



SPONTANEOUS RESOLUTION OF ACUTE SUBDURAL HEMATOMA : AN INSTITUTIONAL EXPERIENCE

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ABSTRACT Acute subdural hematoma (SDH) caused by traumatic brain injury are a common emergency in neurosurgery. Mortality rate of an ASDH in the setting of major head trauma is estimated between 50 and 90%. The decision about conservative or surgical management is based on the neurological status of the patient, size of hematoma, and midline shift. We are reporting a case series of acute subdural hematoma with spontaneous resolution with its pathophysiology and review of literature.

KEYWORDS : Sub Dural hematoma, Spontaneous resolution , Coagulopathy

INTRODUCTION:

Acute subdural hematoma (SDH) caused by traumatic brain injury are a common emergency in neurosurgery. It is rapidly clotting blood collection below the inner layer of the dura but external to the brain and arachnoid membrane. Mortality rate of an ASDH in the setting of major head trauma is estimated between 50 and 90%. [4] The decision about conservative or surgical management is based on the neurological status of the patient, size of hematoma, and midline shift.

Traumatic acute subdural haematoma (ASDH) is a life threatening condition. The phenomenon of spontaneous regression of an ASDH as observed by CT scan has been reported for the first time in 1986 [5, 6] and several cases have been described ever since. Rapid resolution of ASDH related to coagulopathy is a rare phenomenon; to our knowledge, only one case has been reported [10]. We are reporting a case series of acute subdural hematoma with spontaneously resolution.

CASE 1: 78 year male presented to J.A.H Trauma centre with history of being hit by stray animal (bull). On presentation his Glasgow coma scale was E4V5M6. Noncontrast CT brain was suggestive of right fronto-temporo-parietal acute Sdh of maximum depth of 2.2cm with left side midline shift of 7mm with fracture of left parietal & temporal bone. Patient was having history of coronary artery disease, underwent PTCA about 3 years, taking tablet ecospirin, clopidogrel, isosorbide. His prthombin time was 23.2 sec, INR Value- 1.8 and 2D-ECHO was suggestive of Severe LV Systolic dysfunction , LVEF-30%, Mild MR , Mild PAH. Patient was given injection Vitamin K along with fresh frozen plasma transfusion and anti-coagulants were stopped. Patient was planned for conservative management in view of his good neurological status & de-arranged coagulation profile. Plan was correction of coagulopathy in order to reduce the risk of hematoma expansion and emergency surgery whenever required. Patient repeat NCCT head was done on 22/8/21 which was suggestive of complete resolution subdural collection of right fronto-temporo-parietal acute SDH. Patient improved with medication and was discharged in stable condition after 8 days of admission.

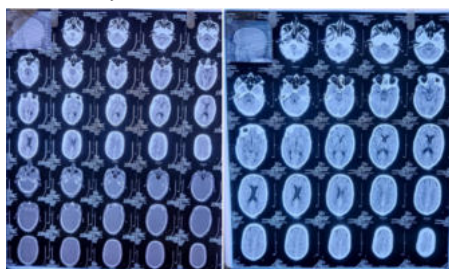


Figure 1.1

Figure 1.2

Figure 1.1-NCCT head of 18/8/21 was suggestive of Right side fronto-temporo-parietal acute Sdh of maximum depth of 2.2cm with left side midline shift of 7mm. with fracture of left parietal & temporal bone.

Figure 1.2-NCCT head of 22/8/21 was suggestive of subdural collection of Right fronto-temporo-parietal region of max depth of 5mm with Lt parital & temporal bone fracture.

CASE 2: 1.5 year male referred to J.A.H trauma centre with history of fall from bed about 4 days back. On examination his Adelaide coma scale was E4VcryM6 with left sided hemiparesis. Previous MRI Brain (figure 2.1), suggestive of hyperacute subdural haemorrhage along right fronto-temporo-parietal convexities causing buckling of underlying parenchyma with effacement of right sulcal spaces, 8mm midline shift towards left. On admission NCCT head (figure : 2.2) was done which was suggestive of complete resolution of hyper acute SDH with mass effect. Patient improved symptomatically and discharged from hospital after 5 days.

