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Case Report

A rare case of chronic subdural haematoma presenting with paraparesis: A case report and review of literature

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ABSTRACT

Objectives: To report a rare case of spontaneous chronic subdural haematoma presenting with paraparesis and to highlight that subdural haematoma as one of the differential diagnoses for unexplained paraparesis.

Summary of background data: Chronic subdural haematoma is common in elderly people usually presenting with altered mental status, monoplegia, headache and seizure. But spontaneous chronic subdural haematoma resulting in paraparesis is not reported in the literature.

Methods: A 58-year-old male patient presented with weakness of bilateral lower limbs and retention of urine and constipation. Patient was thoroughly evaluated and analysed.

Conclusion: Spontaneous chronic subdural haematoma should be considered as one of the deferential diagnosis for paraparesis in elderly.

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1. Introduction

Chronic subdural haematoma (CSDH) is known to occur spontaneously in elder people causing symptoms of altered mental status, monoplegia, headache, seizure, falls and extra pyramidal symptoms.¹ Trauma is an important factor in the development of CSDH. However, a history of head injury (direct trauma) is absent in about 30–50% of the cases. Indirect trauma seems to be more important. About half the patients have a history of fall but without hitting their head on the ground.^{2,3} In many situations, the trauma is so trivial that it is forgotten. Even though patients are known to present with

neurological deficits, we could not find any reports of chronic subdural haematoma presenting with paraparesis. Here we report a case of 58-year-old male patient presenting with paraparesis due to chronic subdural haematoma, and review the literature about the causes and mechanism of neurological deficits in chronic subdural haematoma.

2. Case presentation

A 58-year-old gentleman presented to us with weakness of bilateral lower limb since 1 week and was not able to pass urine and stool since 2 days. It was insidious in onset and gradually

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Abbreviations: CSDH, chronic subdural haematoma; MRI, magnetic resonance imaging; ICBF, intracranial blood flow.

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progressive. There was no history of any trauma, back pain or radicular pain. No history illness such as fever, cough, weight loss, anorexia, headache, altered mental status, diabetes or hypertension was noted.

On examination, there was no tenderness or deformity of spine. Neurological examination revealed increased tone in his lower limbs, weakness of his bilateral hip flexors (3/5) and knee extensors (4/5). All sensations were decreased below L1 dermatome. The deep tendon reflexes of both knee and ankle were exaggerated in both lower limbs. Babinski's reflex was flexor. Peri-anal sensation was decreased and voluntary anal contraction was weak.

We investigated the patient for spinal pathology, and lumbo-sacral magnetic resonance imaging (MRI) was done which revealed that there was no compression of spinal elements. Then after re-examining patient, we went ahead with the screening MRI of whole spine. During the process of the whole spine MRI, the radiologist incidentally noticed a hyperintense lesion in the skull. So we decided to do brain MRI and it showed "A fairly large capsulated extra-axial chronic haemorrhagic collection in right fronto-parieto-occipital region causing significant mass effect on the adjacent parenchyma, effacing the sulci and right lateral ventricle with gross midline shift towards left. Cerebellum, brainstem and cerebral parenchyma were normal intracranial vessels displayed normal expected flow void".

After the MRI of brain (Fig. 1), patient was diagnosed to have large chronic subdural haematoma, and neurosurgeons help was sought for the evacuation of the haematoma. Twist drill trephination was done and the haematoma was drained out and thorough wash was given. Post-operatively patient

general condition was stable and there was no re-accumulation of the haematoma. Patient was started on intensive rehabilitation programme, now after 2 months of treatment patient has got back his bowel and bladder function and able to walk without support.

3. Discussion

Chronic subdural haematoma is an old partially liquefied encapsulated blood, located between the dura mater and arachnoid. It was first described by Virchow in 1857 as pachymeningitis haemorrhagica interna". Later Trotter put forward the theory of trauma to the bridging veins as a cause and named it as "subdural haemorrhagic cyst".¹

Acute subdural haematomas generally occur in younger adults, after a major trauma, often associated with structural brain injury, and present within 72 hours. In contrast, CSDHs often occur in the elderly after a minor injury without any damage to the underlying brain, and usually there is a period of weeks to months before it becomes clinically evident. It has a peak incidence in the sixth and seventh decade of life.⁴ Our patient was in his fifth decade and did not have any history of trauma. He had spontaneous haemorrhage which is known cause of chronic subdural haematoma in 10% of cases.⁵ The incidence of CSDH is estimated to be 1.72/100,000 per year. The incidence increases steeply up to 7.35/100,000 per year in the age group 70–79.³ This incidence is expected to rise further due to increasing number of older people in the world.

3.1. Risk factors

With ageing, the mass of the brain decreases leading to an increase in the space between the brain and the skull from 6% to 11% of the total intracranial space. This causes greater movement of the brain within cranium causing stretching of the bridging veins, which are more fragile in the elderly than young, which easily get torn with slight movement causing bleeding.^{2,6}

Anticoagulation medications, bleeding diathesis, epilepsy and renal dialysis make this population more vulnerable for bleeding.¹

This population is more prone for dehydration, which causes intracranial hypotension predisposing for the intracranial bleeding. The incidence of fall and trivial injury is also more in these population as compared to the young adults.

In our patient, we looked for these predisposing factors and found none so considered it as idiopathic spontaneous CSDH which is one of the causes of CSDH.

