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"Time is brain"- management of the patient with iatrogenic intracerebral hemorrhage

Kakde Avinash Sahebarav[1], Sathe Kalyani Anand[2], Parmar Swati Deepak[3], Gandhe Rajashree Uday[4]

From [1]Clinical Assistant, [2]Neuroanesthesia Fellow, Department of Neuroanesthesia, [3]DNB, Department of Aneasthesia, [4]Head, Department of Neuroanesthesiology, Kokilaben Ambani Hospital, Mumbai, Maharashtra, India

Correspondence to: Dr. Kakde Avinash Sahebarav, 2001/B, Atelier, Rustomjee Urbania, Nashik highway, Majiwada, Thane – 400 601, Mumbai, Maharashtra, India. E-mail: avinashkakde87@gmail.com

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ABSTRACT

The brain is a critical organ of our body, which depends on a continuous supply of oxygen and glucose for normal functioning. An inadequate supply of oxygen and glucose can trigger a characteristic pathophysiological cascade leading to neuronal death. Multiple neuroprotective strategies have been developed blocking one or more steps along this cascade. Here, we report the case of an intracerebral hemorrhage during neurointerventional procedure in a catheterization laboratory. Accurate decision and timely intervention of neuroprotective strategies resulted in the complete neurological recovery of the patient.

Key words: *Arteriovenous malformation, Cerebral perfusion pressure, Decompressive craniectomy, Hemorrhage, Intracranial pressure*

Ischemic and traumatic brain injuries are among the most common and important causes of disability and death worldwide. Inadequate supply of oxygen and glucose can trigger a characteristic pathophysiological cascade leading to neuronal death. After the interruption of constant supply of oxygen and glucose, the electron transport chain within the mitochondria and thereby oxidative phosphorylation is inhibited. Within 1–2 min, this is followed by decreased levels of high-energy phosphates; within 4 min, it leads to the depletion of glucose and glycogen stores; and after 5–7 min, the cellular adenosine triphosphate concentration rapidly drops to near zero [1].

Permanent structural damage occurs within 5–15 min of oxygen deprivation, while hypoglycemia can be tolerated for up to 60 min. Therefore, accurate decision and timely intervention in a patient with cerebral insult can decrease morbidity and mortality [1]. We report the case of a 24-year-old American Society of Anaesthesiologist (ASA) physical status 1 patient, who suffered intracerebral hemorrhage during embolization of cerebral arteriovenous malformation (AVM).

CASE REPORT

A 24-year-old female ASA physical status 1 was posted for the left sylvian AVM embolization. The patient had the complaint of a headache which was moderate in intensity, on and off with no specific aggravating and relieving factors for the past 3 months. The patient underwent cerebral angiography under local anesthesia which showed left sylvian AVM (Fig. 1). AVM embolization was advised as a treatment, and the patient was posted for AVM embolization as an elective procedure under general anesthesia. After confirming nil by mouth status and obtaining written informed consent from the patient, she was shifted to the neurointervention room. Routine monitoring was done which included non-invasive blood pressure, electrocardiography, and pulse oximetry. The patient received injection glycopyrrolate 0.2 mg, injection midazolam 2 mg, and injection fentanyl 100 µg as premedication. General anesthesia induction was done with propofol (2.5 mg/Kg) and muscle relaxation with atracurium (1 mg/Kg) for tracheal intubation. Tracheal intubation was done with 7.0-mm internal diameter Portex cuffed endotracheal tube. The bladder catheterization was performed for urine output monitoring.

The patient received intravenous heparin 2000 IU before starting the procedure. Neurointerventionist used Histoacryl glue for the embolization of AVM. During the procedure, extravasation of dye was noticed which suggested hemorrhage within AVM. To confirm the diagnosis, immediate head computed tomography (Dyna CT) in the catheterization laboratory was performed which showed temporal hematoma with midline shift (Fig. 2). On clinical examination, the patient's left-sided pupil was 4 mm dilated, fixed, and non-reactive to light, which was suggestive of increased intracranial pressure (ICP) from expanding hematoma and imminent uncal herniation of the brain.

As the patient was anticoagulated with heparin during the procedure, immediately protamine 20 mg was administered to reverse the anticoagulant action of heparin. Injection mannitol 20% (1.5 g/Kg) was administered. Borderline hypocapnia (ETCO2 in range of 26–30 mm of mercury) was maintained to decrease the ICP. The left radial artery cannulation was done for beat-to-beat blood pressure monitoring. As the patient was intubated and

