ACUTE SPONTANEOUS ARTERIAL SUBDURAL HAEMATOMA
A Case report and review of the literature

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SUMMARY :

Acute spontaneous subdural haematoma of arterial origin is very are. We report such a case who presented with sudden severe headache and vomiting rapidly becoming comatose. The patient was hypertensive but had no history of head trauma, and the source of bleeding was identified as a cortical artery located near the sylvian region. Comparable cases in the literature are reviawed and the aetiological and pathophysiological possibilities are discussed.

KEY WORDS :

Arterial subdural haematoma, spontaneous haematoma.

INTRODUCTION

Acute spontaneous subdural haematoma is an entity that has been reported infrequently and has received little attention. We report an additional case with a review of the literature.

CASE REPORT

A 70-year-old-right-handed man developed a sudden severe headache, worse on the right side, while resting at home. The pain was sharp in character and was associated with episodes of vomiting. Within three hours be gradually became less responsive and developed mild weakness on his left side. There was no history of prior or recent head trauma and no history of smoking or alcohol intake. He was hypertensive for years, otherwise had had no serious past illnesses and his healty had been good.

One examination four hours after the onset of headache, he was unresponsive to verbal stimuli and densely hemiparetic on the left side, and withdrew the right limbs to painful stimuli. His right pupil was 4 mm and sluggishly reactive. The blood pressure was 280/120 mm Hg. The rest of the physical and neurological examination was unremarkable.

A CT scan revealed a large acute subdural haematoma over the right cerebral hemisphere (fronto temporo parietal region). The haematoma was 2.5 cm thick, and there was a-1.5 cm shift of midline structures (Fig 1).

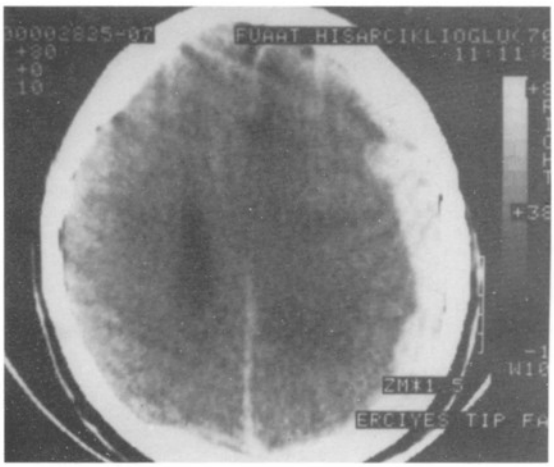

Fig 1 : CT scanning shows an acute right subdural haematoma.

The patient's neurological examination and vital signs were indicative of progressive herniation. Therefore, he was immediately taken to the operating room, where he underwent a craniotomy of the right fronto-temporal region. A thick subdural clot was removed. The source of the bleeding was identified as a small cortical artery in the region of the Sylvian fissure. As the clot was lifted off active arterial bleeding ensued suggesting that his artery had been the site of the initial haemorrhage. The bleeding point was arrested using bipolar electrocoagulation. There was no evidence of subarachnoid haemorrhage, no aneurysm or arteriovenous malformation around the affected artery, and the underlying brain appeared normal.

The patient became concious a short time after operation and made an unremarkable recovery. He was discharged five days after admission in excellent condition. Repeated postoperative loboratory studies were all within normal limits. No angiogram was considered.

DISCUSSION

Nontraumatic or spontaneous acute subdural haematoma of arterial origin is a rare and little recognized entity. Which should fulfil the following criteria (15,17,18): no history of head trauma; no damage to the underlying cortex; no aneurysm or arteriovenous malformation around the affected artery; and identification of the haemorrhage as being arterial at operation. Our case fulfils the above criteria. As far as we have been able to ascertain, there are 21 cases reported in the literature which meet these criteria (1,3,8,11,17,18,21). Patients whose point of bleeding had not been verified at operation or who had a history of even trivial head trauma have been excluded.

