EDITORIAL

Bundle of Neurocare in ICH

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The first evidence of stroke comes from Hippocrates in the 5th century BC. He named the condition apoplexy, which means "struck down". What actually led to apoplexy or stroke was discovered centuries later by Dr Johann Jacob in 1658, after performing post-mortem examination of patients who died due to apoplexy. He came to the conclusion apoplexy is caused by an interruption in blood supply to parts of the brain or brain either from a blockage in a brain blood vessels or bleeding in the brain.

In two types of stroke, viz. ischemic vs hemorrhagic, incidences of hemorrhagic strokes are 10–20% as per high-income country data, rates for middle and low-income countries can be higher.[1] Clinical worsening of hemorrhagic stroke can be faster thus increasing morbidity and mortality.[2] Significantly less number of patients with intracranial hemorrhage (ICH) becomes functionally independent. Those surviving may end up in a long-time vegetative state, if a large area of the cerebral hemisphere or brainstem involved. Clinically can distinguish between two types of stroke based on the progress of worsening of neurological deficit. Analyzing and knowing the risk factors from day to day clinical scenarios in hospitals (Emergency Departments, Intensive Care Units), can help in modifying, and treating risk factors such as hypertension control, and lifestyle modifications.

To analyze the possible risk factors, which can be addressed in the management of ICH a retrospective cohort study spanning 5 years (from 2013 to 2018) was conducted in an Oman-based Tertiary Care Teaching Hospital's emergency room. Many of the patients were between the age of 50 and 70 years as usual with other study findings. Most common comorbidities found were diabetes mellitus, hypertension, and ischemic heart disease.[3]

In July 2023 INTERACT-3 trial was presented with results.[3] The study was conducted in many South-East Asian, South American countries. Study used care bundle protocol of early intensive control of systolic blood pressure below 140 mm Hg, strict blood sugar control, antipyretic treatment targeted for temp below 37.5°C and rapid reversal of warfarin-related anticoagulation effect. Poor functional outcome at 6 months was lower for the bundle care group. The number needed to treat was 35 to save one life free of disability.[4]

Many of the care bundles utilized during emergency care of patients with ICH in this Oman-based Tertiary Care Teaching Hospital's emergency room.

Even though this study has marked to say that there was no difference in increased severity of dysfunctional outcome irrespective of hematoma location, those with brainstem location had more mortality, considering a total number of patients who had brainstem bleed. This is likely to have a worse outcome compared to another location bleed. Whereas the correlation of poor outcome in those with more midline shift and larger hematoma size correlates with previous studies.

The size of the hematoma and intraventricular hemorrhage caused a mass effect, hydrocephalus respectively, ultimately raising intracranial pressure, effecting neuron, glial structures. Thus more severe will be neurological deficit.

The statistical non-significant effect of coagulopathy on poor neurological functional (mRS) outcome at discharge, which does not correlate with previous trials.[5] Overall as a number of patients with coagulopathy was less, it may be difficult to make a statement that coagulopathy does not have an impact on hematoma size, ventricular bleed, thus on neurological functional outcome. Only one patient showed coagulopathy as per an INR value of more than 2. Even though the majority of ICH occurs at INR values above 2, when associated with hypertension, INR values above 1.5 may contribute to hematoma expansion.[4] In this retrospective study, the majority of intracranial bleed was for those with INR below 2, but here not mentioned how many patients with INR above 1.5. Treating coagulopathy for those with INR above 1.5 might have helped in preventing further expansion of hematoma in size and in critical location, as they have not mentioned about INR cut-off value for labelling and treating as coagulopathy.

About 5% of patients, who had intracranial, bleed in this retrospective study were found to be taking oral anticoagulants, which was dismay with previous trials, where the context of OAC and ICH was much higher. This context dissimilar compared to others, may be related to demographic, comorbidities related to a given geographical population.

Even meta-analysis, RCTs have shown no significant increase in the size of the hematoma, or neurological deterioration even in patients with IC bleed on pharmacological DVT prophylaxis, starting 48 hours post incidence of IC bleed, whereas decreased incidences of DVT and PE.[6–10]

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Only very few patients were on anticoagulant treatment at the time of admission (five patients on warfarin, one patient on rivaroxaban), not mentioned about dosages these patients were taking for their underlying cardiac conditions, as only one patient was found to have INR above two and at least five patients were taking tab warfarin. As previous randomized trials have mentioned significant incidences of intracranial bleed, and hematoma expansion with the use of warfarin which they were taking for atrial fibrillation, etc. Whereas newer oral anticoagulants have fewer incidences of intracranial bleeding as well as hematoma expansion compared to warfarin. For rivaroxaban, half-life is 5–10 hours. Whereas half-life of warfarin 20–60 hours. Newer OAC's effect of anticlotting wears off within 24 hours, duration of action of a dose of warfarin lasts for 2–5 days. It means, that even after stopping medication on arrival to hospital, hematoma has scope to get expanded further, from prolonged sustained action of warfarin even after hours of stopping.

Even though this retrospective trial not mentions neurosurgical intervention, neurosurgical decompression might have been considered and executed in appropriate conditions as per neurosurgical discretion.

In day-to-day care of patients of ICH liaison with intraventricular hemorrhage, intraventricular thrombolysis (by ventriculostomy) may positively favor neurological outcome as well as overall outcome.[11]

As on admission significant number of patients had GCS 11–15, i.e., 60%, with hematoma size below 30 cc in 75%, while at discharge GCS of 11–15 was found in 85% of patients. At the time of discharge, there was a decrease in hematoma size in a significant number of patients. Three patients had hematomas sizes less than 30 cc, but they died at the location pontine, which did not spare vital centers. Mortality as well good functional neurological outcome reflected in this retrospective study reflects meticulous neurological care involved in managing the neurosurgical patients as well partly related to smaller sample sizes compared to previous trials and absence of follow-up beyond discharge.[12–14]

The study did not find a difference in differences in grades of severity of functional neurological impairment at the time of discharge with underlying comorbidities viz. diabetes mellitus, hypertension, ischemic heart disease, age and gender.

So intense neurological care involved in intracranial bleeding helps in not only improving neurological functional outcome but also mortality, viz. control of blood pressure, reversal of anticoagulant effect, surgical decompression wherever appropriate, antiepileptic, antipyretics, blood sugar management, intraventricular thrombolytic agents in selected cases, DVT prophylaxis, etc.

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