Chronic subdural haematoma in the elderly

Chronic subdural haematoma is predominantly a disease of the elderly. The commonest mode of presentation is an altered mental state, which causes diagnostic difficulties. Although morbidity and mortality is high in untreated cases, early detection and neurosurgical intervention improves outcome significantly. In this article, **Drs Sanjeevikumar Meenakshisundaram and Sanjay Suman** discuss the risk factors, clinical presentations and management of this condition.

DR SANJEEVIKUMAR MEENAKSHISUNDARAM is a Specialist Registrar in Geriatric Medicine at Norfolk and Norwich University Hospital. **DR SANJAY SUMAN** is a Specialist Registrar in Geriatric Medicine at West Suffolk Hospital, Bury St Edmunds

Chronic subdural haematoma (CSDH) was first described by Virchow in 1857 as 'pachymeningitis haemorrhagica interna'. It is defined as a collection of encapsulated blood that is mostly liquefied and present between the dura mater, which is the outer membrane covering the brain and the arachnoid – often from ruptured veins crossing this potential space. The space enlarges as the brain atrophies and so subdural haematomas are more common in the elderly. To be chronic, symptoms need to be at least more than 20 days old according to the patient's history[1].

The incidence of CSDH in the elderly is about seven per 10,0000 per annum and it is expected to rise further owing to the consistent growth of the elderly population[2]. There is a male bias with a male to female ratio of approximately 5:1[3].

Important risk factors for CSDH include (*Table 1*)[1,4]:

- Advancing age
- Direct head trauma – this is an important risk factor (even if trival) but a positive history is only present in 30 to 50 per cent of cases. History of indirect trauma, commonly resulting from falls, is present in upto 75 per cent of cases[5,6]
- Increasing use of anti-platelet and anticoagulants drugs – drug usage has significantly increased in the past 10 years and has been implicated as a risk factor for CSDH[7,8].

In addition to these risk factors, alcohol abuse can lead to increased falls, altered platelet function and an impaired coagulation process – all of which predispose an individual to developing CSDH[9].

Pathophysiology

The current widely accepted hypothesis for the growth of CSDH is recurrent bleeding from the haematoma capsule that contains fragile newly formed blood vessels[5,10] (*Table 2*). Whereas a previous osmotic theory for expansion of a haematoma has been rejected by demonstration of the same osmolality in both the cerebrolspinal fluid and haematoma[10].

Clinical presentations

CSDH most commonly presents as altered mental state (50 to 70 per cent) and this includes confusion, agitation, unexplained drowsiness and coma (*Table 3*). The second most common presentation is fixed focal neurological deficit (40 to 60 per cent) such as a hemiparesis. Also in one to 10 per cent of patients, CSDH presents with transient neurological deficits such as aphasia, hemiplegia or hemisensory loss[9].

Drowsiness caused by CSDH is usually gradual in onset and progressive, in contrast to a fluctuating course as described in textbooks[4,7].

Table 1. Risk factors for developing CSDH

- Age greater than 75 years
- History of falls
- Use of anticoagulants and anti-platelet drugs
- Alcoholism
- Head injury
- Bleeding disorders
- Epilepsy
- Haemodialysis.

Table 2. Pathophysiology of CSDH

Trauma to bridging veins causes haemorrhage, which is covered by a layer of fibrin

↓

Fibroblast proliferation leads to formation of outer membrane

↓

Fibroblast invasion leads to formation of inner membrane in two weeks

↓

Liquefaction of haematoma occurs due to phagocytosis

↓

Haematoma resolves spontaneously or gets bigger resulting in CSDH.

Uncommon presentations

Seizures are thought to be rare in CSDH, but they have been reported in upto 10 per cent of cases as the presenting symptom[11]. Patients with CSDH may also present with headache as the first symptom, although this is less common in the elderly[9].

Other uncommon presentations of CSDH include new onset parkinsonism or sudden worsening of previously controlled Parkinson's disease. This is usually associated with bilateral CSDH and is reversible in many cases with neurosurgical treatment[12]. Isolated neurological deficits, postural instability due to basal ganglia lesions secondary to CSDH, Gerstmann's syndrome and vertigo have also been reported, with complete recovery following evacuation of

Table 3. Common clinical presentations of CSDH

- Altered mental state
- Focal neurological deficit
- Unexplained drowsiness
- Falls
- Headache
- Seizure.

the clot. CSDH has also been recognised as a reversible cause of dementia.

