Chronic Subdural Haematoma: Aeromedical Disposition

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Abstract: Traumatic closed head injury and subsequent subdural lesions like chronic subdural hematoma (CSDH) carry a high risk of recurrence and post-traumatic seizure (PTS). This is particularly significant for an aircrew as sudden incapacitation due to inflight seizure can have disastrous consequences. This article reviews various policies and aeromedical consideration on chronic subdural hematoma for determining fitness of an aircrew for flying in civil and military aviation.

Keywords: Chronic Subdural Haematoma, Aeromedical, Aviation, Disposition

1. Case History

55 yrs old civil helicopter pilot with 3485 hrs of flying experience reported at Medical Evaluation Centre at Institute of Aerospace Medicine IAF for renewal class – I medical assessment. Upon review of his medical history, the pilot revealed that he had sustained multiple musculoskeletal injuries (compression fracture L1, Multiple fracture transverse process D11- L3, undisplaced hairline fracture iliac bone, friction burn left arm and forearm with Radial nerve palsy) following crash landing in Aug 2012 for which he was managed at a military super-speciality hospital. According to first responders, he had questionable loss of consciousness (LoC), but was definitely mildly lethargic, which cleared over the course of his visit to local emergency department following crash landing. He was airlifted to Army Hospital (R&R) for further evaluation and treatment. He was alert during transfer without further alteration of consciousness, and his total alteration of consciousness was estimated to be greater than one hour but less than 24 hours. He appeared to have retrograde amnesia, initially not recalling the event. The time course of his amnesia was not clear but appeared to be concurrent with the period of alteration of consciousness. GCS score was 12/15. He was discharged after 03 weeks of institutional treatment. His hospital records revealed that a month of initial treatment he reported back to the Hospital with headache. On evaluation he was diagnosed to have chronic Subdural Hematoma (SDH). He underwent craniotomy for removal of haematoma (burr-hole evacuation). Post-op period was essentially uneventful. Past medical, personal and family history were not contributory. There was no history of any medication.

He was evaluated at Air Force Central Medical Establishment (AFCME) in Nov 12 for Class-I Medical Assessment and was assessed temporary unfit for duration of 04 weeks. At the end of this period he reported to IAM IAF for fitness for Commercial Helicopter Pilot License (CHPL).

2. Chronic Subdural Hematoma

Subdural lesions occurring after trauma is typically classified into - Acute Subdural Hematoma (ASDH), Chronic Subdural Hematoma (CSDH) and Subdural Hygroma (SDG). It is postulated that CSDH usually develop from ASDH and SDG, mostly SDG (1). Although etiology and location of these lesions are common, they have quite different clinical and radiological features. It is postulated that slow leakage of blood in subdural space from a torn bridge vein cause CSDH in aged and alcoholics due to cortical atrophy, SDG is believed to develop when there’s sufficient subdural space (subdural space is not present in normal condition) produced by any pathological condition (even minute trauma can separate dura- arachnoid space) leading to cleavage of tissue within the dural border cell layer which induce proliferation of dural border cells layer with production of neomembrane. Once neomembrane is formed, hyperpermeable capillaries follow with time. If the absorption takes time, hemorrhage into the subdural fluid would occur either by tearing of bridge veins or bleeding from neomembrane. The majority of SDG are asymptomatic and most SDG will disappear when the brain expansion or absorption exceeds effusion (1). CSDH differs from SDG in many aspects, such as the contents of subdural fluid, radiological appearance and clinical symptoms. However, an absolute distinction between SDG’s and CSDHs is often difficult, since the subdural fluid within SDG is frequently a mixture of blood and CSF.

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When violent shaking or impact injury causes shearing of the bridging veins (right), the subdural space fills quickly with blood (contemporary Paediatrics).

**Etiopathogenesis**

The most commonly accepted mechanism of injury is a blow to the head. It is suspected that this happens because the brain moves freely in the cerebral spinal fluid and the venous sinuses are fixed within the skull. Therefore, when trauma occurs, displacement of the brain can tear these vessels, causing intracranial bleeding.

SDH is most commonly seen in infants and elderly. In infants the brain is quiet compressible and in the elderly brain atrophy leads to potential space, where any fluid can be easily collected (2). Incidence of TBI in India is 80 - 320 lac person-years and four major causes are Road Traffic Accidents (RTA), fall, violence and fall of objects (3). A significant number of these TBI cases are expected to lead to CSDH; however, authors couldn’t find any study which brings out its incidence. It is also associated with consumption of drugs like anticoagulants which prolongs bleeding time or long term alcohol abuse which causes brain atrophy.

