

# Histopathological study of the outer membrane of the dura mater in chronic sub dural hematoma: Its clinical and radiological correlation

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## ABSTRACT

**Background:** A chronic subdural hematoma is an old clot of blood on the surface of the brain between dura and arachnoid membranes. These liquefied clots most often occur in patients aged 60 and older with brain atrophy. When the brain shrinks inside the skull over time, minor head trauma can cause tearing of blood vessels over the brain surface, resulting in a slow accumulation of blood over several days to weeks.

**Aim of the Study:** To evaluate the role of membrane in hematoma evaluation and to correlate its histopathology with clinic-radiological aspects of the condition and overall prognosis of patients.

**Material and Methods:** The study incorporated all cases of chronic SDH admitted to the Neurosurgery department of JLN Hospital and Research Centre, Bhilai, between November 2011 and November 2013. All such cases were analyzed clinically, radiologically like site, size, thickness in computed tomography, the attenuation value, midline shift and histopathological features were recorded.

**Criteria for Inclusion:** All cases of chronic subdural haematoma irrespective of age and sex were incorporated into the study.

**Criteria for Exclusion:** All cases of acute subdural haematoma and cases of chronic sub dural hematoma which were managed conservatively irrespective of age and sex were excluded from the study

**Results:** In our series of cases, the most common histopathological type of membrane was the inflammatory membrane (Type II) seen in 42.30% of cases followed by hemorrhagic inflammatory membrane (Type III) seen in 34.62% of cases while scar inflammatory type of membrane (Type IV) was seen in 23.08% of cases. No case with noninflammatory type (Type I) was encountered.

**Key words:** Chronic sub dural hematoma, histopathology, Glasgow coma scale, hounsfield unit

## Introduction

Chronic subdural hematoma (SDH) starts as a flat blood clot between the dura and the arachnoid membrane. Initially, it is not attached to the dura. Fibroblasts, growing from

the dura into the clot, organize it. In 5-6 days, fibroblast growth causes the blood clot to be loosely attached to the dura. In 10-20 days, a loose fibrous membrane is formed between the dura and the clot (outer membrane). Fibrous tissue then grows around the edges of the hematoma and along its inner surface (inner membrane), encapsulating it completely. Maturation of connective tissue results, after several weeks or months, in the formation of a sac with a fibrous wall (chronic SDH). Blood in this sac is absorbed to a variable degree, and the cavity contains clear or hemorrhagic fluid and a loose, vascular connective tissue. Rupture of delicate vessels may cause repeated bleeding in the sac. Fluid may also leak into the cavity from immature capillaries. If a large amount of cerebrospinal fluid enters the subdural space during the traumatic event, it washes off the blood, and no clotting or organization takes place. The histological appearance of the sac is helpful in estimating the duration of the SDH.

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## Case Study

Fifty-two cases of chronic subdural hematoma (SDH) were evaluated on the basis of their clinical, radiological and histopathological features between November 2011 and November 2013 in the Department of Neurosurgery of JLN Hospital and Research centre, Bhilai.

## Age and Sex Distribution

In the study, there were 40 males and 12 females. Male preponderance among cases was as high as 3.33:1. This may be due to (1) More exposure of the male population to injuries than females. (2) Females are seeking less of medical advice.

## Clinical Presentation

In our study, most common symptom at presentation was headache which was observed in 66.67%, altered level of consciousness in 28.84%, vomiting in 25%, vertigo and giddiness in 8.33%, blurring of vision in 1.92%, urinary and fecal incontinence in 13.46%, hemiparesis in 26.91%, paraparesis and quadriparesis in 1.92% each, papilledema in 5.76%, 3<sup>rd</sup> and 6<sup>th</sup> nerve palsy in 1.92% each and 7<sup>th</sup> nerve palsy in 3.84%, 92.30% of patients had Glasgow coma scale (GCS) between 13 and 15 while only 3.85% of patients had GCS  $\leq$  8.

