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ICU management in massive spontanous intracerebral hemmorrahge: Case report

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Abstract
Spontaneous intracerebral hemorrhage is a nontraumatic bleeding into brain parenchyma, it's the most common subtype of stroke, case fatality is extremely high and those who survive are independent within 6 months. Chronic arterial hypertension, coagulopathy, anticoagulation therapy is the common risk factors. Timely and aggressive management in the acute phase may mitigate secondary brain injury. Initial medical stabilization, neuro imaging to establish diagnosis, timely intervention may prove helpful. This case report involves the intensive care management in a 25year female who presented with left sided acute onset weakness with drop in consciousness. Neuro imaging was suggestive of massive intracerebral hemorrhage. Surgical evacuation of hematoma was done and patient was managed in SICU.

Keywords: ICH, intensive care unit, surgery, hematoma, neuro imaging

Introduction

Spontaneous intracerebral hemorrhage (ICH) is defined as nontraumatic bleeding into the brain parenchyma, which can extend into the ventricles and into the subarachnoid space. ICH is the second most common subtype of stroke, accounting for 10–50 % of all cases, depending on the population, race, and region studied. The case-fatality rate ranges from 35 % at 7 days to 59 % at 1 year. Half of fatal cases occur in the first 48 hours after presentation. Survivors are often left with severe disability, with less than 40 % of patients regaining functional independence. The epidemiology of ICH may change in the future with better control of risk factors such as hypertension, but the use of newer anticoagulation therapies may influence the acute management and possibly prognosis of the disease [1-14].

Risk factors for ICH include genetics, medical conditions, and lifestyle. Genetic risk factors include the presence of an apolipoprotein E2 or E4 allele and a first-degree relative withICH. Other known risks for ICH include increasing age, race, history of hypertension, smoking, and frequent alcohol use. Though some risk factors clearly cannot be modified, some can, and there is evidence that doing so can actually decrease an individual's risk of ICH. The PROGRESS trial showed that blood pressure lowering treatment reduced the risk of ICH in patients with cerebral amyloid angiopathy and may be protective against ICH from other causes. Additionally, a large observational study found that less exercise, heavy alcohol use, and smoking predicted increased risk of ICH [15-21].

Early and accurate diagnosis of ICH is critical. Initial presenting symptoms can include abrupt onset of headache, vomiting, seizure, and any focal or generalized neurologic symptoms. The differentiation of ischemic from hemorrhagic stroke cannot be made in the absence of neuroimaging. The initial test of choice in most centers for patients with an acute neurologic complaint is a CT scan of the brain. CT scanners are widely and rapidly available in the United States and are highly sensitive for ICH [22-23].

Though most patients have primary ICH, many have what is termed secondary ICH, or ICH that is due to a cause other than small arteriolar disease. Causes of secondary ICH can include aneurysm, arteriovenous malformation, Moyamoya disease, tumor, cerebral venous sinus thrombosis, or hemorrhagic transformation of ischemic stroke. Features suggesting high risk for secondary ICH include lobar ICH, intraventricular blood, and younger age [27]. Younger patients with lobar ICH are at higher risk for underlying vascular malformation, but this risk is present even for deep hemorrhages. A scoring system has been suggested to risk-stratify patients for risk of secondary ICH [28]. Those who have aneurysms or arteriovenous malformations may be candidates for surgical or endovascular interventions [24, 25].

Managing acute intracerebral haemorrhage is a challenging task for physicians. Evidence shows that outcome can be improved with admission to an acute stroke unit and active care, including urgent reversal of anticoagulant effects and, potentially, intensive blood pressure reduction. Nevertheless, many management issues remain controversial, including the use of haemostatic therapy, selection of patients for neurosurgery and neuro critical care, the extent of investigations for underlying causes and the benefit versus risk of restarting antithrombotic therapy after an episode of intracerebral haemorrhage.

Case Report

25year young female presented to emergency department of SMHS hospital with; sudden onset left sided weakness. With no past medical or surgical history, no history of coagulopathy no history of any medication intake, no family history of any coagulopathy, neurological disease or cancer. On examination she had. Glassgow Coma Scale; E3V2M5, PUPILS; bilateral equal size and sluggishly reacting to light.

motor power left upper and lower limb 0/5 and right limb 5/5 SPO2;98%, BP;112/67, PR;73/min Investigations; HB=14, Platletcount;180000, WBC;13000, Blood urea;39, creatinie;1.02, Sr. sodium;139, sr potassium;3.8 Pt/INR;13.2/1.02, LFT; WNL. NCCT HEAD E/O; 8×6cms bleed seen in righttemporoparietal lobe with effacement of right lateral ventricle.

Ct Angiogram: Unremarkable Study
Patient had a drop in GCS from E3V2M5 to E1V1M5 within an hour. Plan for surgical evacuation of hematoma was done by neurosurgeon Patient was intubated and put on mechanical ventilation.

Surgery: Right frontoparietotemporoocipital decompressive craniotomy with duroplasty with evacuation of hematoma was done.

