CASE SERIES

Unusual Causes of Chronic Subdural Hematoma

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ABSTRACT

Chronic subdural hematoma (CSDH) is a common but frequently under-investigated intra cranial lesion. Most times it is attributed to an obvious or trivial head injury, but it possibly may also be a presenting feature of a more serious underlying pathology. This article highlights three patients who presented with CSDH with varying underlying pathologies: acute lymphocytic leukaemia and prostate cancer. The need for thorough investigation of patients with recurrent CSDH and the challenges of emergency neurosurgical intervention were emphasized.

***Key words**: CSDH, Acute Lymphocytic Leukaemia, Prostate Cancer*

INTRODUCTION

Chronic subdural haematoma (CSDH) is one of the most common intracranial lesions encountered by neurosurgeons. Although majority of cases are related to a previous trivial or major head trauma, many other non-trauma related risk factors are known in the literature.[1] It may therefore be dangerous to trivialize the management of these CSDH since the underlying cause may be a sinister lesion. The aim of this paper is to report 3 cases of CSDH managed by the authors recently with underlying advanced acute lymphocytic leukemia (ALL) and metastatic cancer of the prostate, and to highlight the need for proper investigation of cases of CSDH.

CASE PRESENTATIONS

Case 1

A 51 year old male presented with headache and altered sensorium. Within the month preceding his admission, he had received two units of blood for unexplained anemia and an episode of epistaxis. There was no history of fever, seizure, haematuria, haematemesis, haemoptysis or haematochezia. He had a history of fall in his bedroom after he experienced dizziness on his way to the bathroom.

Physical examination revealed an unconscious man in moderate respiratory distress. On admission, he had a blood pressure (BP) of 120/80mmHg, pulse rate (PR) of 62/min, respiratory rate (RR) of 16 cycles/min and oxygen saturation ($SPO_{2)}$ of 96%. Admission Glasgow coma score (GCS) was 7/15, about 30 minutes later he suddenly deteriorated to 4/15 with left sided anisocoria. He had right spastic hemiparesis with grade 3 deep tendon reflex. The chest and pelvic examinations were clinically unremarkable.

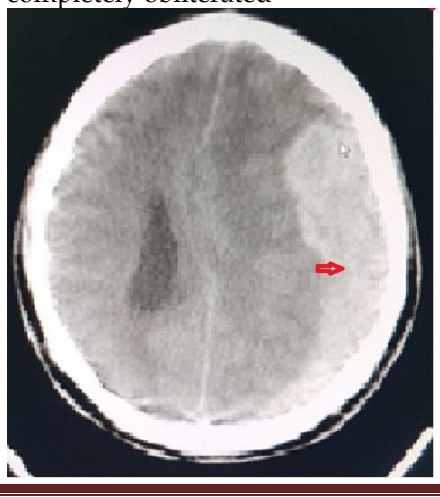
Emergency non-contrast enhanced cranial CT scan (Figure 1) showed left acute on chronic massive fronto-parietal subdural hematoma with midline shift, tight ambiens and quadrigerminal cisterns. Complete blood count (CBC) sample showed Haemoglobin (Hb) level of 6.6g/dl, platelet count of 37,000cells/mm^3 and white cell count of 13,200cells/mm^3 with 80% lymphocyte differential. Clotting profile revealed prothrombin time (PT) of 17.3 secs (normal =10-15), partial thromboplastin time (PTT) of 38.6 secs (normal=21.0-36.0) and international normalized ratio (INR) of 1.47.

Following the sudden deterioration, he was rushed in for an emergency left parietal mini-craniectomy with evacuation of CSDH under intense pressure mixed with blood clots, while laboratory investigation results were still being awaited. There was immediate neurological improvement on table. However there was continuous copious oozing of blood from the surgical field which was difficult to control despite all haemostatic measures applied. He was transferred to intensive care unit (ICU) for close observation. Subsequently, he experienced another acute deterioration, necessitating a repeat evacuation of acute subdural haematoma and a haemostatic pack of cottonoids was left in-situ as a last resort to tamponade the bleeding area.

He was reviewed by the haematologist. Bone marrow aspiration and cytology were done and a diagnosis of acute lymphocytic leukaemia (ALL) was made. Repeated blood transfusions and platelet concentrates were administered to help boost the haemostatic mechanism but these were not very helpful. Attempt to remove the haemostatic cottonoids packing four days post-surgery was unsuccessful as the oozing failed to stop, and hence had to be re- packed.