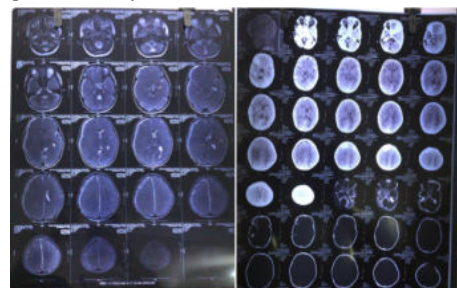


Figure 2.1

Figure 2.2

Figure 2.1: MRI Brain -Acute subdural haemorrhage along Right fronto-temporo-parietal convexities causing buckling of underlying parenchyma with effacement of right sulcal spaces, 8mm midline shift towards left.

Figure 2.2: NCCT Head -A crescentic shaped hyperdense lesion of blood attenuation seen in subdural space of right frontal region of max. depth 4mm.

CASE 3: 60yr female had alleged H/O of RTA (fall from bike) patient was a pillion rider, following which patient presented to J.A.H Trauma centre. At presentation her GCS was E4V5M6 , Non-contrast CT head was suggestive of left fronto-temporoparietal acute SDH with midline shift of 5mm. Patient was managed conservatively & repeat non-contrast CT was done after 36hrs showed resolution of acute SDH. Patient was discharged after 2 Days.

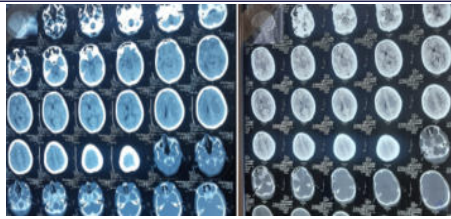


FIG3.1

FIG3.2

12. Chaudhary N, Krivosheya D, Small E, Hsia C, Ng W, Leung A. Rapid resolution of acute subdural hematoma in a coagulopathic patient. *Can J Neurol Sci.* 2013;40:599–600

DISCUSSION:

Although infrequently reported, rapid spontaneous resolution of an ASDH may be underestimated [3]. As per Guidelines for surgical management of Traumatic Brain Injury by Brain Trauma Foundation & the Congress of Neurological surgeons, surgery is indicated in acute subdural haematoma if CT scan shows midline shift > 5 mm, or haematoma thickness > 10 mm regardless of patient's Glasgow coma scale score.[2] Healthy individuals possess adequate amounts of clotting factors, regulatory proteins, and platelets to achieve optimal clot formation[10].

Rapid resolution of Acute SDH, associated with brain atrophy due to age[7]. The presence of abundant space in subdural and subarachnoid compartments help wash out the hematoma. There are two main mechanisms for the spontaneous resolution of acute subdural haematomas:

1. Dilution in subarachnoid space- The hematoma may be diluted by flow of cerebrospinal fluid through the arachnoid tear, followed by retrograde flow into the subarachnoid space[1]

2. Redistribution of the haematoma in the subdural space- Brain swelling, caused by cerebral contusion, compresses the hematoma and may contribute to redistribution[9]

In addition, the accompanying fracture might have facilitated the redistribution of the ASDH and improvement of the brain shift. This process may be explained by the prolapse of dura mater into the fracture sites by the force of the SDH (11)

The NCCT scan finding of “hypodensity on clot,” suggesting mixing of CSF, was found to be an important predictor for rapid resolution of Acute SDH. Since resolution of Acute SDH is a dynamic process, CT scan done at different stages of dissolution of hematoma may show different patterns of hypodensity of the clot, and these may further help in identification of patients with the possibility of rapid resolution of Acute SDH[8]

CONCLUSION:

Purpose of this case report was to make neurosurgeons aware to give chance for spontaneous resolution of acute sdh with dearranged coagulopathy before surgical intervention. As it will save patients from unnecessary Surgery and its complications.

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