The haematoma once accumulated grows slowly by absorbing fluid from neighbouring vessels due to higher osmotic pressure and due to rebleeding from the vessels in the capsule.^{7–9}

The intracranial pressure will slightly increase because of the place available in the cranium due to atrophied brain. The nature of the subdural collection may be watery, altered blood and fresh blood clots, depending on the age of the CSDH and the frequency of recurrent haemorrhages.¹⁰

CSDH patients usually present with altered mental status (50–70%).¹¹ It may vary from depression to coma. The altered

Fig. 1 – A large capsulated extra-axial chronic haemorrhagic collection in right fronto-parieto-occipital region causing significant mass effect on the adjacent parenchyma, effacing the sulci and right lateral ventricle with gross midline shift towards left.

mental status has to be differentiated from psychiatric illness and Alzheimers. Headache is found in 14–80%. It is more common in older people than younger.¹² Another most common symptom is neurological deficits. Hemiparesis was found in 58% of cases in one series.¹³ Weakness of the limbs is usually mild but drowsiness is out of proportion to the degree of neurological deficit. Mostly the deficit is contra lateral but ipsilateral symptoms are also reported.^{13,14} The deficits start insidiously, and are gradually progressive.

History of frequent falls is also one of common symptoms (74%). The frequent falls and CSDH form a vicious cycle one causing another one to increase. Seizure is another symptom, which causes vicious cycle (6%).

The other uncommon presentations are isolated neurological deficits such as cranial nerve palsy and extra pyramidal symptoms such as Parkinsonism like symptoms.

After all these literature survey we could not find any mention of paraplegia and bowel bladder involvement, so we did extensive re-evaluation of our patient and found other than subdural haematoma there was no other abnormality to explain the symptoms. After consultation with the neurosurgeons and physicians, we came to the conclusion that the huge size and gross midline shift the causes for the symptoms. So this is first of its kind of CSDH, which presented with paraplegia.

3.2. Mechanism for neurological symptoms in CSDH

There are many theories to suggest the neurological symptoms in CSDH. Initially, it was thought that direct compression is the cause for the neurological deficits by Rozzelle et al. and Luxon et al.¹³ Ropper¹⁴ while explaining the cause for the neurological symptoms in intracranial masses reported that in patients with acute masses, horizontal displacement of the brain above the tentorium measured by CT is more closely related to impairment of the conscious level than to the brain stem compression due to the tentorial herniation. From further analysis using MRI, Ropper¹⁴ stressed that most patients with acute unilateral masses have upper brain stem displacement predominantly due to horizontal shift at or above the tentorium.

Inao et al.¹⁵ studied the relation among neurological symptoms, size of the haematoma, amount of displacement and amount of intracranial blood flow (ICBF) and reported that ICBF in all areas decreased linearly with an increase in haematoma thickness or the severity of brain displacement. When the haematoma thickness increased beyond about 20 mm, both the superficial and deep brain structures get deformed, and the neurological signs of hemiparesis and mental changes occur in relation to the degree of midline displacement. They also inferred that hemiparesis may be induced by the reduction of ICBF in the ipsilateral pyramidal tract, including both the frontal cortex and subcortical structures. They clearly demonstrated that the ICBF reduction in the ipsilateral thalamus alone did not relate to mental changes; changes in cognition were associated with the reduction on both sides of the thalamus. Depending on the reduction of blood flow, they concluded that the patients with mental change had the greatest reduction of ICBF, those with paresis had a moderate reduction of ICBF, and those with

headache alone had the least reduction of ICBF in both the frontal cortex and thalamus on the haematoma side.

After going through all the literature in our case considering the large collection of blood, we thought that the cause paraparesis with bowel and bladder involvement is deformation of the bilateral hemispheres with a decrease in blood flow. It was rare case where the haematoma caused deformation of the hemispheres and a decrease in blood flow in the both cortices as it was noticed in thalamus in the study of Inao et al.¹⁵

3.3. Diagnosis

The most important step in the diagnosis of CSDH is a high index of suspicion. It should be considered in any patient with or without a history of trauma presenting with⁴ a change in mental status or worsening of pre-existent neurological or psychological illness,⁶ focal neurological deficit² and headache with or without focal neurological deficit. Computed tomography of the brain should be strongly considered in these patients to exclude a CSDH. We did not suspect CSDH in our patient at presentation, because the patient presented only with paraparesis and without any mental confusion or headache or any symptom related to cranium. In a series of 194 cases (in 1979), CSDH was suspected only in 28% of patients.¹³

In the era before computed tomography, the diagnosis was usually made by angiography or diagnostic burr holes. The advent of computed tomography has made a major impact on the radiological diagnosis of CSDH, and nowadays most of the cases are diagnosed on cranial computed tomography.

However, MRI scan may be required in patients with isodense haematoma without midline shift and in identifying small collections at the vertex, base of the skull and in the posterior fossa. It has been clearly shown that MRI is better than computed tomography in identifying small and transversely oriented collections where the computed tomogram has failed to identify a collection in as much as 80% of cases.¹⁵

Even though MRI has advantages, computed tomography remains the procedure of choice in the acute setting because of shorter examination time, which is important in acutely ill patients, reliability in identifying other parenchymal lesions, no magnetic interference (especially in patients on life support machines) and the ready availability.

3.4. Management

Treatment of CSDH is by surgical evacuation. Small haematomas resolve by themselves, but they require careful monitoring and if they deteriorate, they require repeat scan. A recent study has shown that 23% of patients did not warrant surgery because of small size of haematoma.

The commonly followed surgical procedures include drainage by twist drill/burr hole craniotomy or craniotomy. Twist drill trephination was associated with lower mortality rate and reoperation rate, and the duration of inpatient stays compared with burr hole craniotomy. Craniotomy is usually reserved for those patients in whom there is re-accumulation with recurrence of symptoms or where there is a solid haematoma.

4. Conclusion

After evaluating this case and literature review, we would like to conclude that CSDH can cause paraparesis with involvement of bowel and bladder, when it is large. So CSDH should be considered as one of the differential diagnoses for unexplained paraparesis in elderly.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Conflicts of interest

All authors have none to declare.

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