## Risk Factors

A history of head injury was elicited in 66.02% of cases, clinical history of chronic alcoholism in 3.84%, diabetes mellitus and hypertension in 3.84% and 5.76% of cases respectively.

## Radiological Features

In our series, most common site of chronic SDH was frontotemporoparietal region (58.34%), next most common site being frontoparietal (13.46%).

About 87.18% of cases had unilateral while 12.82% of cases had bilateral hematoma. Hematoma is slightly more common on the right side when compared to the left side.

## Histopathological Features

In our study, the most common histopathological type of membrane was the inflammatory membrane (Type II) seen in 42.30% of cases followed by hemorrhagic inflammatory membrane (Type III) seen in 34.62% of cases while scar inflammatory type of membrane (Type IV) was seen in 23.08% of cases. No case with noninflammatory type (Type I) was encountered.

## The following conclusions were drawn from the study

- Incidence of chronic subdural hematoma is more in males as compared to females. Male female ratio was 3.33:1

- Higher percentage of cases occurred in >50 years of age groups
- Most common symptom at presentation was headache (66.67%) while 35.89% of cases complained of weakness of one side of body which was of sudden onset and with varying severity and improved after the evacuation of chronic subdural hematoma
- History of head injury could be obtained only in 66.02% of cases while in remaining 33.98% of cases they could not remember any episode of trauma at all
- Most of the patients (92.30%) at the time of presentation had GCS between 13 and 15
- Chronic SDH had more predilection toward frontotemporoparietal region (58.34% of cases)
- Hypodensity on computed tomography (CT) scan was seen in 82.70%, and midline shift was present in 53.85% of cases

Table 1: Age and sex distribution

| Age         | Male | Female | Total | Percentage |
|-------------|------|--------|-------|------------|
| 30-40 years | 2    | 1      | 3     | 5.76       |
| 41-50 years | 12   | 4      | 16    | 30.76      |
| >50 years   | 26   | 7      | 33    | 63.48      |
| Total       | 40   | 12     | 52    | 100        |

Table 2: Symptoms at presentation

| Symptoms                       | Number of patients | Percentage |
|--------------------------------|--------------------|------------|
| Headache                       | 35                 | 66.67      |
| Fully oriented                 | 39                 | 75.64      |
| Altered sensorium              | 15                 | 28.84      |
| Vomiting                       | 13                 | 25.00      |
| Weakness of one side of body   | 19                 | 35.89      |
| Vertigo and giddiness          | 4                  | 8.33       |
| Blurring of vision             | 1                  | 1.92       |
| Urinary and fecal incontinence | 7                  | 13.46      |

Table 3: Risk factors

| Risk factors       | Number of patients | Percentage |
|--------------------|--------------------|------------|
| Head trauma        | 34                 | 66.02      |
| No head trauma     | 18                 | 33.98      |
| Alcohol abuse      | 2                  | 3.84       |
| Hypertension       | 3                  | 5.76       |
| Diabetes mellitus  | 2                  | 3.84       |
| Drug intake        | 1                  | 1.92       |
| Bleeding disorders | 0                  | 0          |

Table 4: GCS

| GCS   | Number of patients | Percentage |
|-------|--------------------|------------|
| <8    | 2                  | 3.85       |
| 9-12  | 2                  | 3.85       |
| 13-15 | 48                 | 92.30      |

GCS – Glasgow coma scale

**Table 5: Side of the lesion**

|            | Number of patients | Percentage |
|------------|--------------------|------------|
| Unilateral | 45                 | 87.18      |
| Right      | 26                 | 58.08      |
| Left       | 19                 | 41.92      |
| Bilateral  | 7                  | 12.82      |

**Table 6: Histopathological findings**

|          | Number of patients | Percentage |
|----------|--------------------|------------|
| Type I   | 0                  | 0          |
| Type II  | 22                 | 42.30      |
| Type III | 18                 | 34.62      |
| Type IV  | 12                 | 23.08      |

- Most common type of membrane encountered was the inflammatory membrane (Type II)
- Patients with clinically severe manifestations as measured on GCS (<8) had inflammatory type of membrane (Type II)
- Increased radiodensity measured by Hounsfield unit was encountered mostly in Type II cases, followed by Type III and Type IV cases
- Larger size (>2) of the hematoma measured by the thickness of hematoma in centimeters was seen in Type II cases
- Midline shift was seen most commonly in cases with inflammatory type of membrane (Type II).

