

CASE REPORT ΕΝΔΙΑΦΕΡΟΥΣΑ ΠΕΡΙΠΤΩΣΗ

Bilateral chronic subdural hematoma An unusual presentation with progressive spastic paraplegia

This is a case of progressive lower limb weakness in a 72-year-old man secondary to chronic bilateral subdural hematoma. This patient presented with a 3 week history of progressive difficulty in walking, with upper motor neuron signs but no sensory deficit. He had no significant risk factors for chronic subdural hematoma. Literature search revealed only one similar documented case of painless bilateral paraplegia secondary to chronic subdural hematoma, which, in contrast to this case, had fluctuating symptoms and signs.

Chronic subdural hematoma (CSDH) is usually the consequence of rupture of the bridging veins that are normally located in the subdural space. A history of head trauma is not essential to consideration of a diagnosis of CSDH;¹ up to half of the patients presenting with CSDH will not report any history of trauma.² CSDH can present with a vast range of symptoms and signs. Global neurological deficits are commoner than focal deficits,³ and it can present with gait deficits and falls,⁴ dysphasia,⁴ seizures,³ cranial nerve dysfunction,^{5,6} and parkinsonian⁷ or cerebellar features.⁸ A few cases of quadriparesis⁹ and a single case of transient paraparesis¹⁰ have been described. The diagnosis can usually be established by computed tomography (CT) scan of the brain. The treatment of choice is surgical evacuation, although a "watch-and-wait" (conservative) approach can be employed for selected patients.

This is a case report of an elderly gentleman presenting with increasing difficulty in walking over a period of 3 weeks. On examination he had features of spastic paraplegia. Initial investigations, including magnetic resonance imaging (MRI) of the spine for spinal cord pathology, were unfruit-

ful. Subsequent CT brain scan revealed a CSDH. Surgical evacuation was successful in restoring this patient's mobility.

CASE REPORT

A 72-year-old man presented to the Accident and Emergency Department with a 3 week history of progressive difficulty in walking. By the time he was seen in the hospital, he could not weight-bear on either leg. There were no other symptoms and on direct questioning he denied headache, backache, or visual, speech, sensory or sphincter disturbances. He reported no history of trauma. Socially, the patient was living at home alone, managing activities of daily living, such as cooking, cleaning and shopping independently and had an unlimited exercise tolerance before the onset of symptoms.

His past medical history included hypercholesterolemia, and polymyalgia rheumatica which was in remission. He had not used prednisolone for over 10 years prior to this event and his only medication was simvastatin.

On examination, he was comfortable, alert, orientated and hemodynamically stable. Examination of the cranial nerves and the peripheral nervous system of the upper limbs was unremarkable.

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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2012, 29(5):623-626

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Αμφοτερόπλευρο χρόνιο
υποσκληρίδιο αιμάτωμα:
Ασυνήθης εμφάνιση
με προοδευτική σπαστική
παραπληγία

Περίληψη στο τέλος του άρθρου

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Weakness

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Examination of the lower limbs revealed bilateral diffuse lower limb weakness (grade 3/5), bilateral hyperreflexia, increased tone in both legs, and down-going plantar reflexes. All modalities of lower limb sensation, perineal sensation and anal sphincter tone were intact and coordination tests were normal. Examination of all other systems was normal.

Liver function, urea and electrolytes, full blood count, clotting profile, chest X-ray and electrocardiogram were unremarkable.

Initial investigation included magnetic resonance imaging (MRI) of the spine which was normal (fig. 1). CT scan of the brain (fig. 2) showed an extensive bilateral subdural hematoma, up to 2 cm thickness on the left and 3 cm thickness on the right, involving the frontal and parietal regions, consistent with bilateral CSDH. No midline shift was noted.

The case was discussed with the local neurosurgical team who advised urgent transfer to their unit. Bilateral burr hole operation was performed to evacuate the subdural haematoma. Post-operatively, he was able to walk with a frame and his mobility gradually improved with physiotherapy. At 3 month follow-up, his mobility had returned to normal with no objective deficits identified on neurological examination.