A day later, he experienced another crisis with his GCS dropping to 3/15 and the left pupil was unreactive to light. An urgent CT scan revealed an acute subdural haematoma which was evacuated through the previous parietal burr hole. He also had episodes of haemoptysis and haematemesis. Intravenous vincristine and prednisolone were commenced. The patient's clinical state progressively worsened until he died 14 days post admission.

Figure 1: Axial image of a non-contrast enhanced cranial computed tomography scan of a 51 year-old male who presented in coma as an emergency. The scan revealed a left fronto-parietal acute on chronic subdural hematoma with mass effect (arrow). The ipsilateral lateral ventricle was completely obliterated



Case 2

An 81 year old male admitted for irrational speech and aggressive behaviour of one week duration. He was a known hypertensive, diabetic and asthmatic and being managed for cancer of prostate. He had bilateral orchidectomy 14 months earlier and was receiving Bicalutamide. He had not been on anti-platelet medications.

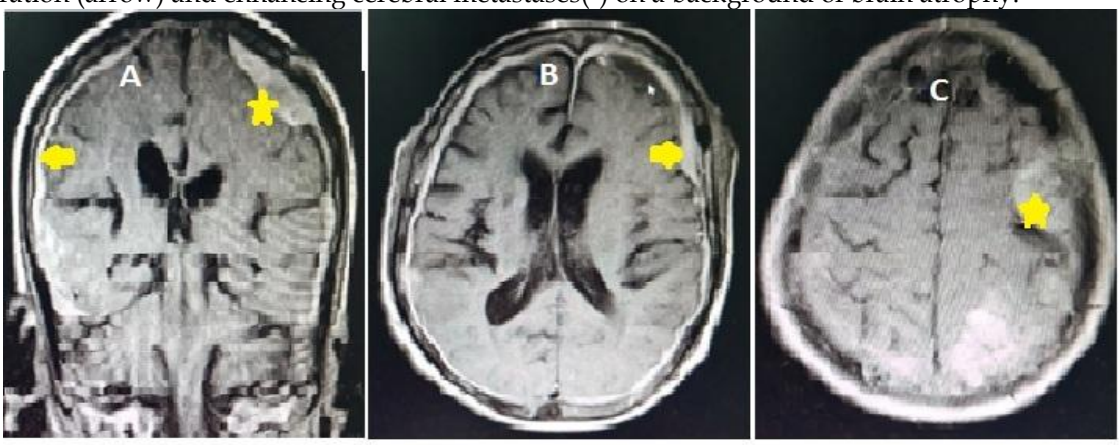
Physical examination revealed admitting: BP 100/60mmHg, PR 72/min, RR 22/min and SpO_2 98%. Glasgow Coma Score was 14/15. Pupils were 3mm and reacted briskly to light. He had no long tract signs. The chest, abdomen and pelvic examinations were clinically unremarkable.

Emergency cranial CT scan revealed left sub acute subdural haematoma with significant mass effect. Complete blood count showed Hb level of 11.5g/dl, platelet count of 140,000cells/mm^3 and WBC of 6,500cells/mm3 with 68% lymphocyte differential. Clotting profile revealed PT of 13.3secs (10-15), PTT of 31.7secs (21.0-36.5) and INR of 1.07.

He had left frontal and parietal burr holes with drainage of CSDH. Post-surgery, GCS improved to 15/15 and he was discharged home on the fourth day.

Ten weeks later, he was readmitted with irrational speech, bi-sphincteric incontinence, grade 4/5 paraparesis and GCS of 14/15. Cranial MRI (Figure 2) revealed left sub acute frontal subdural haematoma, generalized meningeal thickening and multiple metastases. He was reviewed by the Urologist who made a diagnosis of hormone escape prostate cancer. Family members declined consent for further surgical intervention.

Figure 2. Post contrast enhanced T1Wi brain MRI scan of an 81 year old man being managed for recurrent CSDH and cancer of the prostate. The image A is a coronal view that revealed a left sided subdural collection (*) exerting some mass effect with widespread meningeal enhancement (arrow). The image B is an axial view of same patient showing the diffuse thickened meninges as a result of metastatic dura infiltration (arrow) and enhancing cerebral metastases(*) on a background of brain atrophy.



