รายงานผู้ป่วย

The Life Threatening Bilateral Subdural Haematoma : A Case Report

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Abstact : **Chronic subdural haematona (CSH) is one of the great mimicker in neurology and usually leading to misdiagnosis, because the symptoms and signs are variable. Most patients usually had no previous history of head injury. Bilateral subdural haematoma is even more difficult to diagnosis and it is one of the most life threatening condition, but fortunately it is a very rare event. We report a 52 year old man who had suffered from severe headache for 1 mouth and later developed confusion. CT scan and lumbar puncture were done at the other hospital. But the diagnosis was dubious and paracetamol was prescribed. He was referred to Siriraj Hospital by himself. Repeated lumber puncture was done and the result was still normal then he was consulted for definite diagnosis. Reviewing history and physical examination showed no clue for the diagnosis but his previous neuroimagine revealed bilateral isodensity subdural haematoma. Haemolytic clot from subdural haematoma was removed bilaterally in the same day which resulted in complete resolution of confusion and headache.**

เรื่องย่อ : **ภาวะเลือดคั่งที่ผิวสมองทั้งสองข้างที่คุกคามต่อชีวิต : รายงานผู้ป่วย ๑ ราย**

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สารศิริราช ๒๕๔๑; ๕๐: ๑๑๗๖-๑๑๗๙.

ภาวะเลือดคั่งที่ผิวสมองเรื้อรัง (chronic subdural haematoma) เป็นภาวะที่แพทย์ให้การวินิจฉัยโรคยาก, เนื่องจากมีอาการและอาการแสดงทางระบบประสาทได้แตกต่างกันและมีหลากหลายมาก. ยิ่งกว่านั้นแพทย์มักซักไม่ได้ประวัติของการเกิดอุบัติเหตุทางสมองมาก่อน. ในรายงานนี้เป็นตัวอย่างของผู้ป่วยชายไทย อายุ ๕๒ ปี, ที่ไม่มีประวัติอุบัติเหตุทางสมองใด ๆ, แต่ผู้ป่วยมีอาการปวดศีรษะมากมา ๔ สัปดาห์ และต่อมามีอาการสับสน. ผู้ป่วยได้ไปโรงพยาบาลเอกชนแห่งหนึ่ง ซึ่งได้รับการตรวจร่างกายและสืบค้นโรคด้วยคอมพิวเตอร์สมองและเจาะหลังตรวจน้ำไขสันหลัง, แต่ยังไม่สามารถให้การวินิจฉัยโรคได้, ผู้ป่วยจึงมารับการรักษาตัวที่โรงพยาบาลศิริราช. ขณะรับการรักษาตัว

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ในโรงพยาบาลศิริราชผู้ป่วยได้รับการเจาะน้ำไขสันหลังอีกครั้งซึ่งได้ผลปรกติ. เมื่อประสาทแพทย์มาซักประวัติ, ตรวจร่างกาย และดูคอมพิวเตอร์สมองที่เคยทำมาแล้ว, จึงวินิจฉัยโรคว่ามีเลือดคั่งที่ผิวสมองทั้งสองข้าง. ประสาทศัลยแพทย์ได้ทำผ่าตัดเอาเลือดที่คั่งที่ผิวสมองทั้งสองข้างออกโดยด่วน. ผู้ป่วยหายจากอาการสับสนและปวดศีรษะโดยสิ้นเชิงภายหลังการผ่าตัดเสร็จ.

CASE REPORT

A 52 year-old man was referred from private hospital because of his worsening headache and not response to any medication. He enjoyed his good health untill 4 weeks ago that he had developed nonthrobbing headache. The headache was characterised by tightness around the head and the neck with fluctuation in each day. No nausea nor vomiting or double vision was observed. His severe headache was worsening with postural change and some improving after taking paracetamol. He was treated as a vascular headache condition but did not improved. Two weeks later he developed confusion, CT brain scan was done and official report was as the following: mild diffuse cerebral oedema, calcified basal ganglias both sides and increased enhancement at basal cistern and parietal cortical sulci but no space occupying lesion was seen (Figure 1, 2). Lumbar puncture (LP) demonstrated normal initial pressure and normal cerebrospinal fluid (CSF) profile (no details in the referral letter). After LP, his confusion and headache improved, then he referred himself to Siriraj Hospital. At the Outpatient Department (OPD), complete physical examination revealed normal finding including fundoscopy. Provisional diagnosis of meningoencephalitis was made, the repeated LP was done at the OPD with the intitial pressure was 8 cmH_2O, CSF was clear and revealed only few lymphocytes, 92 mg/dl of protein, 85 mg/dl of sugar and Indian ink preparation for cryptococcal infection was negative. His blood sugar was 117 mg/dl, anti HIV showed non-reactive. He was referred to us (NP) for definite diagnosis and proper management. After reviewing history, complete neurological examination and viewing CT scan, he was admitted in the neurological investigation bed and neurosurgeon was notified immediately for surgical intervention.

On examination, the temperature was 37° C, the pulse rate was 84/min, the respiration rate was 24/min and the blood pressure was 140/100 mmHg. General appearance showed sthenic build, not pale, no cyanosis, no edema, no skin lesion and no oral thrush. Nervous system demonstrated alert but in severe distress due to headache with some confusion. Fundoscoic examination showed no evidence of papilloedema. No muscle weakness was detected with normal deep tendon reflexes and no signs of meningeal irritation. Other systems were normal.

Neurosurgical intervention was performed on the admission date and 60 ml of haemolysed blood was removed from each side of his brain. After operation, his headache and the fluctuation of confusion were disappeared. He was discharged home 1 week after admission without any complication.

