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An Unconscious Old Man With Dramtic Post-Operative Recovery

A case of Chronic Subdural Hematoma

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By convention the term Chronic Subdural Hematoma (CSDH) applies to those hematomas which present over 20 days following injury[1].

On 15th November, 1987 our attention was drawn to a patient admitted in Medical unit for five days. Patient could not be diagnosed and now was gasping with periods of apnea, when C.T. Scan brain showed a bilateral CSDH. A rapid infusion of mannitol and an immediate simple burr-hole evacuation made him to sit and talk few hours later. This case is instructive as it illustrates the diagnostic difficulties of this common neurosurgical entity.

CASE REPORT

N.A. a 70 years old active farmer from Okara presented in medical outpatient on 11th November, 1987 with complaints of progressive weakness of legs without pain or sensory disturbances. Weakness progressed to upper limbs until he was finally unable to walk without support. Patient also developed confusion and would not reply correctly for the past 15 days. There was no bistory of head injury. One admission he was drowsy and disoriented in time and space. Motor power was decreased on left side as compared to right and in lower limbs as compared to uppers. Tendon jerks were brisk, abdominal reflexes absent and plantar response was extensor bilaterally. On ophthalmoscopy papilloedema was present. One the basis of history and clinical examination a provisional diagnosis of midline S.O.L. or encephalitis was made. Patient's level of consciousness and general condition continued to deteriorate rapidly. One the 5th post-admission day he developed cheyne-stockes breathing and signs of tentorial herniation. At this stage C.T. Scan of brain showed bilateral chronic subdural hematomas (Fig.1). An urgent call was sent to department of Neurosurgery. Examination at this stage revealed a deeply unconscious patient with no response to painful stimuli. Pupils were

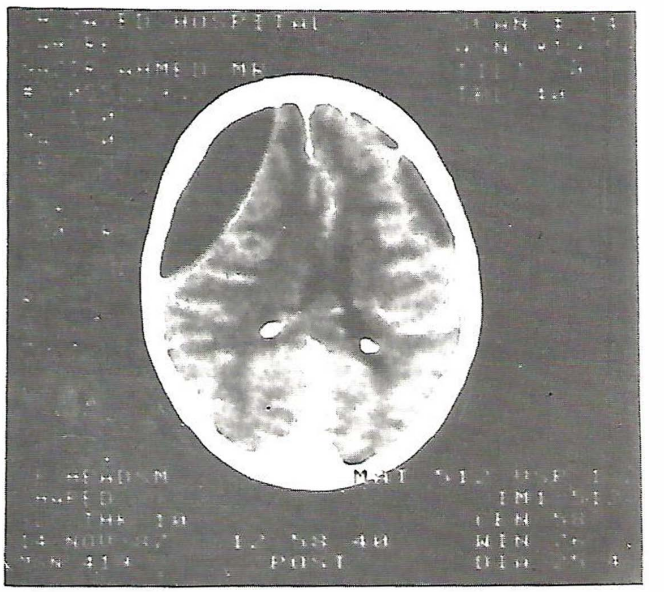

Fig.1 C.T. Scan of brain showing bilateral subdural hematoma.

bilaterally dilated and fixed. There were periods of apnea. He was moribund and was declared critically ill 200 ml of Injection mannitol 20% was rapidly infused. Patient was taken to operation immediately and five burr-holes were made. Bile coloured yellow fluid was drained from anterior burr-holes and cherry red from posterior ones. Subdural spaces was washed with normal saline.

Post-operative recovery was dramatic. Patient regained consciousness a few hours after the operation and hemiperesis resolved completely. Two days after operation patient was fully alert, oriented, cheerful and walking with help. Patient has been followed up and he is back on his farms, carrying out his routine tasks.

