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Chronic Subdural Haematoma in Liver Disease Patients with Coagulopathy, Local Experience

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Abstract

Background: hepatitis is one of endemic diseases in Egypt represent in almost 15-18% of population, beside bilharzial preportal fibrosis with its sequelae of thrombocytopenia and bleeding disorders making a treating of chronic subdural haematoma challenging neurosurgeon for getting best results especially no guidelines to follow.

Aim of the work: to evaluate the surgical results clinically, radiologically and efficacy of rapid correction of coagulopathy and platelets for evacuation of chronic subdural hematoma (CSDH) in prospective controlled manner.

Material and method: it includes two groups: group A which had bleeding disorder due to liver disease (hepatitis & bilharsiasis) 75 cases and the other group B which is normal control group 150 cases) in the period from March 2008 till July 2014 with follow up range from 4 months to 4 years. The surgical procedure was done under general anesthesia by two burr holes' evacuation. Clinical, neurological examination and Computed topography (C.T.) was done for all cases pre & post-operative, serial follow ups. The discharge from the hospital range from 5 days to 10 days according to the condition of the patient.

Results: The mean age was 61,3 years old ± 15,7 collectively in both groups, male predominance 173 cases (76.9%) while female were 52 (23.1%). The hematoma was located over the left or right convexity in 40.2% and 37.2%, respectively. A bilateral hematoma was noted in the remaining patients (22.6%), main symptoms & signs in both groups: headache, hemiparesis, gait abnormalities, dementia. Haematological correcting protocol in group A improve INR ≤ 1.6 and platelets ≥ 80000 in 68 cases (90.7 %) intraoperatively lead to Excellent & Good post-operative result of 82.6% in group A compared to 93.3% in group B. Fair and poor outcome associated in our series with old ages, hypertension and INR ≥ 1.6 & platelets ≤ 60000

Conclusion: good hemostasis and correction of associated coagulopathies is the cornerstone of satisfactory results in chronic liver disease patients with chronic subdural haemorrhage. Increased awareness especially in developing countries is therefore important to ensure proper follow-up and early intervention.

Keywords: *Chronic Subdural; Coagulopathy; Disease; Patients; Local Experience; Neurosurgery; Hypothesis; Anesthesia*

Introduction

Chronic subdural hematoma (CSDH) is an intracranial hemorrhage that is still associated with significant morbidity. Although it is a well-known entity and daily neurosurgery practice, with clearness of its cause, clinical picture and surgical management, still some condition related to elderly (who is commonest group to be affected) not have the same known guidelines in the management. Egypt has the highest prevalence of chronic hepatitis C infection worldwide that affect nearly 15-18% of population with cirrhosis as a sequence [1]. In addition, the effect of bilharzial preportal fibrosis complicated by hyper spleenism in some cases developed thrombocytopenia. These

associated co morbid conditions; especially the case came with delayed diagnosis and deterioration in conscious level and neurological function making us in critical situation between urgency of evacuation versus control of coagulopathy and platelets dysfunction in reasonable time. However, some patients developed recurrence after surgery, with an incidence as variable as 3.7 to 30%. Numerous factors responsible for recurrence can be seen in the literature especially in chronic liver disease patients [2].

Our hypothesis is that correction of coagulopathy by perioperative trantransfusion of fresh frozen plasma and platelets can reduce the recurrence of CSDH in chronic liver diseased patients with Chronic subdural hematoma.

Material and Method

This study is to evaluate the surgical results clinically, radiologically and efficacy of rapid correction of coagulopathy and platelets for evacuation of chronic subdural hematoma (CSDH) in prospective controlled manner between two groups: group A which had coagulopathy and platelets deficiency in number and function (50 cases) and the other group B which is normal control group (100 cases) in the period from March 2008 till July 2014 with follow up range from 4 months to 4 years.

In both groups the surgical procedure was done under general anesthesia by two burr holes evacuation and the irrigation with isotonic saline then closed tube drainage was applied for 48 hours postoperatively then removed.

Computed topography (C.T.) was done for all cases as diagnostic tool and post-operative follow up 3 days after then after two weeks unless deterioration of neurological state so was done immediately. The discharge from the hospital range from 5 days to 10 days according to the condition of the patient.

Clinical assessment of the case by Glasgow coma score and motor power grades pre and post-operative the after two weeks, one month, three months then every 6 months.