Nearly a half of patients with CSDH have no history of head injury, and CSDHs are seen only uncommonly following severe brain injury. ASDH may be produced from CSDH by a repeated trauma or operative removal of CSDH. Patients with CSDH are prone to repeated accidents. CSDH with recent bleeding is not rare. SDGs also occur after operative removal of CSDHs. Thus, all three traumatic subdural lesions may change into another lesion under certain condition (1).

**Clinical Presentation**

CSDH is usually insidious in onset and hence, may not present with any symptoms long time before it is recognised. Traditionally subdural hematomas are classified as acute, sub-acute and chronic subdural hematomas according to the time interval. Temporal classification of SDH by time duration is quiet confusing and arbitrary. The intervals used to distinguish between acute, sub-acute and chronic vary widely according to the author (4). A CSDH is characterised by well-formed inner and outer membranes and regardless of the time interval when the capsule is confirmed surgically, the haematoma should be regarded as chronic (5). Typical neurological signs are due to pressure exerted on the adjacent brain. Many times a dilated pupil will be found on the same side of the head as the lesion. Often, neurologic symptoms and signs are non-focal, including headache and confusion (2). Signs of weakness, numbness, inability to speak, slurred speech, or abnormal level of consciousness should prompt a brain imaging study such as a CT scan or MRI. Level of consciousness may also alternate between stupor and lucidity (6).

**Diagnosis**

Definitive diagnosis of CSDH is by imaging (6). Concave sign with midline shift is pathognomonic for SDH. Various density and intensity patterns are detected with CT and MR imaging, but most CSDHs are mixed density and predominantly hyperintense on T1W images. MR imaging is more accurate than CT when delineating the extent and determination of membranes of CSDHs, and with it hypodense CSDHs can be differentiated from other subdural collections. Small and isodense CSDHs are better evaluated by MR. Mixed density CSDHs on CT are generally layered and membranedhaematomas and the
internal structures can be clearly shown by MR imaging (7).

Treatment

A subdural hematoma can be an emergency condition. Treatment goals include lifesaving measures, control of symptoms, minimizing or preventing permanent brain damage, and possibly breathing and/or circulatory support. Medications may be used, given the symptoms and the extent of brain damage. Diuretics may reduce swelling. Anticonvulsants can assist in seizure control. Emergency surgery may be warranted to reduce intracranial pressure. Typically, a burr hole is drilled in the skull to allow drainage of the hematoma. Large hematomas or solid blood clots may require craniotomy (6).

Figure 3: Concave sign with midline shift in SDH

Prognosis

This varies widely, depending on the mechanism of head injury, the size of the subdural hematoma, and how rapidly treatment is begun. Acute subdural hematomas have high rates of death and injury. Sub-acute and CSDHs generally have good outcomes, with symptoms going away after drainage of the blood collection. Unfortunately, seizure frequency is high following a SDH (6). Although these can be controlled with medication, seizures present a much more grave problem for the aircrew.

Slow, progressive neurological deterioration may occur with re-bleeding. Some important sequelae include post-traumatic epilepsy, brain tumors, infections, and psychiatric disorders, as well as post-traumatic dementia due to repeated head trauma (2). Some research shows evidence of ischemic brain damage as sequelae to acute SDHs as acute on CSDHs are not very uncommon; however, most of the ischemic brain damage resulted from arterial compression due to herniation of the brain and brain shifting and not due to hematoma effect on the underlying brain tissue (8).

Aeromedical Considerations

Head injury does not necessarily mean brain injury. Whenever there’s loss of consciousness (LOC), any neurological deficit or abnormal imaging traumatic brain injury (TBI) is likely. Post-traumatic amnesia (PTA) of any duration and Glasgow coma scale (GCS) are predictor for TBI. There are three main aeromedical considerations for TBI: Permanent Neurological Deficit (PND), Post-Traumatic Syndrome (PTS) and risk for sudden incapacitation e.g. post traumatic epilepsy (PTE), seizures. PND may manifest in variety of ways which include impaired motor, cognitive or language function. PTS is a symptom complex which may include impaired concentration and behavioural changes, irritability, vertigo and more. Careful history taking, examination and follow up by family members and friends can rule out any PND and PTS. Hence, aeromedical consideration for PND and PTS post- TBI in an aircrew is relatively straight forward depending on the history, residual disability and clinical presentation.

