BILATERAL CHRONIC SUBDURAL HEMATOMA AN UNUSUAL PRESENTATION

INTRODUCTION:
Chronic subdural hematoma (CSDH) is usually the consequence of rupture of the bridging veins that are normally located in the subdural space usually caused by trauma(1). A history of head trauma is not essential for consideration of a diagnosis of CSDH; up to half of the patients presenting with CSDH will not report any history of trauma(2,3). CSDH can present with a vast range of symptoms and signs. Global neurological deficits are common and it can present with gait deficits and falls, dysphasia, seizures, cranial nerve dysfunction, parkinsonian or cerebellar features(4). A few cases of quadriparesis and paraparesis have been described(5). 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(6).

This is a case report of an elderly gentleman presenting with cognitive impairment due to bilateral subdural hematoma without any history of head trauma.

CASEREPOR:
A 72 – years old man presented to the Neurology department with a 3 months history of progressive cognitive impairment. On detailed history taking from his daughter, he was found to have mental defects like memory deficits for immediate and recent events and way finding difficulties. He was suffering from memory loss for immediate and recent events, way finding difficulty, difficulty in handling money and depressed affect. As a consequence, he is not able to carry out his daily activities without any difficulty.

Examination showed amnesia for recent events. He was able to register but was not able to recall. Examination of the cranial nerves was unremarkable. Examination of the limbs revealed increased tone in both lower limbs, bilateral upper and lower limb weakness (grade 4/5), bilateral hyperreflexia, and extensor plantar reflexes. All modalities of lower limb sensation, perineal sensation and anal sphincter tone were intact except hyperesthesia over the soles, coordination tests were normal. Examination of all other systems was normal.

Investigations like Liver function test, urea and electrolytes, full blood count, clotting profile, chest X-ray and electrocardiogram were unremarkable. CT scan of brain 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.

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 prerequisite for the development of a CSDH and sufficient potential subdural space is required. 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(7). CSDH is also common in patients using anticoagulant or antiplatelet treatment(8). Other predisposing factors include falls, head injury, bleeding diathesis, epilepsy, low intracranial pressure or hemodialysis. CSDH commonly presents insidiously, and symptoms and signs may not become evident until weeks or months after the initial injury. A history of trauma may be lacking altogether in 25-50% of patients(9), as the injury is often so slight that it is not considered important or even forgotten by the patients.

CSDH may have variable presentation and course; the most common presenting features are headache, confusion and alteration in higher cerebral function. Light headedness and seizures may also occur as a consequence of CSDH. The common focal neurological deficits associated with CSDH include 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. Third cranial nerve palsy, as a result of transtentorial herniation, and sixth cranial nerve palsy, presumably caused by increased intracranial pressure, were reported in 10% and 7% of patients with CSDH, respectively (10). 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, cerebellar or vestibular features, due to CSDH involving the posterior fossa, and Gertsmann’s syndrome.

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.

KEYWORDS :
The treatment of choice for CSDH is surgical evacuation(11). 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, age over 75 years, with CSDH showed that only 37% were treated with surgery, while 63% were managed conservatively(12). The most common serious post operative complication is recurrence of hematoma(13).

When an elderly patient presents with such progressive cognitive decline of a sufficient duration which interfere his daily activities it is commoner for the neurologist to consider it as a case of dementia syndrome and evaluate the cause for it at this age with the temporal profile of presentation with predominately temporal lobe features like memory loss and it is possible to consider Alzheimer's disease first in the differential diagnosis after that normal pressure hydrocephalus, multiple secondaries and chronic SDH was kept in mind. Keeping in view of the presence of long tract signs a CT scan was taken, it showed bilateral significant SDH. Retrospective questioning did not give any history of trauma.

As the CT scan did not show, midline shift or mass effect and as the density of blood was less, it was decided to treat the patient conservatively in consultation with the neuro surgeons. The patient was improving well during discharge

CONCLUSION:
Chronic SDH is very evasive in its presentation and very great suspicion must be kept to diagnose it. Chronic cognitive decline is not an uncommon presentation of bilateral SDH and should be considered in the patients of dementia in the elderly even with no history of head trauma.

REFERENCES