DISCUSSION

CSDH is usually caused by slight or moderate head trauma, with consequent rupture of the bridging veins that are normally located in the subdural space. Pre-morbid conditions are an important pre-requisite for the development of a CSDH,⁴ and sufficient potential subdural space is required;⁴ the elderly and those with history of chronic alcohol abuse form a high-risk group for CSDH, due to a combination of brain atrophy and increased venous fragility. CSDH is also common in patients using anticoagulant or antiplatelet treatment; in one series of patients with CSDH, treatment with an anticoagulant, aspirin or heparin was present in 21%, 13%, and 5% of patients, respectively.¹¹ Other predisposing factors include falls, head injury, bleeding diathesis, epilepsy, low intracranial pressure or hemodialysis.³ CSDH commonly presents insidiously, and symptoms and signs may not become evident until weeks or months after the initial injury.¹ A history of trauma is lacking altogether in 25–50% of patients in most series, as the injury is often so slight that it is not considered important, or even forgotten by the patients.²

CSDH has variable presentation and course; the most common presenting features are headache, confusion and alteration in higher cerebral function.³ Light-headedness and seizures may also occur as a consequence of CSDH.³ Global neurological deficits, such as disturbance of consciousness, are more common than focal deficits. The common



Figure 1. Magnetic resonance imaging (MRI) of the spine of a 72-year-old man with spastic paraplegia. No abnormalities identified.

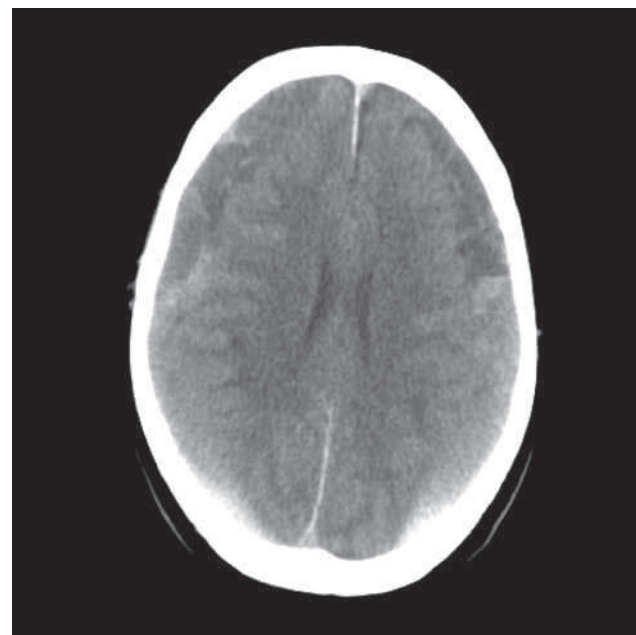


Figure 2. Computed tomographic scan of the brain of a 72-year-old man with spastic paraplegia showing extensive bilateral chronic subdural hematoma involving the parietal and frontal lobes.

focal neurological deficits associated with CSDH include papilledema,⁷ hemiparesis and hemisensory changes, which may be ipsilateral or contralateral. CSDH may also present with other neurological clinical features such as gait dysfunction, falls, and dysphasia.⁴ Third cranial nerve palsy, as a result of transtentorial herniation,⁵ and sixth cranial nerve palsy, presumably caused by increased intracranial pressure, were reported in 10% and 7% of patients with CSDH, respectively.⁶ The signs and symptoms of CSDH are usually persistent and or progressive but can occasionally be transient or fluctuating. Uncommon modes of presentation include Parkinsonian syndromes,⁷ cerebellar or vestibular features,⁸ due to CSDH involving the posterior fossa, and Gertsman's syndrome¹² (consisting of right-left disorientation, finger agnosia, agraphia and acalculia, with the lesion often localised in the left parietal cortex). Quadriparesis has also been described in two cases of CSDH.⁹ CSDH should be considered in the differential diagnosis of transient ischemic attack (TIA) or cerebrovascular accident (CVA),⁴ dementia,² epilepsy,³ and rapid onset parkinsonism. CSDH may be misdiagnosed as TIA or CVA,⁴ with a potentially harmful outcome if antiplatelet therapy is instituted.

One case report of paraparesis caused by CSDH was published by Schaller et al, who described a patient with intermittent, painless paraplegia precipitated by bitemporal CSDH with bilateral extension to the parietal lobes; there was no associated sensory or higher cerebral functional impairment.¹⁰ The authors hypothesized that CSDH caused impairment of blood flow in the area of the middle cerebral artery, with resultant paraplegia.¹⁰ The patient presented here also had intact sensory modalities and higher cerebral function, but showed progressive paraparesis.