Sudden incapacitation, mainly by seizure, is the main concern for any aircrew being considered for fitness for flying post TBI. Immediate or impact seizures are typically not considered a risk factor for subsequent future seizures. However, early onset seizures, happening post one- to two weeks post- event, suggest scar formation or gliosis, and are correlated with risk for future seizures. Late- onset seizures typically occur two or more weeks post-event and may also be due to scar formation; however it may take several years for the first seizure to manifest (9). Authors could find only one study which looked into the risk of seizure post CSDH. This is a retrospective study where 100 CSDH cases were followed up for 18 months. This study brought out that incidence of acute symptomatic seizure was 6% and unprovoked seizure 5% where most occurred after 03 mths (10).

The main question is: What seizure-free interval is sufficient following mild, moderate and severe closed-head injury to consider the risk of seizure low enough? The standardized incidence ratio of seizure (SIR) for mild closed-head injury is 1.5 overall, with no increase over expected number after 5 years. The SIR for moderate closed head injury is 2.9 overall and 6.7 for the first year following injury. The SIR for severe closed-head injury is 17 overall, 95 for the first year (11). In another study relative risk of initial seizure post TBI as per severity of head injury in first 5 years is as shown in Table 1 (12).

Table 1: Relative risk of initial seizure following traumatic brain injury

<table>
<thead>
<tr>
<th>Grades of TBI</th>
<th>Relative risk after 1 yr</th>
<th>2-4 yrs</th>
<th>5 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>3.1</td>
<td>2.1</td>
<td>0.9</td>
</tr>
<tr>
<td>Moderate</td>
<td>6.5</td>
<td>3.1</td>
<td>3</td>
</tr>
<tr>
<td>Severe</td>
<td>96.9</td>
<td>17.4</td>
<td>12.2</td>
</tr>
</tbody>
</table>

Most neurological recovery occurs in 6-12 months. In 1-2 years, further recovery is expected but to a lesser extent. Fifty percent of individuals in whom seizures will occur will experience it within the first 6 months following the
brain injury, 75% within 1 year, 90% within 2 years, and 97% within 3 years (9).

Another factor particular to CSDH is its propensity for recurrence and possibility of subtle signs and symptoms which may not be recognized easily during the initial stages. Various studies have reported recurrence rates ranging from 9.2 to 26.5% (13, 14, 15). Various risk factors for recurrence of CSDH have been reported in studies, including advanced age, cerebral atrophy, bleeding tendency, chronic alcohol intake, bilateral location of hematoma and postoperative pneumocephalus (16, 17, 18). Imaging findings may also predict possibility of recurrence of CSDH. The high-density and isodense types of CSDHs on CT have been reported to have higher recurrence rates than the low-density types (19). Recurrence rates are higher during first few months of surgical treatment of CSDHs which diminishes drastically with time. Hence, observation period for PTS in CSDHs will safeguard against recurrences of CSDH as well.

### Aeromedical Disposition

All closed head injuries are classified as mild, moderate or severe to determine the medical course of action necessary to recertify the aircrew. According to Indian Air Force Publication (20), head injury is graded based on Loss of consciousness (LOC) and/or Post- traumatic amnesia (PTA), CT/ MRI findings, EEG, psychometry, neurological and neuropsychiatric evaluation and Glasgow Coma Scale (GCS) immediate post injury. Table 1 compares the various criteria to grade head injury world over and their permissible disposal based on objective findings. Indian Air Force criteria appears to be the most stringent where multiple criteria is used to grade the severity of head injury whereas most of the other regulatory agencies use only LOC and PTA as defining criterion.