## Recommendations

Histopathological typing of outer membrane in chronic subdural hematoma is recommended for following reasons.

- Membrane study acts as a rough guide to estimate the time since injury
- A retrospective analysis of the patient's clinical condition can be made if membrane report is known (specially with Type II and III membranes)
- Membrane study helps in correlating the clinical and radiological parameters of a case of chronic subdural hematoma
- Role of membrane in hematoma evaluation can be roughly assessed
- Histopathological study of the membrane widens/completes the spectrum of chronic SDH in terms of severity of disease and overall prognosis of patient [Tables 1-6].

## Review of Literature

### Historical background

Collection of blood under the dura mater has been recognized for about 3000 years. The first description of a chronic SDH was made in 1658 by J. J. Wepfer, followed in 1761 by Morgagni. A possible case was described by Honore de Balzac in 1840 including its traumatic origin and surgical treatment.<sup>[1]</sup>

Virchow, in 1857, denied a traumatic origin, and gave the name of "pachymeningitis hemorrhagica interna" to this pathology, which he explained by inflammatory processes. The traumatic etiology of chronic SDH was recognized in the 20<sup>th</sup> century, especially by Trotter in 1914. Drake has produced fairly convincing evidence to prove that the famous Austrian musician Wolfgang Amadeus Mozart who lived in the 18<sup>th</sup> century suffered from a chronic subdural hematoma, which was not recognized.<sup>[2]</sup>

### Etiology

The origin of blood accumulation within the subdural space is usually traumatic caused either by direct or indirect trauma to the cranium, such as acceleration injuries with tearing of the parasagittal bridging veins or Mittenzweig's vessels, by movement of the brain in relation to its coverings [Figure 1].

### Nontraumatic causes of subdural hematoma

- Bleeding diathesis secondary to use of anticoagulant drugs for cardiovascular diseases<sup>[3]</sup>
- Malignancy-meningeal carcinomatosis or sarcomatosis<sup>[4]</sup>
- Traumatic subdural hydroma<sup>[5,6]</sup>
- Associated with arachnoid cyst<sup>[7]</sup>
- Hemodialysis<sup>[8,9]</sup>
- Associated with cerebral aneurysm.<sup>[10]</sup>

### Histology of Subdural Membrane

Sato *et al.* studied the membrane and found that neomembrane consisted of elastic-hard, hypertrophic granulation tissue and yellowish, sticky fluid in the lumen, was readily freed and totally extirpated.<sup>[11]</sup> Light microscopic examination detected the sinusoidal channel layer and the fibrous layer in an alternating configuration, along with intramembranous hemorrhagic foci. Such hypertrophy must have been caused by repeated intramembranous hemorrhages and reactive granulation.

Nagahori *et al.* studied the histological nature of outer membrane of chronic subdural hematoma [Figure 2]. Outer membrane was examined by hematoxylin and eosin staining, and elastic van Gieson staining.<sup>[12]</sup>

Histological features were classified into four types according to maturity and intensity of the inflammatory reaction and hemorrhage:

Type – I (noninflammatory membrane) [Figure 3].

- This membrane containing immature fibroblasts and collagen fibers was associated with very slight or sparse cell infiltration and neocapillaries.

Type – II (inflammatory membrane) [Figure 4].

- This type consisting of one layer of immature connective tissue was associated with marked cell infiltration and vascularization throughout the entire thickness.