Table 1a. Clinical Summarry of Cases

No	Author Year	Age Sex	Past history	Initial semptoms	Conciousness	Pupil	Weakness
1	Talala and Mckissock, 1971	61 m	Bronchitis	Headache, loss of consciousness	Drowsy	R>L	R
2	Talala and Mckissock, 1991	54 M	Hypertension	Sudden headache	Drowsy	R>L	R
3	Talala and Mckissock, 1991	57 M	–	Sudden headache	Drowsy	R>L	R
4	Talala and Mckissock, 1971	66 F	Hypertension	Headache.distur bance of consciousnes	Deeply unconscius	R=L (dilated)	R
5	Talala and Mckissock, 1991	54 M	Hypertension alcohol abuse	Sudden headache	Drowsy	R=L	L
6	Talala and Mckissock, 1991	59 M	Seizure	Sudden headache	Drowsy	R>L	R
7	O Brien et al 1974	61 M	Polycythemia vera	Headache, vomiting	Stupor	R=L	–
8	O Brien et al 1974	57 M	Old head injury (2 yrs, before)	Headache	Confusion	?	L
9	Byun and Patel 1979	37 F	Seizure	Left hemiparesis	Alert	R=L	L
10	Byun and Patel 1929	37 F	Seizure	Headache, vomiting	Alert	R=L	–
11	Yamanake et al, 1982	48 M	Hypertension Lung tuberculosis	Sudden headache	Coma	R=L	L
12	Hasegave et al, 1982	60 M	Hypertension arteriosclerosis	Headache, vomiting	Drovsy	R<L	R
13	Hasegava et al, 1982	80 F	Hypertension arteriosclerosis	Sudden dysphasia	Semicoma	R=L	R
14	Aral, 1983	50 F	Hypertension	Sudden headache	Alert	R=L	–
15	McDermott el al, 1984	53 M	Old head injury (26 yrs, before)	Sudden headache, vomiting	Coma	R=L (dilated)	L
16	McDermott et al, 1984	55 M	Alcohol abuse	Sudden headache, vomiting	Coma	R=L (dilated)	Decortice
17	McDermott et al, 1984	45 F	–	Sudden headache	Alert	R=L	R
18	McDermott el al, 1984	65 M	Hypertension	Sudden headache	Alert	R=L	R
19	Yanai et al, 1985	40 M	Liver cirrhosis	Sudden vomiting	Coma	R<L	Decerebrate
20	Tokoro et al, 1988	53 M	Alcohol abuse	Sudden headache	Coma	R>L	R
21	Tokoro et al, 1988	45 M	Hypertension alcohol abuse	Headache	Coma	R=L (dilated)	Decerebrate
22	Paşaoğlu et al, 1990	70 M	Hypertension	Sudden headache, vomitng	Coma	R>L	R

M : male, F : female, R : right, L : left

Table 1b. Neuroradiologycal and operative findings, outcome of cases

No	Angiography	CT	Operation, hematoma	Bleeding point	Interval onset treatment	Treatment	Outcome
1	Mass sign	–	R. clot	Temporal artery	4 days	Cr	D
2	Mass sign	–	R. clot (membrane)	Lateral artery	5 days	Cr	D
3	Mass sign	–	R. clot	Sylvian artery	4 days	Cr	D
4	Mass sign	–	R. clot	Motor artery	2 weeks	Cr	D
5	Mass sign	–	L. clot	Lateral artery	10 days	Cr	G
6	Mass sign	–	R. clot	Parietal artery	5 weeks	Cr	D
7	Mass sign	–	R. clot	Middle cerebral artery	>1 day	Cr	Alive
8	Mass sign	–	L. clot	Middle cerebral artery	2 weeks	Cr	Alive
9	Mass sign	–	R. clot (membrane)	Cortical artery	2 months?	Cr	G
10	Mass sign	–	R. clot?	Sylvian artery	8 days	Cr	G
11	Normal	+	L. clot	Central temporo occipital arteries	>1 day	Cr	G
12	Extravasation	+	L. clot	Temporal artery	7 days	Cr	G
13	Mass sign	+	L. clot (membrane)	Angular artery	17 days	Cr	D
14	Eptravasation	+	R. clot	Central artery	5-8 hours	Cr	G
15	Normal	+	L. clot	Supramarginal artery	8 hours	Cr	G
16	–	+	L. clot	Frontal opercular artery	7 hours	Cr	G
17	Mass sign	+	L. clot	Sylvian artery	12 days	Cr	G
18	Mass sign	+	L. clot	Temporal artery	8 days	Cr	G
19	–	+	L. clot	Temporal artery	11 hours	Cr	D
20	Mass sign	+	R. clot	Temporal artery	7 hours	Cr	MD
21	–	+	R. clot	Temporal artery	2 days	Cr	D
22	–	+	R. clot	Cortical artery	5 hours	Cr	G