DISCUSSION

Incidence of CSDH is 1-2/100,000yr. Majority of tye patients are elderly or n late middle age. Age

specific incidence for third decade is reported to be 0.13/100,000/yr. whereas 7.4/100,000yr for seventh decade i.e., 60 folds to that for third decade[1-3]. Regarding pathogenesis of CSDH it is agreed that initial bleed is from the rupture of the bringing veins from cortex to venous sinuses. This initial hemmorrhage fails to compress underlying brain, because either hemmorrhage may be small or in elderly patients a large amount may not produce cerebral compression or its symptoms because of brain atrophy of old age. Within 24 hours the hematoma is invaded by fibroblasts from undersurface of the dura. These organize into an outer thick membrane after one week. In the third week an inner membrane forms, thus the hematoma becomes encapsulated. Subsequent enlargement of CSDH has been a matter of great controversy. In 1932 GARDNER postulated, that the capsule of CSDH acted as an osmotic membrane and that fluid was drawn into the hematoma from the C.S.F. GITLIN suggested that effusion of albumin must be taking place through the walls of the capillaries of the hematoma membrane. It was suggested that recurrent hemmorrhage may cause progression of CSDH as it is not uncommon to find CSDH of various age & consistency co-existing within that capsule. Currently accepted view is that osmotic effect is not responsible for hematoma enlargement, but escape of proteins through leaky vellels and recurrent hemmorrhage are the cause. The studies with intravenous injection of Isotope labelled RBCs have shown that the labelled cells appear in the CSDH[1-3].

Regarding diagnosis the key word is "suspicion" especially in elderly but no age is immune to CSDH. WE have recently encountered as isodense CSDH in a one year old child. A history of head injury, may lead to suspicion of CSDH, but between 25-48% of patients do not give any history of head injury. Even when remembered, injury is often mild. Chronic Alcoholism may account for the failure to obtain a history of head injury. Fortunately it is not a problem in this country. Other precipitating factors could be epilespy, shunting procedures of anti-coagulant therapy. In most series, less than half of all patients presented with impaired consciousness. Headache is a cardinal symptom and is frequently found in patients without other signs ore symptoms. Patient may present with confusion, mental changes, nausea, vomiting or seizures. In fact what makes the diagnosis more complex is its multiple pattern of presentation. Various authors have emphasized and described its various "imitations". Potter and Fruin in their book "GERIATRICS" have called it "THE GREAT IMITATOR". Because the sings and symptoms of CSDH are non-specific and a history of head injury may not be obtained, the admission diagnosis is in error as often as 40% of the times in some of the world's best centers. Unfortunately they diagnosis may not be made until Autopsy. A study conducting computerized tomography of all the patients kept in a mental asylum showed that 2% had CSDH. Common misdiagnosis include stroke or transient ischemic attacks, brain tumor, senile dementia, mental illness and encephalitis[1-3].

Skull x-ray are of little use in diagnosis of CSD Hexcept for the pineal shift that may be found occasionally. Overall accuracy of isotope brain scan in detecting CSDH has been reported to range from 50-100% Its use has been superceded by cerebral angiography and C.T. Scan. EEG findings are non-specific. Most authors stress the dangers inherent in performing lumbar puncture in the presence of such mass lesion. Cerebral angiography is 99% accurate. Hematoma appears as an avascular area between the cortical surface and the inner table of skull in the frontal projection. C.T. Scan is now the preferred method for the evaluation of patients with CSDH, because it is rapid, accurate, non-invasive and permits the evaluation of all intracranial structures in a single study In the first week following injury, all hematomas appear hyperdense compared to Brain. In the second and third weeks they are usually isodense and that is the time when they can be missed if contrast material is not given. After the third week, however, three quarters are hypodense[1,3,4]. Correlation between the computerized tomographic finding sand the colour of hematoma evacuation have shown that all hypodense CSDH consisted of golden yellow or bile coloured fluid and all the isodense CSDH were cherry red or brown in colour[5].

Although regimens of diuretics, bedrest, or corticosteroids have been advocated but prevailing neurosurgical opinion is that symptomatic patients are best reated with surgery. A number of operations have been proposed. Craniotomy (with or without excision of membranes), burr-holes or twist drill evacuation of the hematoma. Each surgical procedure has its specific indications depending on the age and chronicity of the hematoma. Residual collection may be due to incomplete removal or recurrence of hematoma. Recurrence is almost twice as common in patient who have craniotomy when compared with patients who have burr-hole evacuation. Rapid evacuation of large CSDH can cause deterioration of patients and may lead to death. Infection after CSDH is uncommon. The

common infectious complications include subdural empyema, brain abscess, bone flap infections and meningitis. Seizures in the post-operative period occur in 11% of the cases and all patients should be treated prophylactically with anti-convulsants. Most larger series report a mortality rate of 10% or less. Luxon and Harrison report a 14% incidence of minor morbidity reports that three quarter of all patients resume normal functions after operation for CSDH[1-3].

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