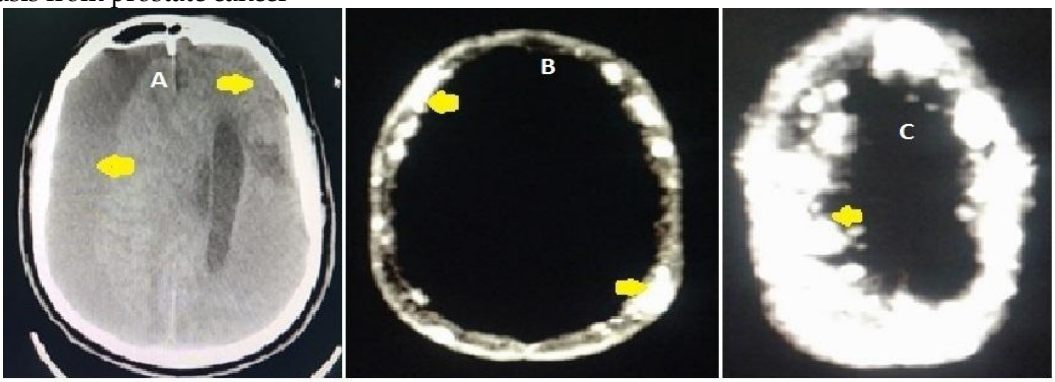
Case 3

A 77-year old man was admitted on account of lower limb weakness with subsequent alteration in his level of consciousness of two weeks and eleven days duration, respectively. He was involved in road traffic accident six weeks prior to onset of symptoms. He was a known hypertensive on clopidogrel and has a cardiac pacemaker. He had bilateral orchidectomy a year ago for prostate cancer.

On examination, GCS was 3/15. Pupils were 3mm on the left and 2mm on the right. There was no sign of meningeal irritation. Motor examination revealed increased tone globally; deep tendon reflexes were grade 3+ globally with no ankle clonus. The chest revealed

diffuse coarse crepitations. Other systems were essentially normal.
Brain CT scan showed bilateral CSDH significantly worse on the right. Bone window reconstruction revealed multiple hyper dense deposits in the diploic space of the skull bone consistent with metastatic prostate deposits. (See Figure 3). Total Prostate Specific Antigen on admission was 100ng/dl.

Figure 3. Axial image of an emergency cranial CT scan (A) of a 77-year old man admitted in coma showing bilateral CSDH worse on the right side with midline shift (arrows). B and C are bone window reconstruction showing hyper-dense infiltrates of the bone suggestive of osteoblastic calvarial bone metastasis from prostate cancer



He had an emergency right frontal and parietal burr holes drainage of CSDH. Repeat scan done a week later revealed expansion of the left subdural collection, prompting left frontal and parietal burr hole drainage of the CSDH. The patient made good neurologic improvement and was discharged to the urologist six weeks later with a GCS of 13/15.

DISCUSSION

Although CSDH following trauma is very common, some rare causes of CSDH exist. 'Acute Lymphocytic Leukaemia' and metastatic cancer of the prostate highlighted in this article have been implicated. Possible mechanisms that may explain the CSDH in these cases include dural metastases, abnormal function or reduced number of platelets in circulation and impaired coagulation.[1,2]

In some instances, cases of CSDH have been managed without extensive work-up of the patients, often because a prior history of trivial trauma and aspirin medication are obtained. Although the first case reported in this study had a trivial trauma, there was a more serious haematological cancer responsible for the CSDH. One lesson from this report is the need to extensively review patients managed for CSDH for other underlying pathologies no matter the clinical response, more especially in recurrent CSDH.[2,3,4]

In the second case reported, the patient had an initial clinical improvement but following a recurrence, he was further investigated. MRI with contrast revealed metastatic dura infiltration which prior brain CT scans did not show. The underlying pathology was linked to metastatic hormone-escape prostate cancer. In both cases, the CSDH was just a 'red herring'. The management plan must therefore accommodate proper treatment of the underlying cause of the CSDH with other specialties.

These cases have some surgical management challenges. Thrombocytopenia of <

50,000cells per mm^3 is generally a contraindication for surgery. Even if the platelet count is normal, the platelet quality in ALL patients may be impaired, generally making surgery unsafe. Unfortunately, options for achieving hemostasis are very limited in the brain and this further compound the risk of re-bleed.[4,5] It may be safer to argue in favour of conservative care in the face of such findings of severe thrombocytopenia or prolonged bleeding time because of the challenges of hemostasis. However, this is difficult to justify in emergency situations like the first case that suddenly deteriorated before full pre-operative work-up investigations were available.

CONCLUSION

Chronic subdural haematoma may be a red herring to a more sinister underlying malignancy. Therefore, the investigation of patients with chronic subdural haematoma should not be trivialized no matter the initial clinical response following surgery.

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