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Research Article

MANAGEMENT OF HYPERTENSION-RELATED INTRACEREBRAL HEMORRHAGE

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ABSTRACT
Intra cerebral hemorrhage (ICH) is a devastating disease with high rates of mortality and morbidity. ICH occurs as a result from rupture of blood vessels in the brain. This health problem has been increasing in hospital admission since the past 10 years. Patient is a 64 years old male with complain of sudden onset of right sided body weakness and slurred speech in the morning of the day he was admitted to HUSM. Patient presented with drooling saliva, mild tachypneic, aphasia and unable to move right sided limb. He had no history of headache, no chest pain, no blurred vision and no palpitation. Patient was alert and conscious during admission. ICH is a medical emergency, and delays in treatment result in worse outcome. Initial management should focus on urgent stabilization of cardio respiratory variables and treatment of intracranial complications.
Keywords: ICH, medical emergency, intracranial complications

INTRODUCTION

Intracerebral hemorrhage (ICH) is a devastating disease with high rates of mortality and morbidity. ICH occurs as a result from rupture of blood vessels in the brain. This health problem has been increasing in hospital admission since the past 10 years.[1] This could be as a result from increasing number of geriatric patients that lack of blood pressure control and increasing use of anticoagulant, thrombolytic and also antiplatlet agents. Intracerebral haemorrhage commonly affects cerebral lobes, the basal ganglia, the thalamus, the brain stem (predominantly the pons), and the cerebellum as a result of ruptured vessels affected by hypertension-related degenerative changes or cerebral amyloid angiopathy.[2] Most ICH occurs as primary (spontaneous) event related to rupture of small penetrating arteries and arterioles that have been damaged by chronic arterial hypertension or amyloid angiopathy and most of the primary ICH is hypertension related and only a portion is caused by amyloid angiopathy.[2] Nevertheless, secondary hypertension is related to multiple causes, as shown in Table 1. CT scan and MRI can be used to detect ICB. CT scan is the first line diagnostic tool to detect ICB. It can detect ongoing bleeding. However MRI with gradient echo can detect hyperacute intracerebral haemorrhage with equal sensitivity and overall accuracy and is more accurate for the detection of microhaemorrhages.[2] Secondary causes of intracerebral haemorrhage, such as aneurysms, arteriovenous malformations, dural venous thromboses, and vasculitis, cerebral angiography, MRI and magnetic-resonance angiography can be used to identify but their sensitivity is not well established.[2,3] A rapid onset of focal neurological deficit with clinical signs of increased ICP, such as a change in consciousness, headache, and vomiting, are important symptoms of ICH. Headache often occur with similar frequency at the putaminal, thalamic, and lobar locations. Headache was more common in patient with cerebellar hemorrhage. In conscious patients, the initial clinical features depend on the location and size of the hematoma.[2,4] A recent study showed that headache at onset of ICH not only with the presence of mass effect, but also with features of antecedent infection, as well as markers of inflammation such as higher body temperature, elevated leukocyte count and sedimentation rate, and biochemical markers of inflammation (interleukin-6, tumor necrosis factor-alpha). Seizures at onset of ICH are rare.[5] There are also symptoms that are specific with location of intracerebral bleeding for example at putaminal region. Patients present with contra lateral hemiparesis and hemi sensory loss, but without abnormalities of ocular motility, visual fields, or level of consciousness when they have medial putaminal hemorrhage. In patients with lateral putaminal hemorrhage, they may present with hemiplegia and sensory deficits, often accompanied by either aphasia or hemi neglect syndromes. It is this type of lateral putaminal ICH in the dominant hemisphere that has been associated with the syndrome of conduction aphasia, with fluent speech and preserved comprehension but with markedly impaired repetition. The clinical picture of massive putaminal-thalamic hematoma is characterized by impaired consciousness, hemiplegia, abnormalities of horizontal gaze and homonymous hemianopia.[4,6]

Table 1: Causes of Intra cerebral hemorrhage

Primary	Secondary
• Hypertension • Amyloid angiopathy	• Coagulopathy • Trauma • Arteriovenous malformation • Intracranial aneurysm • Dural venous sinus thrombosis • Cavernous angioma • Intracranial neoplasm • Dural arteriovenous fistula • Hemorrhagic conversion of cerebral infarct • Cocaine abuse • Vasculitis[2]