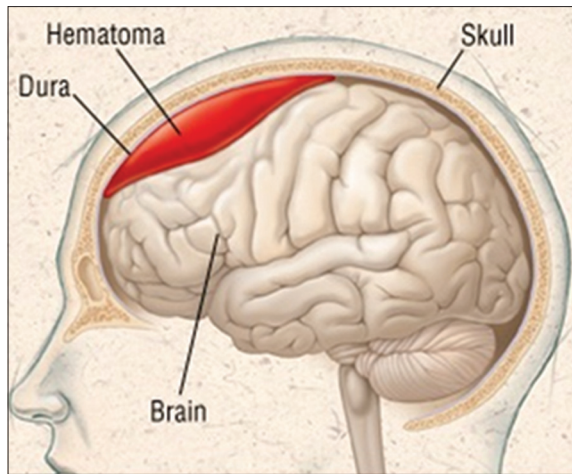


Figure 1: Diagrammatic depiction of subdural hematoma

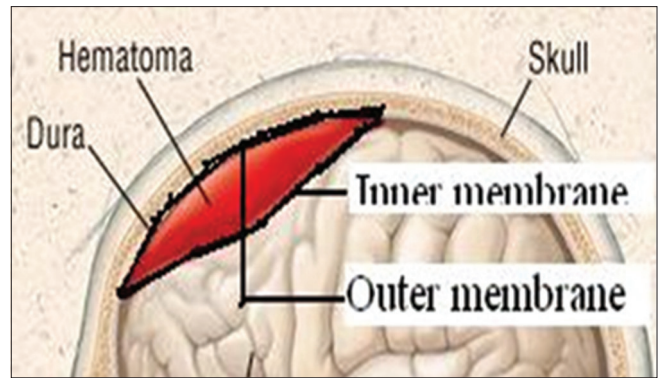


Figure 2: Depiction of outer and inner membranes

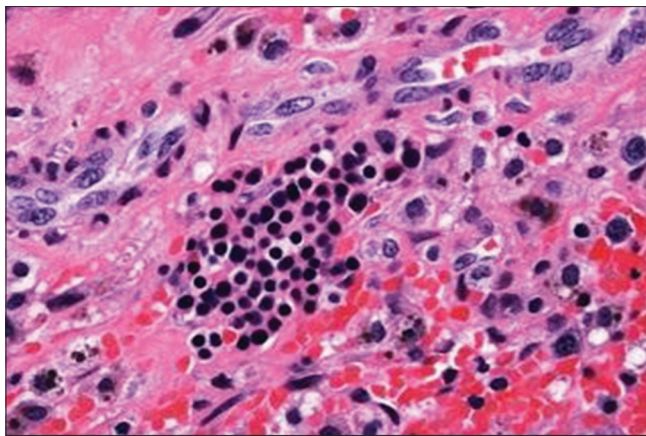


Figure 3: Type I membrane (high-pressure freezing view)

