The Central African Journal of Medicine

Editor:
MICHAEL GELFAND, C.B.E., M.D., F.R.C.P.

Assistant Editor:
JOSEPH RITCHKEN, M.D.

Volume Nineteen

JANUARY - DECEMBER

1973

SALISBURY : RHODESIA

The Central African
Journal of Medicine

Volume 19 DECEMBER, 1973 No. 12

A Study of Subdural Haematoma in an African Medical Ward

BY

LESLIE LEVIN, M.B. (Birm.), M.R.C.P.
AND
MICHAEL GELFAND, M.D., F.R.C.P.

Introduction

Most cases of subdural haematoma in the wards are chronic in nature and its diagnosis may be difficult. A subdural haematoma can be regarded as chronic if the haematoma has a membrane around it or is operated on more than 10 days after the receipt of the trauma. The typical features one would expect such as ipsilateral pupillary dilatation (caused by compression of the third cranial nerve) and contralateral hemiplegia (caused by compression of the crus cerebri against the falx) are not commonly encountered.

We present a study of 11 cases of chronic subdural haematomas presenting to the University medical ward in Harare Hospital, Salisbury, between 1968 and 1971.

Methods and Materials

In the examination a special note was taken of the age, sex, symptoms, history of trauma, level of consciousness on admission, pulse, blood pressure, pupillary changes, evidence of localising signs, fundoscopy and evidence of liver disease. In addition the laboratory investigations included a blood VDRL, liver function tests, prothrombin index and cerebro-spinal fluid changes (pressure, biochemistry, VDRL and microscopy).

The result of carotid angiography and operative or post mortem findings were recorded; an electroencephalogram was included when this examination was carried out.

Results

Age and sex:

The mean age of patients was 50 years. The age distribution is broken down in the table below.

Table I

Age Group of Patients Presenting with Chronic Subdural Haematoma

Age Group (Years)	Number	%
0-30	0	0
30-39	2	18
40-49	4	36
50-59	1	0
>60	4	36

There were nine males and two females.

History of Trauma

In five cases there was either a history of trauma, or evidence of trauma was found at autopsy. In three cases there was no history of trauma, and in the other three cases the question relating to history of trauma was not recorded in the notes.

Initial Symptoms and Signs.

A. *State of Consciousness on Admission.*

The state of consciousness was assessed according to the changes in behaviour (mental changes), those who were stuporose (drowsy but rousable) and comatose (unable to be roused). The results are shown in Table II.

Table II

State of Consciousness on Admission

State of Consciousness	No. of Cases	%
1. Alert	—	—
2. Mental changes	5	45
3. Stuporose	4	40
4. Comatose	2	15

One patient presented in a catatonic state, whilst another who was unconscious on admission to the medical ward, was referred from the psychiatric unit, where he had presented three days previously and was diagnosed as having a depressive affective disorder.

B. *Ocular Manifestations.*

Only pupil size and examination of fundi were recorded.

Table III

OCULAR MANIFESTATIONS IN CHRONIC SUBDURAL HAEMATOMA

	No. of Cases	%
1. *Pupillary Inequality*		
a. Ipsilateral pupil dilated	—	—
b. Contralateral pupil dilated	2	18
c. Both pupils dilated	1	9
d. Equal and reacting	8	72
2. *Papilloedmea*	1	9

One patient with unilateral pupillary change had an irregular outline of the pupil and this simulated an Argyll-Robinson pupil. Of note is that in both cases with unilateral pupil dilation the haematomas were on the opposite side.

Table IV.

RELATING LOCALISING SIGNS TO PROVEN SITE OF THE HAEMATOMA

Nature of Sign	Pupillary Change	Site of Subdural Haema-toma
Spastic (L) Hemiplegia	(L) Pupil dilated	Right
Spastic (L) Hemiplegia	Both pupils dilated	Right
Spastic (L) Hemiplegia	Pupils equal reacting	Right
Decreased reflexes (L) arm	(L) pupil dilated	Right
Symmetrical shifting gait	Pupils equal reacting	Right
Spastic (R) hemiplegia	Pupils equal reacting	Right
Weakness (R) hand	Pupils equal reacting	Bilateral
No localising signs (4 cases)	Pupils equal reacting	Left (4 cases)

In four cases there were no localising signs and in all these the pupils were equal and reacting. There was one case of ipsilateral hemiplegia; besides this case the presence of hemiplegia was a valuable localising sign in the other five cases who presented in this way.

C. Seven patients complained of a headache two stated that they had not experienced a headache and in two information was not available on this point.

D. *Blood Pressure and Pulse*

The blood pressure and pulse in each case was recorded below and the cerebro-spinal fluid pressure included to relate intracranial pressure to these parameters.