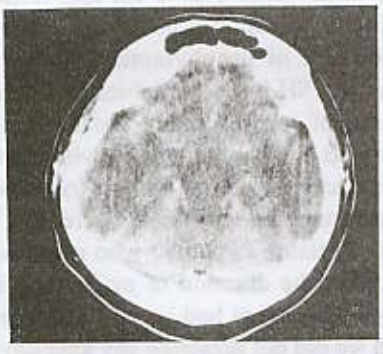

Figure 1. CT brain scan showed markedly diffuse cerebral oedema with compression of the quadrigeminal cistern.

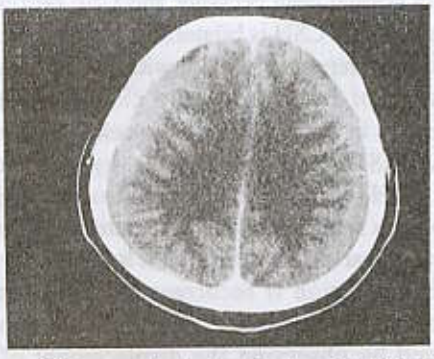

Figure 2. Demonstrate the isodensity bilateral subdural haematoma with diffuse brain oedema resulting in obliteration of lateral ventricle of both sides.

DISCUSSION

Chronic subdural haematoma (CSH) is a treatable condition that is frequently misdiagnosed because of its difficulty to diagnose by history taking and physical examination alone. About 20-30 % of patients failed to inform the history of head injury. Trivial trauma is usually the causation of this condition and it is often unnoticed or forgotten, particularly the elderly patients. Most CSH patients are usually aged over 50 years. Chronic alcoholism, epilepsy, bleeding diasthesis and elderly patients are the predisposed conditions to subdural haematoma (SDH). Mojor causes of SDH are often related to trauma and intracranial hypotension as cerebral atrophy or intracranial shunt. Spontaneous intracranial hypotension had been reported to be the causation of SDH. CSH patients are usually presented with variable in the symptoms and neurological signs and most of them are not pathognomonic. Fluctuation of headache is usually related to position, slow in thinking, confusion, change in personality and seizure are often the manifestations of CSH and all of these symptoms are nonspecific to any neurological condition. Common signs of CSH are impairment of consciousness (53%), hemiparesis (45%), papilloedema (24%) and of third cranial nerve abnormality (11%).[2] Uncommon manifestations are parkinsonism, dystonia and paranoid behaviour.

In our case, he had experienced the fluctuation and progressive severe headache and then developed confusion. In spite of these symptoms, the physical examination was still normal. Differential diagnoses of CSH include space taking lesions such as brain tumour or abscess, subarachnoid haemorrhage with hydrocephalus, stroke and depression. CT scan is the most useful investigation. LP in CSH is usually normal both pressure and CSF profile. It can be harmful if CSH is bilaterally. Xanthochromic CSF had been reported in CSH with SAH.[7] CT scan appearance of SDH is usually characterised as hyperdensity in acute SDH (first week), isodensity in subacute SDH (1-3 weeks) and hypodensity in chronic SDH (over 3 weeks). Acute isodensity SDH has also been reported in anaemic patients. Chronic SDH can appear as mixture of both hypodensity and hyperdensity lesion if recurrent bleeding occur.[1] Diagnosis of an isodensity subdural collection is fascilitated by observing the displacement of the gray-white junction or cortical sulci away from the calvaria, or by the obliteration of lateral ventricle and midline shift or the enhancement of fibrous capsule surrounding the blood clot. Bilateral isodensity SDH are more likely to be misdiagnosed because they usually produce no midline shift. MRI is more sensitive to identifiy and characterises subacute and chronic SDH but it is double the cost in comparison to the CT scan. Subacute SDH in the MRI usually shows higher signal than general cortex on both T_1 and T_2 weighted images and lower signal on T_1 and T_2 weighted sequences in CSH. Cerebral angiography is now rarely performed due to the invasiveness of the procedure and its high risk for complication (1 percent). In this patient, misdiagnosed of CSH was due to the report of an inexperienced radiologist whiach diagnosed bilateral CSH as generalised brain oedema.

CSH can be managed by both medical and surgical means. Medical management includes combination of bed rest, osmotic diuretic and corticosteroid in some selected patients with minimal blood collection and no neurological symptoms and signs. Most cases of CSH need surgical intervention especially in patient with symptomatic chronic SDH. Evacuation of chronic SDH has been achieved with craniotomy, burr-holes, or twist drill aspiration in combined with closed system drainage. Newly treatment of septated subdural haematoma by using neuroen-doscopic intervention is less invasive than craniotomy and membranotomy technique.

Recent analysis of the prognostic factors in 65 patients of CSH showed 4.6 % of mortality rate and 1.5 % recurrence after surgery.[6] The worst prognostic factors depend on the background of alcoholism, coagulopathy, decreased level of consciousness, lower score of Glasgow Coma Score, maximum thickness of the haematoma over 2 cm and respiratory and neurological complications.[5]

The role of prophylactic anticonvulsive medication for CSH has been debated for years because the onset of recent seizure was found in about 20 % of patients and was associated with increasing in morbidity and mortality. Several retrospective studies demonstrated a significant decreasing in the occurrence of seizure in patient who received anticonvulsant,[8] but the duration of medication is still controversy. Our patient had no complication after surgery and we did not prescribe any anticonvulsant for seizure prophylaxis.

CONCLUSION

Chronic subdural haematoma is a treatable condition but may be harmful if misdiagnosed, especially bilateral lesions. CT scan is the most important and appropriate for diagnosis but its finding should be interpret cautiously by the attending physician.

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