar puncture repeated, pressure very high. Fluid very red in colour, 140,000 red blood cells per c.mm. Severe lumbar pain immediately relieved by puncture.

November 19th.—Patient seen in consultation by Professor Arthur Hall and G. W. W.

November 20th and 21st.—Patient much easier.

November 22nd to 24th.—Slight return of vomiting. Blood pressure slightly increased (160/110).

November 25th.—Improvement definite. Blood pressure lower (145/98), but pulse rate increased (106).

November 26th to 30th.—Steady improvement. Appetite returning, blood pressure falling. From this date onwards the patient continued to improve steadily.

December 23rd.—Condition highly satisfactory, muscle tone markedly improved, general nutrition good, pulse 80, regular and of good quality. Blood pressure 120 mm. systolic, 80 mm. diastolic. No abnormality in heart. No evidence of paresis, no sensory disturbances. Superficial abdominal reflexes of normal activity, except in the left upper quadrant, where the reflex was elicited with difficulty. Knee-jerks absent, ankle-jerks active, plantar response flexor, no ankle-clonus. Patient can sit up without undue fatigue, appetite excellent, bowels regular.

December 24th.—Patient left the clinic convalescent.

DISCUSSION

The case described differs in many respects from a typical case of spontaneous subarachnoid haemorrhage of cerebral origin. Loss of consciousness and subsequent confusion were entirely absent. There were no ocular symptoms, neither paralysis nor photophobia, and no fundal changes. There was no suggestion of paralysis of any of the cranial nerves or other symptomatology denoting a lesion of the cerebral hemisphere.

The suggestion that the haemorrhage was intraspinal in origin is supported by several facts: (1) The almost complete absence of cerebral symptoms at onset. (2) The severity and localization of the pain in the lumbar region, the pain being excruciating at times and increased by movement. (3) Rigidity of spine, with a mild degree of opisthotonos. (4) Marked irritability and hyperaesthesia of the spinal roots and nerves. (5) The almost instantaneous relief of symptoms on lumbar puncture.

From whatever cause the haemorrhage arose there is no doubt that on four separate occasions bleeding of an extensive nature occurred, and though the illness was a protracted one, recovery appears to be complete and the sequelae insignificant. It is also of interest to note the close association of the fall of the blood pressure with improvement in the clinical condition.

SPONTANEOUS SUBARACHNOID HAEMORRHAGE

WITH REPORT OF EIGHT CASES

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Leakage of blood into the cerebral subarachnoid space apart from any trauma is being recognized clinically with increasing frequency. The blood escapes from an artery, perhaps sometimes from a vein, into the meshwork of trabeculae which intervenes between the pia mater and the arachnoid membrane: this occurs most often from a vessel lying at the base of the brain. Haemorrhage into the subarachnoid space of the spinal cord has been recorded,^{6,7} but is much rarer. Blood escaping from a vessel within the brain may find its way either through the nervous tissue or through the foramina in the fourth ventricle into the subarachnoid space, but this paper is not concerned with these secondary types.

The underlying pathological lesion falls under several heads, and individuals of all ages may be affected. "Arteriosclerosis," in the form of medial degeneration, associated with high blood pressure and cardiovascular hypertrophy, is probably a common cause of spontaneous subarachnoid haemorrhage at or past middle life. In cases occurring in young adults the blood pressure is usually not raised, and the heart and peripheral arteries generally appear normal. Deficiency of the media has, however, been found in the arteries at the base of the brain, and it has been suggested that this is a congenitally installed defect.¹⁴ Syphilitic arteritis, when the inflammation is severe, leading to necrosis, may end in haemorrhage; when less acute a thickening of the intima occurs and there is no possibility of rupture. Weakening of the arterial wall by an embolus in infective endocarditis may occasionally result in rupture. It is possible that a vessel may give way as a late effect of cerebral embolism when the embolus is sterile; this is indicated by Case II below. Other known causes are: cerebral angioma, blood diseases, acute haemorrhagic infections, sunstroke, eclampsia, haemorrhage from tumours and cysts, and thrombosis of the superior longitudinal sinus.

