Chronic Liver Disease with Alcohol Induced Atrial Fibrillation with Hemorrhagic Stroke: A Case Report

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Abstract: Atrial fibrillation (AF) is a major modifiable factor associated with 4-5 fold increase in ischaemic stroke (1). AF is an independent predictor of mortality and is also associated with poor outcomes among stroke patients (2-4). In CLD patients with decompensated liver failure INR is deranged and is most likely the cause of ICH and this association may be mediated by decreased levels of most procoagulant factors and thrombocytopenia (5). It is also possible that liver disease predisposes to both haemorrhage and thrombosis via a disordered and unstable coagulation system (6). For ICH medical management depends upon: BP, Coagulopathy, Glucose, Temperature, Seizure, ICP.

Keywords: Chronic Liver Disease with Alcohol induced Atrial Fibrillation with Hemorrhagic Stroke

1. Introduction

• Primary intracerebral hemorrhage (ICH), or spontaneous nontraumatic bleeding into the brain parenchyma, constitutes 10% to 15% of strokes.
• ICH causes disability in survivors, with only 20% of patients expected to be functionally independent at 6 months (7)
• Another 15% to 23% of patients demonstrate continued deterioration within the first hours after hospital arrival (8, 9)
• A routine part of the evaluation should include a standardized severity score. The most widely used and externally validated is the ICH Score (10). These severity scales should not be used as a singular indicator of prognosis.

2. Case Report

A 48 years old male patient known case of Atrial Fibrillation with Chronic liver disease and Portal hypertension with Grade 1 oesophageal varices with Grade 1 Haemorrhoids came to emergency with complaints of:

• Severe Headache since 1 day
• Left upper and lower limb weakness along with slurring of speech for 20 minutes

Chronic alcoholic for 20 years and had abstained taking alcohol 2 years back
Patient not on any blood thinners/anticoagulants
No recent hospitalization or surgeries.
Non smoker
• GCS –13/15
• Icterus present
• CNS - left 7th NERVE PALSY – indicated by inability to puff face, close eyes, and raise eyebrows

Power left upper and lower limb = 2/5

Plantar reflexes upgoing (left)

• NCCT Head was done s/o Right Frontal acute bleed/hematoma with proximal callosal body with ventricular extension resulting minimal contralateral midline shift of 3.5mm

• ECG done s/o Atrial Fibrillation

Blood investigations were sent which suggested deranged INR and Liver function tests

• INR - 2.6
• TOTAL BILIRUBIN: 5.57
• DIRECT BILIRUBIN: 2.32
• Patient’s ICH score was 2

Neurosurgery call was taken and Reversal of ICH (11) by FFP, VITAMIN K, TRANEXAMIC ACID was done in emergency department

3. Discussion

• Patients with liver disease had 40% higher risk for stroke.
• Each year stroke occurred in 2.2% of cases with liver disease and 1.1% in those without liver disease. (Houston, Texas, March 2017)

Etiology and Risks Factors:

• It is uncertain as to whether cirrhosis and, broadly, liver disease predispose to hemorrhage since the gastrointestinal bleeding seen in cirrhosis may be mostly due to portal hypertension rather than an intrinsic coagulopathy (40, 41). Therefore, it remains unclear whether liver disease is a risk factor for ICH.
• Liver disease is one of the suspected or possible risk factor.
• Coagulopathies i.e. use of antithrombotic or thrombolytic agents and systemic diseases such as thrombocytopenia are possible causes of ICH.
• Most important modifiable risk factor in spontaneous ICH is chronic HTN which is present in 80% (12). And most common locations of hypertensive ICH are the
putamen, thalamus, subcortical white matter, pons and cerebellum.
• Intracranial aneurysms, AV malformations, Cerebral venous sinus thrombosis, Brain tumors and cerebral metastasis, Drugs of abuse (cocaine, amphetamines).