D : dead, G : good recovery, MD : moderately disabled R : right, L : left

The age of patients ranged from 37 to 80 years with an average of 55.8 years. There was no young adult among them. Most patients were middle-aged. Seventeen of the 22 patients were male, an unexplained striking preponderance. Nine patients were heavy drinkers. Twenty patients presented with sudden severe headache with or without vomiting. Ten patients were comatose and six were stuporous or drowsy. Eight patients had anisocoria, four had bilateral pupillary dilatation, and ten had normal pupils. Fifteen patients had hemiparesis and, of these, ten had the hemiparesis ipsilateral to the haematoma. A CT scan was performed in 12 of the 22 cases, but there were no specific findings in the subdural haematoma of arterial origin. Extravasation was reported in only two out of 18 cases in which cerebral angiography was performed (1,8). Otherwise, angiography shows only an avascular mass over the convexity of the brain.

The interval from onset of symptoms to operation varied from five hours to two months. In eight of the patients, the interval was less than one day, in eight of the remaining 14 cases it was between two and 10 days, and in six cases between weeks and two months. Although this implies a subacute or chronic progression of the symptoms in at least twothirds of the patients, operation was required in all 22. Therefore, even mild symptoms should not delay the surgical intervention. The CT scan is a reliable tool in diagnosing this event and the majority of deaths occured in early studies, before the advent of CT scan.

The inverval onset of symptoms to operation in patients who died varied from eleven hours to five

weeks (mean 10.2 days, median 4.5 days), and in alive patients varied from five hours to two months (mean 8.6 days, median 4 days).

In all cases the site of the arterial bleeding was near the Sylvian fissure affecting one of the cortical branches of the middle cerebral artery. The pathopysiology of cortical arterial bleeding has been well discussed and summarized by Mc Dermott et al(11): a) Spontaneous arterial bleeding may come from the rupture of an arterial twig arising at right angles from a cortical artery which is a point of potential weakness, vulnerable to rupture in the event of increased pressure within the vascular lumen, described by Vance (19) as a "fire hose" rupture; b) rupture from a corcital artery at the site of adhesions with the dura mater which would predispose the artery to tearing with minor trauma (3, 17); c) rupture of a small artery traversing the subdural space and anastomosing a cortical artery to a dural vessel, the "rete mirabile" between cortical and dural arteries (11).

Among 100 cases of subdural haematoma Drake (4) reported 11 patients in whom the bleeding was due to a rent in a surface cortical artery, and presented histological evidence of a fragment of dura mater attached to a side of the arterial opening in one patient. Such an adherence would be torn by a rotatory movement of the brain at the time of trauma creating a defect causing the arterial bleeding. All these patients had a history of minor head trauma without any sign of cortical injury at the time of operation. He stressed that it would be unlikely for an arterial Knuckle or twing to rupture spontaneously.

In the biopsied cases, a hole in the artery corresponded to the point of origin of an avulsed arterial twig was found with no evidence of deficiency in the media of the parent cortical artery or of aneurysm, atheroma, arteritis, or evidence of previous old healed injury that had led to adhesions between the dura mater and the artery (13). Thus, a mechanical force could be blamed for tiggering the bleeding. These patients might have suffered from unrecognized or so trivial sudden motion of the brain as not to have been considered trauma or recorded. Hypertension may also be contributary though it is not considered an important predisposing factor in previous reports. However, it is was present in nine out of 22 patients and one subsequently proved to have severe essential hypertension with no history before the haemorrhage (11). A recent report by Arail (1) focuses on this issue. He demonstrated a leakage of contrast medium out of a small cortical artery in a hypertensive patient during extreme, uncontrollable hypertension who rapidly developed a subdural haematoma from this spontaneous arterial bleeding, and a tiny rupture of a small MCA branch was found at operation. In this case hypertension certainly was the major cause of subdural bleeding (1) though it is possible that the existing brain shift might have torn a cortical arterial adhesion.

Coagulopathies seem to have no role in this entity since coagulation screening tests failed to show any abnormalities in any of the cases who had had coagulation studies performed. Structural lesions such as vascular malformation, neoplastic diseases, and systemic metabolic factors such as infections, avitaminosis and alcoholism are also not contributary in the development of the spontaneous haematoma (2,6,7,9,15,16). If one consider the age of the reported cases and their past history, aside from an unrecognized sudden motion of the brain, hypertension, arteriosclerosis, and brain atrophy seem to be important factors in the pathogenesis. However, no single causative mechanism could be established with certainty in any patient.