Intraoperative Findings: brain tense, edematous, dura thin, brain pulsatile, around 50ml of blood evacuated. Intraoperatively patient was haemodynamically stable and received two units of packed red blood cells. Patient was not extubated and was shifted to SICU. Patient received in SICU intubated, connected to Mechanical ventilator on volume control mode. Patient was sedated with midazolam infusion and fentanyl infusion for 48 hrs. after 48 hours sedation was stopped and patient was assessed GCS; E3VntM5, PUPILS; right pupil could not be assessed because of periorbital edema and LEFT WAS REACTING TO LIGHT. Spontaneous breathing trail given for two hours patient was maintaining spo2 of 96-97% on minimal Fio2 of 35%. Patient was afebrile and haemodynamically stable proper suctioning was done and patient was extubated and was put on polymask with oxygen 10ltrs per min. Post extubation GCS was E2V2M6.Within two days GCS improved E4V5M6. Pupils, bilateral reacting to light, maintaining spo2 on room air. Patient was haemodynamically stable afebrile, chest, bilateral air entry present with no added sound, motor power rt;5/5, left 1/5 both upper and lower limbs. Repeat NCCT head was done, E/O, parietal bleed. Treatement received in icu; injection;pipercelline tazobactum 4.5gm iv 8hrly, inj. Levofloxacin 750mg iv OD, inj. pantop 40mg iv OD, inj phynetoin 100mg iv 8hrly, inj levitireacetam 500mg iv OD, inj mannitol, inj paracetamamol, Blood sugar was kept between 14-180, normocapnea and oxygen saturation was maintained. pneumatic compressions were used to prevent venous thromboemboism. physiotherapy started. Patient was shifted to HDU of our department for further monitoring. GCS improved over time at time of shifting to parent unit the GCS WAS E4V5M6.
Physiotherapy was started on affected limb Motor power also got improved. Patient was shifted to parent unit after 11 days of icu stay Follow up was done in neurosurgery Opd and physiotherapy was continued.

Condition on Discharge: GCS; E4V5M6 with left hemiplegia. follow-up with neurosurgery OPD and physiotherapy OPD was advised on discharge. After two months the motor power in right upper and lower limb was 5/5 and on left side 4/5.