The most widely utilized diagnostic imaging technique for CSDH is CT scan of the brain, as it is readily available and can provide information concerning the differential diagnosis. Contrast enhancement may aid its sensitivity. Magnetic resonance imaging (MRI) scan of the brain has certain advantages over CT in imaging extra-cerebral fluid collections, in terms of evaluating their size, and diagnosing small collections.¹³

Initial management consists of resuscitation to stabilise the patient's condition. The ABCDE (i.e., airway, breathing, circulation, disability and exposure) approach should be employed, with fluid and oxygen resuscitation administered as required. Capillary blood glucose should be tested. The

level of consciousness should be assessed objectively using a validated score, such as the Glasgow Coma Scale (GCS). Intubation and ventilation should be considered if the patient has a GCS $\leq 8/15$.

The treatment of choice for CSDH is surgical evacuation. A conservative approach can be employed with a small CSDH when the patient is asymptomatic or minimally symptomatic, or as palliative care for patients with significant co-morbidities who are deemed unfit for surgery. A prospective study of elderly patients, aged over 75 years, with CSDH showed that only 37% were treated with surgery, while 63% were managed conservatively.¹⁴ Surprisingly, when surgeons were asked how they manage CSDH, 94% reported that they utilize a conservative approach in less than one quarter of CSDH cases.¹⁵ Worse outcomes were observed in patients treated conservatively if they had midline shift on the CT brain scan.¹⁴ Conversely, a retrospective review of 114 cases treated surgically for CSDH showed that patients presenting with coma had a much better outcome than was originally anticipated.¹⁶

The most common serious post-operative complication is recurrence of hematoma. Post-operative CT has demonstrated that recurrent hematomas are common regardless of the technique used.¹⁷ The recurrence rate varies from 9.2% to 26.5%.¹⁸ Risk factors for recurrence include increasing age, bleeding diathesis, brain atrophy (acute or chronic), alcohol abuse and hematoma density.¹⁸ Maintaining a supine posture for 3 days post-operatively has been reported to reduce the risk of hematoma recurrence.¹⁸ Other complications include seizures, pneumocephalus, subdural empyema, intracranial hemorrhage, pneumonia, pulmonary embolism,¹⁷ infections (local, meningeal and systemic), and even death.

A striking finding by the study of Jones et al was the poor prognosis of CSDH, with a 31% 6-month mortality rate,¹⁴ although this may be related, at least partly, to the advanced age and multiple co-morbidities of the specific cohort of patients. About half of these patients were either diseased or had residual morbidity at 6 months.¹⁴

To the knowledge of the authors, this is the first reported case of progressive paraparesis secondary to CSDH. This is a rare presentation of CSDH, but this case demonstrates yet another in the vast range of modes of presentation of CSDH, and the need to consider the entire neuro-axis in the differential diagnosis of (sub)acute paraplegia.

ΠΕΡΙΛΗΨΗ

Αμφοτερόπλευρο χρόνιο υποσκληρίδιο αιμάτωμα: Ασυνήθης εμφάνιση με προοδευτική σπαστική παραπληγία

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Το χρόνιο υποσκληρίδιο αιμάτωμα προκαλείται συνήθως από ρήξη των αναστομωτικών φλεβών που εδρεύουν στον υποσκληρίδιο χώρο. Το ιστορικό εγκεφαλικού τραύματος δεν περιλαμβάνεται αναγκαστικά σε μια τέτοια διάγνωση. Το χρόνιο υποσκληρίδιο αιμάτωμα μπορεί να παρουσιάσει ευρεία κλινική συμπτωματολογία. Το παρακάτω ενδιαφέρον περιστατικό αφορά σε έναν άνδρα ηλικίας 72 ετών, ο οποίος εισήχθη στο νοσοκομείο λόγω δυσκολίας στη βάδιση και σπαστικής παραπληγίας και κατόπιν διαγνώστηκε με χρόνιο υποσκληρίδιο αιμάτωμα. Στο παρόν άρθρο συζητείται η κλινική εικόνα, οι διαγνωστικές εξετάσεις και η αντιμετώπιση αυτής της νόσου. Παρ' όλο που η παραπληγία είναι μια ασυνήθιστη μορφή παρουσίασης του χρόνιου υποσκληρίδιου αιματώματος, το συγκεκριμένο άρθρο επιθυμεί να αναδείξει το εύρος των πιθανών συμπτωμάτων του, καθώς και την ανάγκη να λαμβάνεται υπ' όψη μια πλήρης νευροανατομική διαφορική διάγνωση στην περίπτωση της σπαστικής παραπληγίας.

Λέξεις ευρητήριο: Αδυναμία, Παραπληγία, Υποσκληρίδιο αιμάτωμα**References**

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