The clinical picture shows wide variations, depending on the severity and site of the bleeding. In the most severe cases there is a sudden loss of consciousness and such compression of the brain that death supervenes within a few hours. In the mildest cases there will be headache, vomiting, and some pyrexia, followed by gradual recovery. Between these two all intermediate forms occur. The following is an example of a rapidly fatal haemorrhage.

CASE I

A man of 58 was admitted to the London Hospital under Dr. Robert Hutchison on April 22nd, 1929. He had been taken into the hospital seven weeks previously because of severe headaches beginning in the occipital and extending to the frontal region, each lasting about twenty-four hours and recurring weekly. Examination then showed subacute glaucoma of the left eye. The blood pressure was normal, and there was only slight arteriosclerosis.

On April 21st, 1929, the patient had several attacks of faintness of a few minutes' duration. Next day he became completely unconscious, and was admitted to hospital. The temperature was 99° and the pulse rate 60. On the left side the tendon reflexes were brisker, the plantar response was extensor, and there was ptosis of the left eyelid. Consciousness was temporarily recovered at 11 p.m., when he complained of headache. During the night there were three fits, and death occurred at 6.15 a.m. on April 23rd. The blood Wassermann reaction was negative.

Post-mortem Findings

The following is an extract from the records of the Bernhard Baron Institute of Pathology, by the kind permission of Professor H. M. Turnbull.

"There was a collapsed ruptured aneurysm, 1.2 cm. long and 0.7 cm. broad, at the junction of the left carotid, middle cerebral, and posterior communicating arteries. The aneurysm was covered by thin layers of adherent clot, and lay in a haemorrhagic depression in the left uncus, from which a haemorrhagic track led into the left lateral ventricle. The left lateral ventricle contained 4 to 5 drachms of clot and the right lateral and fourth ventricles a smaller amount. Subarachnoid haemorrhage was confined to the immediate neighbourhood of the aneurysm. The arachnoid membrane here was ruptured, and blood had passed into the subdural space. In this space some 6 to 7 drachms covered the left hemisphere, whilst less lay over the right hemisphere and still less in the posterior fossa; a layer of blood 1 mm. thick filled the subdural space of both optic nerves. There was rusty brown pigmentation of the dura in the posterior fossa and of the pia-arachnoid upon the inferior surface of both cerebral hemispheres, being greatest upon the left. An unruptured aneurysm, 1 mm. in diameter, projected from the junction of the right carotid and middle cerebral arteries, with an accessory posterior communicating artery. Microscopic examination of the aneurysm and adjacent arteries showed that the media and internal elastic lamina ended abruptly at the mouths of the aneurysms, whilst the walls of the aneurysms were formed by adventitia lined with a variable thickness of tissue continuous with the intima. The media of the right middle cerebral artery showed at some distance from the aneurysm patches in which muscle fibres were almost entirely absent. There was no evidence of arterial inflammation or degeneration. The aneurysms were therefore considered to be due to rupture of the intima and media at sites of congenital malformation of the media."

A haemorrhage causing initial coma may abate or cease and consciousness be subsequently regained. Symptoms of meningeal irritation, of general cerebral compression, and of local cerebral irritation or paralysis will then probably be observed; these symptoms persist for a variable period; a fresh leakage of blood may suddenly, or perhaps gradually, lead to a fatal conclusion. The following three patients illustrate this type.

CASE II

A man of 38 suddenly fell unconscious on March 17th, 1926, and was admitted in a stuporose condition to the Middlesex Hospital under Dr. E. A. Cockayne. He had had acute rheumatic carditis in 1912, and in 1924 a cerebral embolus causing left hemiplegia. On admission he was mentally confused and disorientated, complained of headache, and was constantly putting his hand to his head. The pupils were unequal, but the cranial nerves were otherwise normal. There was neck rigidity, and Kernig's sign was