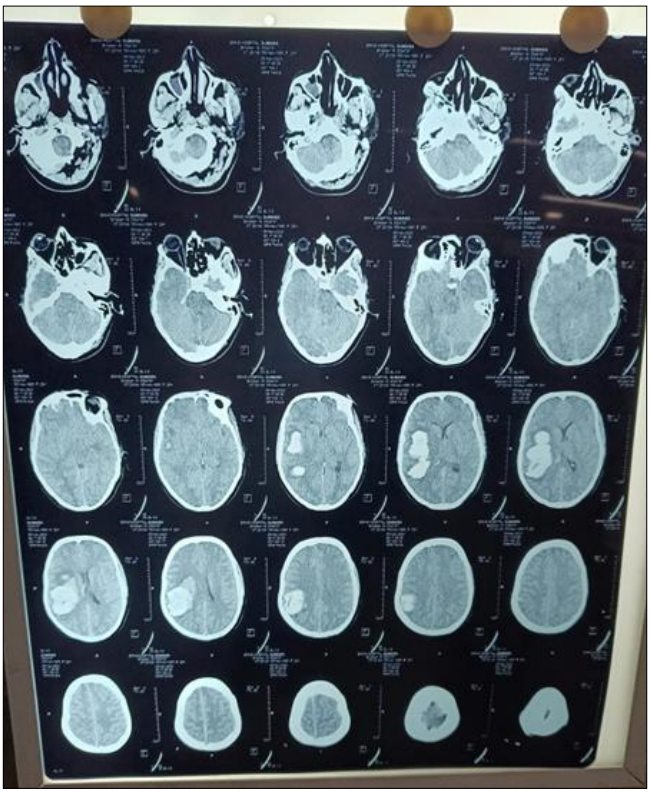

Fig 1: Preoperative scan

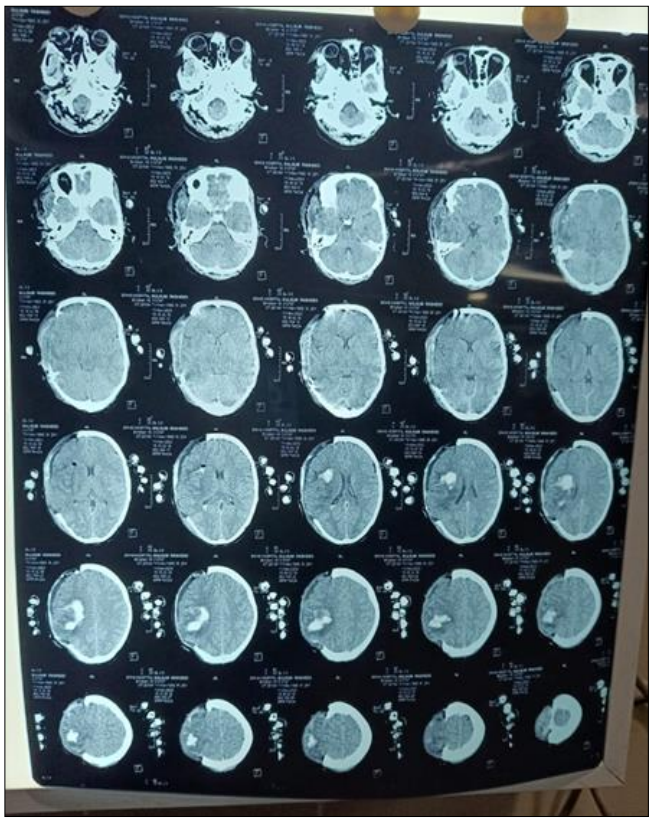

Fig 2: Postoperative scan

Discussion
Despite the progression in ICH management, the significant reduction in mortality and morbidity is not reduced still. Getting out alive from ICH does not mean the patient survived without complications. In our case, the patients showed GCS; E4V5M6 with left hemiplegia on discharge. Based on the last reports, just 10–20 % of ICH patients

survive hospitalization. Severe headaches for more than one week, waking pain, fever, and conscious alteration are accompanied by poor prognosis in non-traumatic headaches such as ICH. Decreasing two or more points on GCS is a warning sign of neurological deterioration (ND) and significantly increases the mortality rate. Identifying and appropriate management in early stage of ND is so critical. It was demonstrated that ultra-early ND in prehospital occurs in 1 of 7 patients in ICH and is accompanied by poor prognosis. Developing ND in ICH frequently occur, even up to 30 % were reported. The time of ND is critical; however, there is no definition cutoff time for ND diagnosis. Nonetheless, there are two ND classifications; early ND which occurs in <48 h, and late ND, which is detected through 48 h-7 days following operation. No definitive criteria were reported for ND diagnosis; however, Weakness or paralysis, Abnormal movement, such as tremors or difficulty walking, Loss of balance, Difficulty swallowing or feeling "a lump in the throat," Seizures or episodes of shaking, loss of consciousness (nonepileptic seizures), and Episodes of unresponsiveness are reported as concerning symptoms of ND. Since ND is one of the primary mortality causes, it leads to a proliferation of literature to determine a reliable scoring system to increase the clinical outcome of patients. Previous ICH, high blood pressure, larger hematoma, subarachnoid expansion, antiplatelet therapy, and higher National Institutes of Health Stroke Scale (NIHSS) are the main prediction factors for early ND. However, most patients develop early ND, but male sex, age, ICH history, larger baseline hematoma, and higher NIHSS account for susceptible factors for delayed ND [26-30]. Due to higher accuracy, using the ICH score rather than GCS is highly recommended. He *et al*. introduced the site, size, gender, National Institutes of Health Stroke Scale, age, leukocyte, sugar (SIGNAL) score, which consists of location, size, NIHSS, age, leukocyte, and sugar scales (0–8 points) for detecting susceptible ICH patients for ND [31]. We did not have any information about our case during the incidence and history of symptoms before the events—just stiffness with detection of considerable ICH in CT scan candidate the patient for emergency operation.

The exact injury mechanism of spontaneous ICH is not well known. Nevertheless, there is considerable literature about the role of inflammatory cytokines ICH. Increasing pro-inflammatory cytokines cause local accumulation of white blood cells in the injury site in ICH and develop leukopenia, which is accompanied by poor prognosis [32]. This association between cytokines and injury severity is linear. In this regard, evaluating the influence of anti-inflammatory agents as therapeutic targets in ICH is recommended. A stiff neck is associated with high hospital mortality rates in patients with ICH.

It was highly recommended to follow ICH patients with serial CT scans to identify any symptoms of ND development [33]. High prevalence of blood-brain dysfunction in small vessels in ICH confirmed the importance of serial CT scans in preventing disease progression [34]. Delays in following ICH patients, developing IVH, and hydrocephaly are expected. In our case repeated NCCT head was done, E/O, parietal bleed. Our patient presented with no identifiable risk factors or past history that could direct us to etiology. This may be because lack of basic healthcare access leads to many hypertensive and other chronic conditions and lack of awareness among general population about regular health checkup. Our patient presented with sudden onset left side weakness with drop in GCS with CT Imaging suggestive of intracerebral hemorrhage with ventricular effacement. Timely diagnosis and intervention followed by medical management and surgical evacuation with decompression and SICU management was the mainstay of treatment.

Conclusion

Spontaneous ICH is a neurological emergency associated with high mortality and morbidity KEY management involves prompt etiological diagnosis, reversal of anticoagulation consideration of surgical management control of blood pressure; in order to overcome high mortality and morbidity. Patients with ICH should be managed in intensive care unit with multidisciplinary team approach. Close monitoring of vital signs, neurological status, proper ICP control, and timely surgical management with prevention of complications and early rehabilitation improves the outcome.

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