Table V

TO RELATE BLOOD PRESSURE AND PULSE RATE TO CEREBROSPINAL FLUID PRESSURE

Blood Pressure (mm. Hg.) Systolic	Diastolic	Pulse Beats/minute.	CSF Pressu (mm. H_2O
150	100	45	150
130	90	60	120
130	80	80	120
120	80	80	260
150	90	80	—
180	90	64	190
60	—	100	Bilateral Papilloedem
140	90	80	—
120	80	70	310
110	80	90	130
110	70	100	250
Mean 127	77	77	190

Four of the above cases had raised CSF pressures (>180 cm H_2O). In only one of these cases was the blood pressure raised and an associated bradycardia present. In a fifth case, bilateral papilloedema was present although the blood pressure was 60/0 mm. Hg. and the pulse 100/minute. The only other case with a raised blood pressure (150/110 mm. Hg.) and bradycardia (45/minute) had a normal cerebro-spinal fluid pressure of 150 cm. H_2O.

LABORATORY INVESTIGATIONS

A. *Cerebro-spinal fluid.*

Table VI

TO SHOW CEREBROSPINAL FLUID FINDINGS IN EIGHT PATIENTS WITH CHRONIC SUBDURAL HAEMATOMAS

CSF (measured in 8 Cases)		No. of Cases	%
1. Xanthochromic		1	13
2. Clear		7	87
3. Protein	> 60 mg.	3	37
	< 60 mg.	5	63
4. Pressure	>200 cm.	4	50
	<200 cm.	4	50
5. VDRL positive		—	—

Of the three remaining cases, one patient had papilloedema so that a lumbar puncture was not done and the other two cases were diagnosed by carotid arteriography on admission.

B. *Blood*

1. VDRL

In eight cases in which this was examined, all were negative.

The cerebrospinal fluid pressure and examin-

ation was completely normal in four of the eight patients, in whom this was performed.

2. *Evidence of Abnormal Liver Function.*

(a) Prothrombin time/index: this was measured in three patients none of whom had evidence of trauma. The P.I. in two cases was 100 per cent. (P.T. 13 secs.); in the other case the P.I. was 70 per cent. (P.T. 20 secs.).

(b) Serum Proteins: there was albumin depletion with an inverted albumin/globulin ratio in one case; there was no history of trauma in this case and the P.I. was 70 per cent.

(c) Hepatomegaly was noted in two patients both of whom had normal serum proteins, PI/PT and liver function tests.

CAROTID ARTERIOGRAPHY

Confirmed the pressure of a space occupying esion in 10 cases.

The average duration between the diagnosis being made and confirmed was five days. One subdural haematoma was diagnosed at autopsy. There were 10 unilateral and 1 bilateral subdural haematomas.

TESTS

Electroencephalograms were done on two patients.

Case 1: E.E.G. — Normal — carotid arteriography revealed subdural haematomas.

Case 2: Diffuse mild abnormalities, maximal over the left motor parietal area. This patient was found to have a right subdural haematoma.

MORTALITY RATE

Four patients died, giving a mortality rate of 36 per cent. One patient died soon after admission and another died on the day of admission, after surgery. The third death had three separate operations for recurrent bleeding and eventually died two months after admission. The fourth case was diagnosed at autopsy — this patient had cirrhosis, pulmonary tuberculosis and typhoid. Of those patients who survived only one was left with a neurological deficit after being discharged.

The initial state of consciousness is related to the ultimate prognosis in the table below.

Table VII

INITIAL STATE OF CONSCIOUSNESS RELATED TO ULTIMATE PROGNOSIS.

Initial State of Consciousness	Survived	Died	% Mortality
Drowsy	3	1	25
Mental changes	3	2	40
Unconscious	1	1	50

DISCUSSION

The male:female ratio of 9:2 is not surprising when one considers trauma to be one of the main aetiological factors in this condition.

In three cases it was clearly established that there was no history of trauma. In trying to establish a possible cause for this we considered bleeding disorders secondary to abnormal liver function, as liver disease (cirrhosis and hepatoma) is so common in our African patients. Three patients in this series had evidence of cirrhosis and in two there was no history of trauma; only one, however, had a prolonged prothrombin time. Abnormal clotting should be considered as an underlying factor predisposing to subdural haemorrhage in patients with impaired liver function. It is of interest to note that Levy *et al.* (1957) considered prothrombin deficiency in premature infants to be a possible predisposing cause to subdural haematoma in infancy.

Examination of the cerebrospinal fluid was abnormal in only four of the eight patients in whom this was performed. It is important to stress that a normal cerebrospinal fluid does not exclude the possibility of subdural haematoma.

Other causes of subdural haematoma such as thrombocytopenia, leukaemia, metastatic carcinoma of the dura and anticoagulants were not encountered in this series. Of particular note is that subdural haematoma is now recognised as a complication of maintenance renal dialysis and in the treatment of acute renal failure with dialysis (Leonard, 1970); we saw no such case in this series.

Denial of a history of trauma does not necessarily exclude the possibility that the patient sustained trauma. The injury may have been trivial, resulting in a small haematoma with organisation and absorption of CSF, giving rise to a progressively large lesion. Tearing of the subdural vessels may also result from falling onto the feet or buttocks (Montgomery, 1965) and the patient may not consider this relevant when giving a history.