Diagnosis

It is not uncommon for clinicians to miss the diagnosis[13]. Symptoms are often attributed to side effects of sedative drugs, underlying dementia or possible sepsis. However, an increasing emphasis on eliciting the history of falls has led to increased numbers of patients being diagnosed with CSDH[9,14].

The most important factor in diagnosis is a high index of suspicion for CSDH in an elderly patient presenting with altered mental state or focal neurological deficit, with or without a history of trauma. A Computed Tomography (CT) scan of the brain usually confirms the diagnosis in most cases. In the early stages the haematoma appears hyperdense on CT scan, but in the later stages due to haematoma resorption it may become isodense or hypodense (at four weeks). Magnetic Resonance Imaging (MRI) may be required in some cases for identification of isodense haematoma or suspicion of small collections where a CT scan is inconclusive.

Differential diagnoses for CSDH include stroke, intra-cranial tumours, metabolic disturbances, psychotropic drug effects, normal pressure hydrocephalus and sepsis[11].

Management

Surgical evacuation of haematoma is the mainstay of management. A conservative approach consisting of inpatient observation, supportive treatment and monitoring of haematoma by serial CT scans may be reserved for patients who are unfit for surgery or those with a small haematoma on CT scan[15]. A small proportion of CSDH resolves spontaneously and this has been demonstrated in one study[14]. The length of inpatient stay is variable but may range from four to 22 weeks[9]. Surgical treatment significantly reduces the period of inpatient stay.

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Surgical techniques

The commonly followed procedures include twist drill craniostomy, burr hole craniostomy and craniotomy. Twist drill craniostomy and burr hole can be performed under local anaesthesia and haematoma is drained via a cannula by gravity.

When a twist drill is used to evacuate the haematoma, a hole is drilled at a 45 degree angle to the skull over the thickest part of the hematoma. The twist drill is then used to perforate the dura to release the subdural haematoma. A thin rubber catheter is gently guided into the subdural space, tunnelled under the scalp, and brought out through a stab incision. The rubber catheter is connected to a closed drainage system that is kept at the level of the ear or below the craniostomy site. This method is gaining popularity due to its relatively lower mortality rate compared to burr hole evacuation.

Burr hole is procedure where a burr is involved in drilling a hole. However, plunging into the brain substance has occasionally been known to occur.

Craniotomy requires general anaesthesia where the skull is opened, but this is reserved for cases with solid haematoma or recurrence following twist drill or burr hole craniostomy[16].

Post surgical complications

Post surgical complication include seizures, which occur in upto 10 per cent of patients. Symptomatic recurrence may also occur between two to four weeks post operatively and is more common in the elderly. Patients with bilateral CSDH and those

Key points

- CSDH is a disease with high mortality and morbidity.
- A high index of suspicion of CSDH is required in patients with a change in mental state with or without focal neurological deficit.
- Prompt referral and diagnosis is essential to influence outcome.
- Neurosurgical intervention leads to reversal of symptoms in most cases.

with inadequate expansion of the brain following surgery are at greater risk.

In addition, tension pneumocephalus can also occur – this is as a result of air trapping following surgery during the brain expansion. It leads to worsening of neurological status in about five to eight per cent of patients and the symptoms and signs are due to the gaseous substances contained in the air. This can be prevented by flushing out air during surgery; and post surgery, it can be prevented by elevating the drainage bag above head level for 48 hours.

Anticoagulation and CSDH

Patients who are anticoagulated on presentation should receive either Vitamin K or fresh frozen plasma to minimise the risk of haematoma progression. A careful risk-benefit analysis should be carried out prior to taking the decision to restart anticoagulation after surgical evacuation. Patients should be fully involved in this process. The recommended time to restart anticoagulation after evacuation is usually three to four weeks.

Prognosis

Mortality rates can be upto 30 per cent and predictors for inpatient mortality include the Glasgow coma scale, age greater than 80 years and time to surgical evacuation. Presentation with isodense CSDH has been associated with better prognosis whereas hypodense CSDH is associated with negative prognosis. Various other neurological gradings are available to predict outcome such as those put forward by Markwalder and Bender[5,17].

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Conclusion

The increasing incidence of CSDH (due to the ageing population) means there should be a high index of suspicion of the condition when an elderly patient presents with an altered mental state. A good functional recovery is possible with prompt scanning and surgical evacuation ■ GM

Conflict of interest: none declared