Table 2: Risk Factors of Intra cerebral hemorrhage

Non modifiable	Modifiable
• male gender • older age • African or Asian ethnicity	• Hypertension • Warfarin • High-dose aspirin • Alcohol intake • use of cocaine • High cholesterol level[2,3]

Case Presentation

A 64 years old Malay male that was admitted to HUSM with complain of sudden onset of right sided body weakness and slurred speech in the morning of the day he was admitted. Patient presented with drooling saliva, mild tachypneic, aphasia and unable to move right sided limb. He had no history of headache, chest pain, blurred vision and no palpitation. Patient was alert and conscious during admission. Patient had history of chronic obstructive pulmonary disorder (COPD) and post herpertic neuralgia. Patient was on medication, and was a regular visitor of cardiac clinic but not on any antihypertensive drug. His blood pressure on admission was 202/137 mmHg, pulse rate was 103 beat/min and oxygen saturation was 94 %. On examination, his lungs produced scattered rhochi and prolonged expiration phase. ECG showed sinus rhythm and S_1S_2 sound heard. CNS examination showed no power on right sided but still has high reflex and patient cannot fully obey command. CT brain scan (Figure 1) found that small amount of pre lesional edema surrounding the clot and clot size is the same. No mass effect found in the brain. Significant lab data finding is in Table 3. Examination from the neurosurgeons found that the patient had basal ganglia bleed, lateral putaminal bleed and thalamic bleed. During in ward, he had one episode of seizure but aborted spontenously. He was diagnosed with acute hemorrhagic stroke and acute exacerbation COPD accelerated by hypertension.

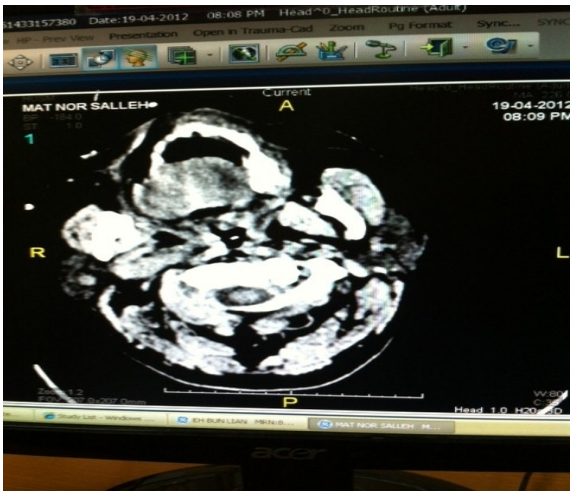

Figure 1: image of CT brain scan of the patient

Table 3: Significant laboratory finding

Parameter	Value	Normal value	Parameter	Value	Normal value
Calcium	10.5 mmol/L	2.1-2.6 mmol/L	**INR**	1.06	0.9-1.2
Serum Creatinine	137 µmol/L	53-115 µmol/L	**PT**	14.7	11-13 sec
Urea	10.5 mmol/L	3.5-7.3 mmol/L	**aPTT**	31.7	20-30 sec