Management:
The big 6 considerations in medical management of ICH in the ED.
1. BP
2. Coagulopathy
3. Glucose
4. Temperature
5. Seizure
6. ICP

Blood Pressure Management:
• Patients with ICH very often present with significantly elevated blood pressure. Elevated systolic blood pressure (SBP) is associated with haematoma expansion, neurological deterioration and poor outcomes after ICH (13).
• Randomised controlled trials (INTERACT II and ATACH II) have showed that early lowering of SBP to <140 mm Hg is safe without significant adverse effects.(14, 15) The INTERACT-2 trial comparing early lowering of SBP to <140 mm Hg with <180 mm Hg showed no increase in adverse events in the aggressive treatment group.(14)
• Nicardipine is the antihypertensive of choice in ICH – a pure arterial vasodilator
• Labetalol - Mixed alpha/beta adrenergic antagonist,
• Short-term- decreases SVR without effect on HR or CO ;Long-term - increases SVR and decreases HR while maintaining CO. Side Effect - postural hypotension
• Avoid Nitroprusside and Nitroglycerine infusions due to cerebral vasodilatory effects(16)
• American Heart Association (AHA)(2015) treatment recommendations (17):
• If SBP >200 mm Hg or MAP >150 mm Hg, aggressive reduction of blood pressure with continuous (IV) infusion, with BP monitoring every 5 min.
• If SBP >180 mm Hg or MAP >130 mm Hg and if there is possibility of elevated ICP, consider ICP monitoring and reducing BP using intermittent or continuous IV medications to keep cerebral perfusion pressure around 60 mm Hg.
• If SBP >180 mm Hg or MAP >130 mm Hg and there is no evidence of elevated ICP, consider a modest BP reduction (MAP -110 mm Hg or target BP of 160/90 mm Hg) using intermittent or continuous IV medications to control BP).
• Do not lower MAP by more than 20% in the acute phase.

Glucose Control in ICH:
• AHA / ASA Recommendation: Closely monitor glucose but avoid both hyperglycemia and hypoglycemia.
• Goal 140-180 mgdL. (Class I; Level of Evidence C).

Temperature Control in ICH:
• Fever is common in ICH in 30-50% patients.(18)
• Presence of IVH is a main risk factor for fever.
• Sustained fever after ICH is an independent prognostic factor for worse outcome.(19).
• Neither therapeutic hypothermia or normothermia was shown to improve outcome.(20, 21)
• Current recommendation is to cool core temperature below 37.5-38 degree C
• Treatment with antipyretics (eg, acetaminophen) and the use of cooling blankets are recommended, though new adhesive surface-cooling systems and endovascular heat-exchange catheters may prove more effective (22).

Seizure Prophylaxis:
• The risk of seizure is highest within first few days in patients with ICH.
• More than 50% of seizures occur in the first 24 h (23–31).
• Though antiepileptic drugs (AEDs) are often provided, it is not clear that their routine use is beneficial. Current AHA guidelines recommend that AEDs not be used routinely in patients with ICH without a specific indication.
• Continuous EEG monitoring is probably indicated in ICH patients with depressed mental status out of proportion to the degree of brain injury
• If AED therapy is to be used, agents to consider include:
  • Phenytoin, 20 mg/kg IV
  • Valproate, 10–15 mg/kg IV
  • Levetiracetam, 500–1500 mg IV
  • Phenobarbital, 20 mg/kg IV
• Most studies suggest that prophylactic anti seizure drugs (primarily phenytoin) are associated with increased death and disability in ICH, (32-34)