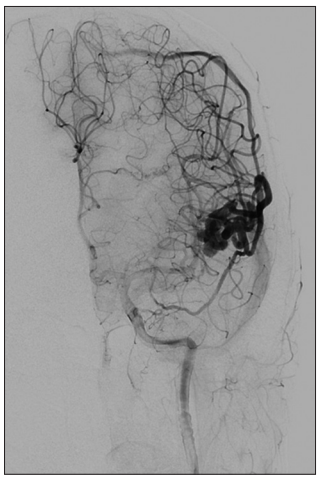

Figure 1: Cerebral angiography showing arteriovenous malformation

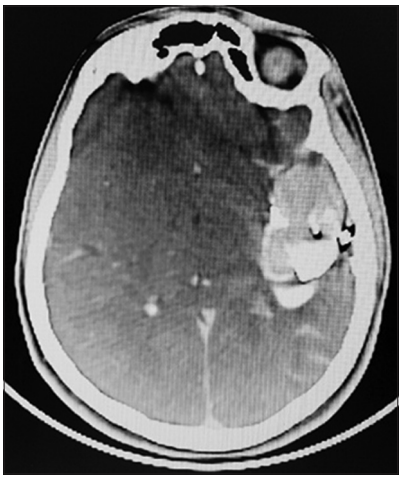

Figure 2: Dyna computed tomography showing intracerebral bleeding

paralyzed, we were unable to document the Glasgow Coma Scale. After repeat head CT, considering the expanding intracerebral hematoma, urgent neurosurgical consultation was sought and emergency decompression in operation theater (OT) was planned. The patient's relatives were explained about the intraprocedural complication and further necessary management. The consent for the procedure was obtained from the relatives. The patient was shifted to OT immediately and received 1.2 g loading dose of injection fosphenytoin.

The left frontotemporoparietal craniotomy was performed. The hematoma was evacuated, and the closure was done without replacing the bone flap. The patient was electively ventilated and paralyzed in the neurointensive care unit for 2 days. On the 2nd post-operative day, in a neurointensive care unit, the patient's pupils were 2 mm and were reacting to light. The patient received injection fosphenytoin 150 mg as an antiepileptic 3 times a day. On the 3rd day, sedation and neuromuscular paralytic drugs were stopped. The patient was completely awake with no neurological deficit and successfully extubated on the 4th post-operative day. She underwent cranioplasty on the 13th post-operative day and discharged home on the 17th post-operative day without any neurological sequelae.

DISCUSSION

The estimates of cerebral AVM prevalence that is published in the medical literature are unfolded. Due to the rarity of the disease and the existence of asymptomatic patients, establishing a true prevalence rate is not feasible. However, it can be inferred from the incidence data to be lower than 10.3 per 100,000 population [2]. The risk of spontaneous ICH without treatment is estimated to be approximately 2–4% per year for all the patients with untreated AVM, and approximately 2–4% of patients present with intracranial bleeding. Most of the patients are commonly present with intracranial hemorrhage than headache. According to the World Federation of Neurointerventional and Therapeutic, self-reported treatment-related complications are in the range of 9–12% [3].

To meet the brain's metabolic demand, an adequate cerebral blood flow (CBF) is required. The brain is enclosed by the non-expandable skull, and an increase in ICP may reduce cerebral perfusion pressure (CPP) and impair CBF leading to cerebral ischemia. Both CPP and ICP are established treatment targets in neurocritical care [4].

"Monro-kellie doctrine" states that the brain is enclosed in a non-expandable case of the bone and the brain parenchyma is nearly incompressible. In other words, the volume of the intracranial compartment must remain constant, if ICP is to remain constant [4]. The CPP is defined as the difference between cerebral arterial pressure and pressure in the cerebral venous bed. Too low CPP causes ischemia, while too high CPP causes hyperemia. Decreasing CPP is particularly dangerous as it decreases the driving force for cerebral blood to flow and compromises autoregulation. CPP-oriented therapy has been introduced to decrease the risk of ischemia in post-injury care. Placement of radial arterial catheter for beat-to-beat blood pressure monitoring can be helpful to maintain mean arterial pressure at an adequate level to enhance CPP [5].

Basic medical management for increased ICP includes head elevation to 30°, strict avoidance of free water, seizure prophylaxis, and fever control. The second line of therapy in ventilated patients with high ICP is sedation to produce a quiet, motionless state. After sedation and CPP optimization, osmotherapy and hyperventilation usually go hand in hand to attain the control of elevated ICP. Mannitol and hypertonic saline have the greatest impact on ICP when they are administered in bolus doses [6].

Hyperosmolar therapy, using infusions of mannitol or hypertonic saline, is frequently administered to reduce ICP [7]. Currently, mannitol (20%) is considered as the gold standard hyperosmolar agent and is by far the most well studied. However, hypertonic saline which comes in a variety of concentrations is increasingly used in these settings [8]. Decompressive craniectomy (DC) first described by Annandale in 1894 performed as a palliative procedure for an inoperable brain tumor.

Kocher (1901) first proposed DC as treatment of raised ICP. Nowadays, DC is the treatment modality of raised refractory ICP and most controversial of all. DC is the most studied intervention for intracranial hemorrhage. The term "refractory intracranial hypertension" implies that ICP increased over a few hours to very high value and leads to the death of the patient unless aggressive ICP treatment is initiated. However, DC's greatest benefit may require that the decision about surgery is undertaken well before an uncontrollable elevation in ICP occurs [9].

CONCLUSION

In this case, advanced technology like the presence of Dyna CT facility in the catheterization laboratory for brain imaging saved our time and gave us the correct diagnosis. Accurate decision and timely intervention of neuroprotective strategies resulted in the preservation of patient's neurological function and life.

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