Inclusions criteria:

1. Thickness of the CSDH more than 10mm.
2. Concomitant relation between neurological state and the haematoma as conscious level and weakness.
3. Controlled comorbid disease as hypertension and diabetes mellitus.
4. Exclusion criteria:
5. Uncontrolled comorbid disease hypertension and diabetes mellitus.
6. Cancer, intracerebral haematoma, renal failure, cardiac or cerebral infarction.

The Group A defined who have coagulopathy as INR ≤ 2,2 AND OR platelets number less than 50000 so we gave them 2 unit of fresh frozen plasma and or two units of platelets super added cyclocapron and vitamin k and steroid by 8 mg/12 hours to decrease hyperspleenism pre-operative, immediate preoperative, intraoperative, immediate post-operative.

The group B patients are with normal variant coagulant profile and platelets number
The result of surgery considered:

a. Excellent if the patient regain pre haematoma neurological condition and C.T reveal complete evacuation of the haematoma,
b. Good if the patient regain pre haematoma neurological condition and C.T reveal more than half size evacuation of the haematoma,
c. Fair if the patient did not regain pre haematoma neurological condition and C.T reveal more than half size evacuation of the haematoma
d. Poor if the patient did not regain pre haematoma neurological condition and C.T reveal less than half size evacuation of the haematoma or COMPLICATED by acute subdural; intracerebral haematoma.

Results

In the whole group of 225 patients: The mean age was 61,3 years old ± 15,7 collectively in both group as the age indifference as a factor, male predominance 173 cases (76.9%) while female were 52 (23.1%). The hematoma was located over the left or right convexity in 40.2% and 37.2%, respectively. A bilateral hematoma was noted in the remaining patients (22.6%).

symptom	Number & % of cases group A (75cases)		Number & % of cases group B (150cases)	
Hemiparesis	46 cases	61.3%	89 case	59.3%
Gait abnormality	40 cases	53.3%	110 case	73.3%
dementia	49 cases	65.3%	87 cases	58%
Headache	67 cases	89%	117 cases	78%
Seizures	11 cases	14.6%	9 cases	12%
Incontinence	8 cases	10.7%	16 cases	10.7%
Dysphasia	5 cases	6.7%	14 cases	9.3%
Decrease in conscious level	42 cases	56%	24 cases	16%

Table 1: *Presenting symptoms (single and combined).*

Hematological profile	Number of cases	%
INR ≤ 1.6 and platelets ≥ 80000	68 cases	90.7 %
INR ≥1.6 & ≤ 2 and platelets ≥ 60000	5 cases	7%
INR ≥2 & ≤ 2.2 and platelets ≥ 50000	3 case	4%

Table 2: *Hematological profile in group A with correction protocol applied intraoperative sample.*

Grade	Number & % of cases group A		Number & % of cases group B	
Excellent	34 cases	45.3%	77 cases	51.3%
Good	28 cases	37.3%	63 cases	42%
Fair	6 cases	8%	7 cases	4.7%
Poor	7 cases	9.3%	3 cases	2%

Table 3: *Outcome after surgical procedure.*

The factor	NO of cases group A & %		NO of cases group B & %	
≥ 70 years	9/13	57%	6/10	60%
Hypertension	10/13	77%	7/10	70%
Diabetes mellitus	4/13	30.8%	3/10	30%
Seizures	8/13	61.5%	4/10	40%
Tension Pneomocephalous	2/13	15.4%	1/10	10%

Table 4: *Factors associated with Fair & poor outcome.*

	NO of cases group A & %		NO of cases group B & %	
Acute SDH	3/7	42.5%		
Recollection of CSDH	2/7	28.6%	1/3	33.3%
Tension Pneomocephalous	2/7	28.6%	1/3	33.3%
Intracerebral haematoma	2/7	28.6%	1/3	33.3%

Table 5: *Post-operative Complication.*

Discussion

The etiology of CSDH mostly due to tear of arachnoid vessel related to head trauma [3,4] leading to leakage of blood with following formation of inflammatory membrane followed by leakage of plasma secondary to osmolality deference especially in elderly due to associated brain atrophy [5,6]. Which make bridging vessels more vulnerable to tear secondary to mild head trauma [7,8]. Guidelines on the management of subdural hematoma are formulated based on traumatic subdural hematoma [9]. So, there is basic difference between the management of CSDH occurring as a consequence of associated hematological disorder and that due to mere trauma.