ICP Management in ICH:
• ED ICP strategies management include:
  • Head of the bed elevation between 30 and 45 degree with the head kept midline
  • Appropriate analgesia and sedation.
  • Elevated ICP is defined as ICP of 20 mmHg or more for over 5 minutes. Large volume ICH is commonly associated with high ICP.
  • The therapeutic goal of treating elevated ICP is to maintain ICP 20 mmHg while maintaining cerebral perfusion pressure around 70 mmHg.
  • Hyperventilation (temporary measure only)if herniating: Raise the ventilation rate with a constant tidal volume, for a goal pCO2 of 30 to 35 mm Hg.
  • Hypertonic solutions frequently used (mannitol, glycerol, and sorbitol), each has characteristic advantages and disadvantages.
  • Sorbitol and glycerol are metabolized by the liver and interfere with glucose metabolism. However, sorbitol is infrequently used due to a short half life and poor penetration into CSF.
  • Glycerol has a half-life less than one hour but it penetrates into the cerebrospinal fluid the best.
• Mannitol is commonly used because it is renally metabolized and has a half-life up to 4 hours.

Coagulopathy:

• Early reversal of coagulopathy is likely to be critical, and all anticoagulation-related hemorrhages should be treated as neurologic emergencies (35).
• Every patient who presents with spontaneous ICH secondary to uncontrolled HTN, which is non-surgically managed, should be treated with a combination of a bolus administration of Tranexamic acid (TXA) (1g) and a maintenance dose via infusion(1g over 8 hours) with strict BP control (36)
• Treatment with IV vitamin K (5–10 mg) allows the patient to begin producing coagulation factors in as little as 4 to 6 h (37).
• Provide factor repletion, which may improve hemostasis in the acute phase while awaiting an effect of vitamin K.
• Prothrombin complex concentrate (PCC), depending on the agent, can provide most or all of the missing factors, and can reverse the INR in minutes.
• PCC which are intermediate purity pooled plasma products containing a mixture of vitamin K-dependent proteins. Concentration of clotting factors in PCC is approximately 25 times higher than that in human plasma (FFP) (38).
• PCC is preferred over FFP due to more rapid correction of INR - risk of infection, pulmonary oedema, Transfusion-Related Acute Lung Injury (TRALI) and Transfusion Associated Cardiac Overload (TACO).
• Coagulopathy, whether medication-induced or due to a systemic disease process, is associated with haematoma expansion, and increased risk of poor outcome and death (39)
• Approximately 12–20% of patients presenting with ICH are taking oral anticoagulants.

4. Conclusion

Stroke is suspected as 4th leading cause of death.

ICH accounts for an increasing share of the 10% of deaths that are caused by stroke worldwide (42), and liver disease accounts for about 2% of global deaths (43), suggesting that further insights on the association between liver disease and ICH may help global efforts to improve public health.

• ICH is the most debilitating and deadly type of stroke.
• Stabilisation of Airway Breathing Circulation is essential for preventing secondary injury from Hypoxaemia, HTN and Haematomata expansion
• A number of factors may affect outcome after ICH, including haematoma volume and location, haematoma expansion, age, GCS score on presentation, intraventricular extension and anticoagulant use.
• Until and unless we don’t exclude haemorrhage Antiplatelets should not be given.
• Although oral anticoagulant therapy can significantly reduce the risk of AF related stroke, numerous international strategies have demonstrated that guidelines recommended prescription of anticoagulation remains suboptimal (44).
• No specific recommendation is made in AF guidelines with regard to anticoagulation in patients with cirrhosis, except for the inclusion of liver disease as a component of the HAS – BLED bleeding risk score (45) (46).
• Low dose NOAC (Non Vitamin K oral anticoagulation) may be safe and efficacious in short term, despite known differences in risks profiles and outcomes in Asian population with AF combined with the balance of thrombosis and bleeding in cirrhosis (47)
• The ICH score was developed to predict 30-day mortality rate.
• Even though if patient suffering from Alcohol Liver Disease with Atrial Fibrillation Aspirin use can be dangerous.
• Though mortality is very high but early recognition and treatment with Vitamin K, Tranexamic acid and Fresh Frozen Plasma may sometimes save the patient.

References

hemorrhage.


[18] https://emergencymedicinercases.com/intracerebralhemorrhage – golden-hour/


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