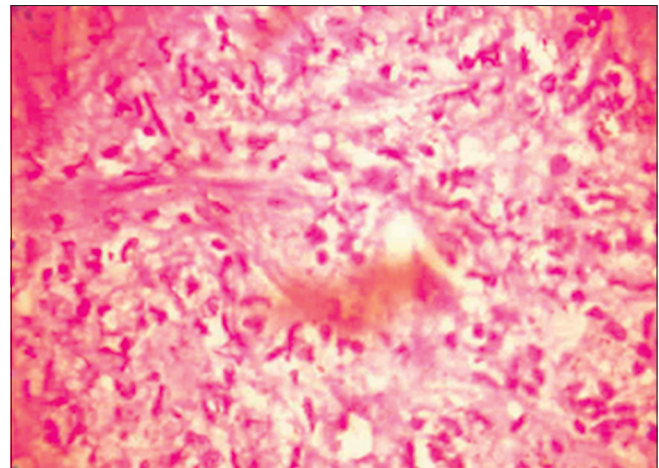


Figure 4: Type II membrane

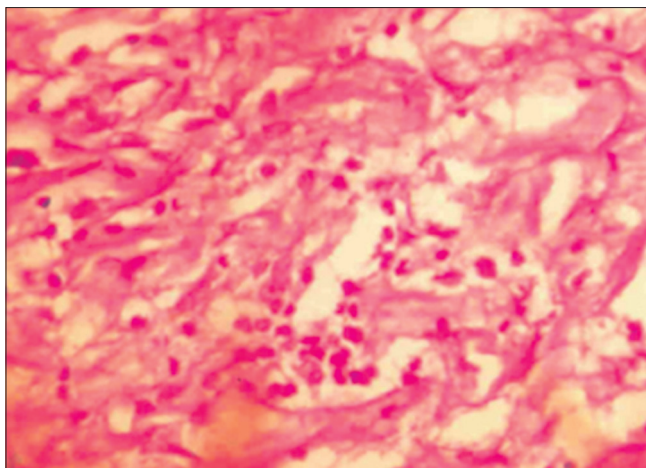


Figure 5: Type III membrane

Type – III (hemorrhagic inflammatory) [Figure 5].

- This type had a structure of 2 or 3 layers and was associated with capillaries with large lumen on the side of the duramater and marked cell infiltration and many thin new vessels on the side of hematoma cavity. Sometimes a layer consisting of only collagen

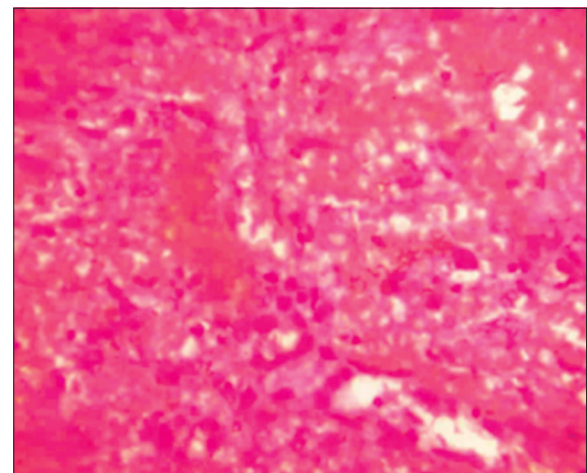


Figure 6: Type IV membrane

fibers and fibroblasts between two such layers was seen. In addition, hemorrhage into the membrane was often observed.

Type – IV (scar inflammatory membrane) [Figure 6].

- This type showed inflammatory cell infiltration, neovascularization and hemorrhage in the outer membrane of cicatricial tissue.

## Location

Chronic subdural hematoma is mostly supratentorial in location, generally situated over the convexity of one or both hemisphere. In about 10–15% of cases they are bilateral. Rarely such hematoma may occur over the vertex, subfrontally or under the temporal lobe. Rare sites are posterior fossa, spinal subdural hematomas,<sup>[13]</sup> interhemispheric region and cerebellopontine angle.<sup>[14]</sup>

## Age and Sex Incidence

Chronic subdural hematoma is common during the 1<sup>st</sup> year of life in the middle aged and elderly people. The incidence is 5 times more frequent in males than in females.

## Common presentations

- Headache
- Epilepsy
- Altered mental state
- Transient neurological deficits
- Focal neurological deficits.

## Uncommon presentations

- Generalized chorea<sup>[15]</sup>
- Unilateral spatial neglect<sup>[16]</sup>
- Parkinsonism<sup>[17]</sup>
- Gerstmann's syndrome<sup>[18]</sup>
- Contralateral dystonia<sup>[19]</sup>
- Isolated oculomotor palsy<sup>[20]</sup>
- vertigo and nystagmus<sup>[21]</sup>
- Korsakoff syndrome.<sup>[22]</sup>

## Investigations

- CT scan
- Magnetic resonance imaging
- Plain X-ray-detectable skull fracture.<sup>[3]</sup>

## Treatment

- Burr hole craniostomy with or without closed system drainage
- Craniotomy and membranectomy
- Percutaneous subdural tapping
- Twist drill aspiration
- Subtemporalis marsupialization
- Endoscopic evacuation
- Replacement of hematoma cavity with oxygen or carbon dioxide
- Subdural peritoneal shunt
- Exteriorization of the subdural pocket
- Omayya cerebrospinal fluid reservoir
- Hollow screw method
- Reduction cranioplasty
- Single needle trephination with open system drainage.

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