None of the patients was alert on admission. Forty-five per cent. of patients had mental changes. Forty per cent. of the patients were stuporose on admission but these patients as well as those with mental changes tended to fluctuate in their level of consciousness. This compares with the findings of Patterson *et al.* (1968) in their series of 10 chronic subdural haematomas in which they found mental changes in 60 per cent. of their patients on admission.

Pohl and Haits (1967) emphasise that acute forms of symptomatic psychoses may occur on

the basis of chronic subdural haematomas with or without disturbances of conscience. The initial state of consciousness on arrival at hospital seems to bear a relationship to the ultimate prognosis; the more severe the change in level of consciousness, the worse the ultimate prognosis appears to be.

Seventy-two per cent. of the patients had no change in pupil size; once again this compares with Patterson *et al.* (1968) who discovered no change in 70 per cent. of their patients. In both cases in this series with unilateral pupil dilatation, the dilated pupil was on the opposite side to the haematoma.

Other localising signs were present in 64 per cent. of the patients; there was one case of hemiplegia on the same side as the haematoma.

Probst (1968) recently stressed that vascular disturbances, oedema, disturbed CSF circulation and mass displacement account for symptoms and signs in subdural haematomas. For this reason, the typical signs one would expect may be somewhat distorted.

It is obvious from this that proper neuroradiological investigation is indicated to localise the size of the lesion.

Raised intracranial pressure usually results in a raised blood pressure and slow pulse. Intracranial pressure exerted upon the freely collapsible capillaries of the brain opposes the flow of blood and this accounts for the raised blood pressure; however, the cerebral blood flow does not diminish until the intracranial pressure reaches about 450 mm. water (Best and Taylor) and this would explain the occurrence of very few hypertensive cases in our series. The absorption of fluid from the brain into the hypotonic liquified chronic subdural haematoma could compensate for the increased pressure resulting from space occupying lesion itself; this might account for the relatively low cerebrospinal fluid pressures of patients in this series.

Taking the upper limit of normal CSF pressure as 180 mm. water, five of the patients in this series had a raised cerebrospinal fluid pressure; of these one was assumed to have a raised pressure because he had bilateral papilloedema. However, a raised blood pressure and slow pulse was only noted in one of these patients.

It seems, therefore, that observations on blood pressure in these patients can only be used if there is a sudden bleed into the maematoma but it would reflect the severity of the haematoma otherwise.

Carotid arteriography still offers the most accurate recognition of subdural haematoma. Because of the difficulty of being sure of the diagnosis in many sases, an urgent arteriogram is often not requested with the consequent dangers of a later diagnosis.

It would appear that the electroencephalogram is of little assistance in diagnosing this condition. This concurs with the findings of Levy *et al.* (1957) in their study of subdural haematomas in infancy.

A quick, cheap and fairly reliable method of diagnosing this condition is with the use of ultrasonic devices. Variable success is quoted in the literature using ultrasonic devices, and correct diagnosis varies from 50-90 per cent. in different series. Rothman *et al.* using a new procedure which does not rely entirely upon mid-line shift claim great sensitivity and the ability to measure the depth of the haematoma. This is an extremely valuable screening procedure; the suggestion of a haematoma with this device should at least prompt more urgent investigations. The device could also be used in psychiatric units.

SUMMARY

A survey of 11 subdural haematomas is presented. In three cases it was clearly established that there was no evidence of trauma; in two of these cases an enlarged firm liver was present, and in one of them the prothrombin time was prolonged. We suggest that abnormal clotting, resulting especially from liver disease which is so common amongst Rhodesian Africans may cause or contribute to the formation of subdural haematomas.

The physical signs in chronic subdural haematomas are inconstant and patients with it usually present with changing levels of consciousness with little or no signs. Pupil size, pulse and blood pressure seem to be of little value in leading towards a diagnosis or assessment of the severity of the underlying haematoma and examination of the cerebrospinal fluid has limited value.

REFERENCES

BEST, C. H. AND TAYLOR, N. B. (1966) The Physiological Basis of Medical Practice. Williams and Wilkens.

LEONARD, C. D. (1970). *New Eng. J. Med.* **282** 1433.

LEVY, L. F., HOEN, T. I., COOPER, I. S. (1957) *C. Af. J. Med.* **3,** 435.

MONTGOMORY, C. D. (1965) Textbook of Pathology. E. and S. Livingstone, Ltd.

PATTERSON, J. F., MOSTELLAR, H. J., WALKER JR. H. J. (1968) *Southern Med. J.* **61,** 52.

POHL, O., HAITS, G. (1967) *al Deutsch Gesundh.* **22,** 1749.

PROBST, C. (1968) *Schweiz Med. Wschr.* **98,** 1793.

ROTHMAN, J., SHATSKY, S., KRICHEFF, I., CHASE, N. (1969) *Amer. J. Roentgen.* **105,** 413.