The outcome of acute spontaneous subdural haematoma is favourable if recognised early and surgically treated. A high index of suspicion for this rare entity should be kept in mind since the clinical presentation with sudden headache and vomiting may closely resemble any from of cerebrovascular accident. The onset is invariably followed by deteriorating neurological status leading always to surgical evacuation. Spontaneous disappearance of acute subdural haematoma is rarely reported in traumatic cases (5,10,12,14), however, rapid and spontaneous resolution of an acute subdural haematoma has been shown to be due to redistribution of blood rather than real disappearence of the haematoma (16). Thus, early recognition of this lesion and prompt surgical intervention, even in patients with mild symptoms, is essential in the management.

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REFERENCES

1. Arai H: Acute subdural hematoma from arterial rupture shortly after the onset of cerebral subcortical hemorrhage: leakage of contrast medium during angiography. Stroke 14:281-285, 1983
2. Bassett RC, Lemmen LJ: Subdural hematoma associated with bleeding intracranial aneurysm. J Neurosurg 9:433-450, 1952
3. Byun Hs, Patel PP: Spontaneous subdural hematoma of arterial origin: report of two cases. Neurosurgery 5:611-613, 1979
4. Drake CG: Subdural hematoma from arterial rupture. J Neurosurg 18:597-601, 1961
5. Erkulvrawatr S, Srinivasan G: Complete spontaneous resolution of bilateral subdural hematoma. South Med J 71:456-458, 1978

6. Furui T, Ischihara K, Ikead A, et al: Subdural hematoma associated with disseminated intravascular coagulation in patients with advanced cancer. J Neurosurg 58:390-401, 1983
7. Hanakita J, Konda A, Nishihara K, et al: Spontaneous subdural hematoma in patients with advenced cancer-report of two cases. Neurol Med Chir 25:568-570, 1985
8. Hasegawa H, Bitoh S, Fujiwara M, et al: Subdural hematoma of arterial rupture: mechanism of arterial rupture in minor head injury. No Shinkei Geka 10:839-946, 1982
9. Kanter R, Kanter M, Kirsch W, Rosenberg G: Spontaneous posterior fossa subdural hematoma as a complication of anticoagulation Neurosurgery 15:241-242, 1984
10. Makiyama Y, Katayama Y, Ueno Y, et al: Acute subdural hematoma spontaneously disapperaing within 3 days following closed head injury: Report of two cases. Nihon Univ J Med 27:123-127, 1985
11. Mc Dermott M, Fleming JFR, Vanderlinden RG, Tucker WS: Spontaneous arterial subdural hematoma. Neurosurgey 14:13-18, 1984
12. Nagao T, Aoki N, Kitamura K: Acute subdural hematoma with rapid resolution in infancy. Neurosurgery 19:465-467, 1986
13. O Brien PK, Norris JW, Totar CH: Acute subdural hematomas of arterial origin. J Neurosurg 41:435-439, 1974
14. Polman Ch, Gijsbergs CJW, Heimans JJ, et al: Rapid spontaneous resolution of an acute subdural hematoma. Neurosurgery 19:466-448, 1986
15. Scoot M: Spontaneous nontraumatic subdural hematoma. JAMA 141:596-602, 1949
16. Shirai S, Owada T, Akimoto M, et al: Intractranial hemorrhage in infancy due to vitamin K deficiency. Brain Nerve 28:1359-1365, 1976
17. Talalla A, Mc Kissock W: Acute "spontaneous" subdural hemorrhage: An unusual form of cerebrovascular accident. Neurology 21:19-25, 1971
18. Tokoro K, Nakajima F, Yamataki A: Acute spontaneous subdural hematoma of arterial origin. Surg Neurol 29:159-163, 1988
19. Vance BM: Ruptures of surface blood vessels on cerebral hemispheres as a cause of cubdural hemorrhage. Arch Surg 61:992-1006, 1950
20. Yamanaka M, Chikuie S, Sasaki U: Acute spontaneous subdural hematoma: a case report. No Shinkei Geka 9:1207-1211, 1981
21. Yanai Y, Kohno N, Mitsui T: Acute spontaneous subdural hematoma of arterial origin. Surg Neurol. 23:417-420, 1985