### Table 2: Grading of head injuries and their disposal by various agencies

<table>
<thead>
<tr>
<th>Agency</th>
<th>TBI</th>
<th>LOC</th>
<th>PTA</th>
<th>Any Other Criteria</th>
<th>Min Observation</th>
</tr>
</thead>
<tbody>
<tr>
<td>USAF</td>
<td>Mild</td>
<td>&lt; 30 min</td>
<td>&lt; 60 min</td>
<td>Non displaced Skull #</td>
<td>1mth</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>1/2h-24 h</td>
<td>1h-24 h</td>
<td>Subdural haematoma, Brain contusion, Structural injury</td>
<td>5 yrs</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>&gt; 24 h</td>
<td>&gt; 24 h</td>
<td>ICH/ haematoma and LOC &gt; 24 h</td>
<td>1 mth</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 15 min</td>
<td>&lt; 12 h</td>
<td>Permanently Unfit</td>
<td>3 mth</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>¼- 2 h</td>
<td>12- 24 h</td>
<td></td>
<td>2 yrs</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>2- 24 h</td>
<td>&gt; 24 h</td>
<td>Non depressed skull #</td>
<td>2 yrs</td>
</tr>
<tr>
<td>USA</td>
<td>Mild</td>
<td>5- 60 min</td>
<td>5- 60 min</td>
<td>Any Intracranial bleeding : Unfit permanently</td>
<td>After work up</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>1- 24 h</td>
<td>1- 24 h</td>
<td></td>
<td>12 mths</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>&gt; 24 h</td>
<td>&gt; 24 h</td>
<td></td>
<td>30 mths</td>
</tr>
<tr>
<td>FAA</td>
<td>Mild</td>
<td>&lt; 1 h</td>
<td>&lt; 1 h</td>
<td></td>
<td>6 mth</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>1- 24 h</td>
<td>1- 24 h</td>
<td></td>
<td>6 mth</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>&gt; 24 h</td>
<td>&gt; 24 h</td>
<td></td>
<td>4- 5 yrs</td>
</tr>
<tr>
<td>IAF</td>
<td>Mild</td>
<td>&lt; 1 h</td>
<td>&lt; 1 h</td>
<td>CT/MRI; EEG, Psychometry, Neurological and Neuropsychiatric evaluation: Normal GCS &gt; 13</td>
<td>6 mth</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>1- 24 h</td>
<td>1- 24 h</td>
<td>CT/MRI: Linear # skull outer table only. Mild cerebral contusion/ edema. No intracranial lesion, EEG/ Psychometry: Normal/ Abnormal. Neurological and Neuropsychiatric evaluation Normal. GCS 9- 13</td>
<td>1 yr</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>&gt; 24 h</td>
<td>&gt; 24 h</td>
<td>CT/MRI: Depressed skull # or contusion brain or ICH/hematoma, Penetrating Wound CSF Rhinorrhoea/ Otorrhoea, Neurological deficit, EEG/ psychometry/ Neuropsychiatric evaluation: abnormal GCS &lt; 9</td>
<td>1 yr (No fighters)</td>
</tr>
</tbody>
</table>

All military regulations of leading armed forces of the world consider any intracranial bleed as severe head injury and, hence, most of them debar them from fighter flying (Table 1). However, civil regulatory bodies like FAA consider any mild or moderate grade head injury associated with intracranial bleed- subdural, epidural or intracerebral bleed- after a minimum of 2 years of observation, off medications with free from seizures. A severe head injury requires 5 years free of seizures while off medications for at least the last 2 years (21).
Indian Air Force does not permit any candidate with history of severe TBI for flying duties during initial entry. Trained aircrew with severe TBI is not allowed to fly fighter post severe head injury. All cases of TBI are observed in ground category for at least 06 months. Mild and moderate TBI cases are gradually upgraded to full flying categories including fighters, however, moderate TBI cases are at least propensity of its recurrence and factors predicting it must be taken into account observed for a year before upgrading them to full flying category. Severe TBI cases are upgraded gradually to A2G2 (P) after 48 wks of observation in ground category- fit only for transport and helicopters (20). Indian Air Force publication does not talk about CSDH in particular but as per definition it is considered as severe TBI.

Even though none of the regulatory bodies talk of CSDHs specifically, propensity of its recurrence and factors predicting it must be taken into account before deciding on fitness of an aircrew with history of CSDH.

Case Disposal

The pilot was fully evaluated and he was found fit with regards to other disabilities except for consideration for PTS and recurrence of CSDH. The pilot was declared permanently unfit for flying duties. He may be considered fit after an observation period of 4 to 5 yrs. On presentation, pilot was not on any anti-seizure medication. In case of pilot being put on any Anti-epileptic drug (AEDs) therapy, he will be required to be drug free seizure free for at least 2 yrs before being considered for fitness for flying (20).

References