DISCUSSION

Hyperglycemia worsens cerebral ischemic injury, and admission hyperglycemia is associated with increased 30-day mortality after ICH. However, the targets for glycemic control are unclear, and there is increasing evidence that "tight" glycemic control with insulin infusion can be associated with a critically low cerebral extracellular glucose concentration after brain injury.[2] Insulin therapy should be given if the blood glucose level > 10 mmol/L. Use of Dextrose solution should be avoided in patient with hyperglycemia.[7] However, in current patient, the blood glucose is in normal range. Dehydration may increase morbidity and mortality of patient with acute stroke. Most patient required 2000-2500 ml fluid per day. Use of hypotonic solution (0.45 % NS or D5 %) should be avoided as this will make added free water produce a relative plasma hypo-osmolarity that can cause subsequent shift of fluid to enter the brain. Use normal saline instead.[7] Thromboembolic prophylaxis with compression stockings and intermittent pneumatic compression is recommended in all patients from admission. Subcutaneous low-molecular-weight heparin should be considered after 24 to 48 hours, when it does not seem to result in an increased risk of recurrent hemorrhage.[2] Literature revealed, initial resuscitative measures should be directed to establishing adequacy of airway, breathing, and circulation.[4,7] Indications for endotracheal incubation include the lack of adequate airway protection (Glasgow Coma Scale [GCS] Score < 8), herniation syndrome, uncontrolled seizures, and respiratory failure. Airway control might be suboptimal in patients even with GCS > 8 in the absence of a good cough/gag reflex who may be high aspiration risk especially with brainstem hemorrhages. Hyperventilation might be necessary in the event of acute herniation, but, extrapolating from brain trauma literature, its prophylactic use is unlikely to be of benefit.[4,7] Current patients had AECOPD. Therefore it is necessary to give oxygen and air support, and was given Nebulizer Combivent (combination of ipratropium bromide and albuterol). This is treated by elevation of the head 30^0, keeping normal PO_2 and PCO_2, CSF drainage and mannitol.[7] Lowering BP should be avoided in first few days unless it is critically high or in patient with CHF, acute MI, Acute renal failure or aortic dissection.[2] Severe hypertension should be lowered to prevent haematoma expansion.[7,8] Current recommendation is that target BP should be around 160/90 mmHg. IV beta blocker (e.g. labetalol, esmolol) nicardipine, hydralazine and nitroglycerine can be used. Oral antihypertensive can be start after 72 hours to control patient BP gradually. In present patient, beta blocker should not be used as it is contraindicated with COPD. ICH is the most serious complication of warfarin anticoagulation. The risk of ICH approximately doubles for each increase of one in the international normalized ratio (INR) and an INR > 3 is associated not only with larger initial hematoma volume but also with an increased frequency of hematoma expansion and higher incidence of neurological deterioration in the first 24 to 48 hours. For patient with anticoagulant therapy, early administration of fresh frozen plasma and vitamin K is recommended to reverse this coagulopathy.[2,7] Approximately 8 % of patients with ICH develop clinical seizures within 30 days of the ictus, and continuous electroencephalographic monitoring demonstrates subclinical seizure activity in up to

25 %. Seizures are more likely to occur in the presence of a lobar hematoma.[2] The use of prophylactic anticonvulsant medication after ICH is controversial, although one small study showed that it does reduce the risk of early seizures. Current guidance does not recommend universal prophylaxis, but that therapy should be considered in selected patients with lobar ICH. If seizures do occur, they should be treated aggressively in the usual manner.[2,7] The application of haemostatic therapy is to minimize hematoma expansion and improve outcome after ICH. Treatment with recombinant factor VII (rFVIIa), a potent initiator of haemostasis, within 4 hours of ICH significantly reduced hematoma growth in association with reduced mortality and improved functional outcome in survivors at 3 months. This improvement was seen despite a small increase in thromboembolic complications in the rFVIIa-treated patients. rFVIIa does not replace all clotting factors, and although the INR may be lowered, clotting may not be restored *in vivo*; therefore, rFVIIa is not routinely recommended as a sole agent for OAC reversal in ICH. Although rFVIIa can limit the extent of hematoma expansion in non-coagulopathic ICH patients, there is an increase in thromboembolic risk with rFVIIa and no clear clinical benefit in unselected patients.[2,8] Surgical evacuation of hematoma may be indicated in surgically accessible cerebral hematoma causing significant mass effect for example more than 30 ml in volume on the CT scan. The outcome is more favorable in young patient and has no other pre morbid illness. However, result from Surgical Trial in Intracerebral Hemorrhage (STICH) showed that there was no difference between patient undergoing early surgical intervention and those that were treated with initial conservative management.[7]

CONCLUSION

Although ICH remains the form of stroke with the least satisfactory treatment options, recent advances in our understanding of its pathophysiology, and the beneficial effect of some interventions, have resulted in a shift away from therapeutic nihilism. ICH is a medical emergency, and delays in treatment result in worse outcome. Initial management should focus on urgent stabilization of cardio respiratory variables and treatment of intracranial complications.

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