Severe hepatocellular disease predisposes to hemorrhage due to disturbed blood coagulation as a sequelae of blood coagulation factors deficiency synthesized by hepatocytes, and/or thrombocytopenia. Coagulopathy results from decreased hepatic production of coagulation factors. If cholestasis is present, diminished micelle into the small intestine leads to decreased vitamin K absorption, followed by decrease in hepatic synthesis of factors II, VII, IX, and X. Patients with cirrhosis also may experience fibrinolysis and disseminated intravascular coagulation. Thrombocytopenia usually is secondary to hyperspleenism and decreased levels of thrombopoietin [10].

Our result (Excellent, Good) which is 82.6% in group A and 93.3% in group B is satisfactory when compared with that in literatures which varies from 72% to 95% [11,12]. However, these results based on correction of coagulopathy.

In a review of 253 patients with head trauma who requested CT scans follow up, the risk of developing a delayed brain insult diagnosed by CT scan increased to about 85% in patients with abnormal prothrombin time (PT), activated partial thromboplastin time (aPTT), or platelet count [2] compared to 31% in patients with normal coagulation profile. A patient with coagulopathy should be transfused with fresh frozen plasma (FFP), platelets, or both to maintain the prothrombin time (PT) within the reference range. FFP is only indicated for surgical bleeding when there is coagulopathy. Coagulation still normal unless clotting factors descend below 30% of their normal values or the fibrinogen concentration becomes less than 0.75g per litre^{-1}. Consequently, FFP replacement aims to exceed at least 30% of normal plasma factor values. This can usually be obtained by transfusion of 10-15 ml kg^{-1}; four units of FFP (i.e. near 800 ml) will elevate coagulation factors about 10% [13].

Platelets transfusion essential for thrombocytopenia or platelet function defects. Ideally, the platelet count should not be < 50 × 109 litre^{-1} [14] However, a higher target level (100 × 109 litre^{-1}) may be appropriate for patients with multiple injuries or central nervous system trauma [15]. One unit of platelets (pooled or single donor) should increase the platelet count by 20 × 109 litre^{-1}. However, the actual increase in platelet count also depends upon sequestration in the spleen. The required dose may be calculated as the desired percentage increment × the blood volume × a correction factor (usually 0.33) to account for splenic sequestration. The usual dose in adults is 10-15 ml kg^{-1} (approximately two units). So, the cornerstone of satisfactory results in group A mainly related to given protocol and good hemostasis of scalp and muscle by bipolar and bone sinuses by bone wax while the hemostasis of dural edges by bipolar coagulation.

In a developing country, where proper follow-up is often lacking these problems are likely to be more common and are less announced. Patients with a platelet count of less than 10,000/mm^3 are at risk of developing ICH. SDH usually occurs as an extension of a parenchymal bleed. The basic pathology in thrombocytopenia is capillary leakage and this may become sever leading to frank intracerebral hematoma [16]. Fair and poor outcome associated in our series with old ages which related clearly to brain atrophy and increase subdural space which also related to atherosclerosis and consequence lead to hypertension in 77% of fair & poor group A in comparison of fair & poor group B 70%. Diabetes mellitus had fewer roles in poor result as other series [17].

In all series the anticoagulant associated with poor prognosis [14, 15,18]. in our series the poor outcome also related to INR ≥ 1.6 & platelets ≤ 60000 .The presence of parenchymal intracerebral haematoma following evacuation of CSDH related to two factors is rapid decompression leading hyper perfusion with consequence lead to hemorrhage breakthrough [18]. while other factor is application of sub dural tube which may lead to cortical injury especially by young neurosurgeon which declined by putting it subgaleal instead . Acute

subdural haematoma developed in one case of group A which treated rapidly by craniotomy with supplementation by coagulant and units of platelets but with poor result [19,20].

Tension Pneomocephalous developed in two cases in group A and case in group B which relate to imperfection closed drainage system which treated by evacuation and replaced by isotonic saline.

In conclusion

Good hemostasis and correction of associated coagulopathies is the cornerstone of satisfactory results in chronic liver disease patients with chronic subdural haemorrhage. Increased awareness especially in developing countries is therefore important to ensure proper follow-up and early intervention.

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