Review article

Indian Journal of Neurotrauma (IJNT)
2009, Vol. 6, No. 1, pp. 17-20

Surgery for cerebral contusions: Rationale and practice

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Abstract: While the indications for surgery in extra- and subdural hematomas are relatively established, the role, timing and the indications for the surgical treatment of cerebral contusions remains nebulous. The pathophysiology of brain edema and the resultant increase in intracranial pressure due to brain contusions is discussed. The role and limitations of early evacuation of contused brain are considered in this article.

Keywords: cerebral contusions, cerebral edema, decompressive surgery

INTRODUCTION

Cerebral contusions are areas of trauma induced brain swelling. These are zones of cellular injury where the microvasculature is also disrupted. The contused area becomes an amalgam of blood and necrotic brain, the so-called 'hemorrhagic contusion'. Sometimes bleeding from a small ruptured vessel dominates and an intracerebral hematoma results. In diffuse axonal injury, inertial stresses cause axonal ruptures. Microvascular rupture would be associated with the axonal disruptions. The resultant micro-hemorrhages provide the basis of the radiological grading of diffuse axonal injury (DAI). Significant brain swelling may not occur in some cases of DAI[1].

The genesis of brain swelling in an area of brain contusion is complex[2]. The mass effect of an area of contusion is conventionally considered to result from a combination of vasogenic and cytotoxic edema. Vasogenic edema results from the breakdown of the blood brain barrier and extravasations of fluid into the extracellular space. Cytotoxic edema is the consequence of a hypoxic insult resulting in membrane pump failure and cellular swelling. The early swelling around a contusion which occurs in the first 24 hours and is often life threatening, cannot be explained by either of these factors. Vasogenic edema sets in only after 12-24 hrs[3]. Cytotoxic edema can occur early, but the quantum of cytotoxic edema is insufficient to explain the mass effect that is clinically encountered.

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There are three clinical phases of brain swelling due to contusion. The ultra early phase occurs within the first 24 hours and is often the cause of clinical deterioration or death. The second, delayed phase sets in after 24 to 72 hrs and progresses for 7-10 days[4]. This swelling rarely contributes to clinically significant intracranial hypertension.

PATHOPHYSIOLOGY

The severe brain swelling which occurs after a traumatic brain injury has three temporally distinct and etiologically disparate phases[5]. In the first phase, there is breakdown of cell membrane proteins and lipids into smaller molecules resulting in an osmotic gradient and an indrawing of fluid into the contusion. In the central area of contusions the cellular elements – the neurons and glia undergo disintegration and homogenization. This results in the formation of idiogenic osmoles and a rise in the tissue osmolality from 310 mosmol in normal brain to 370 to 390 mosmol in an area of contusion[6].

The contribution of electrolytes to the elevation of tissue osmolarity has been studied(5). There is a rise of sodium (from 46.5 mosmol /kg to 74.2 mosmol /kg and chloride from 437 to 51.2 mosmol /kg. This is however partially offset by a decrease in tissue K+ from 92.7 to 71.1 mosmol/kg). It is relevant to note that tissue osmolarity above 300 mosmol may be resistant to hyperosmotic therapy[7].

The second phase of brain edema is dominant 24-48 hrs after trauma. This phase is mediated by activation of thrombin consequent to triggering off of the coagulation cascades[8]. Thrombin and inflammatory

mediators act in concordance to disrupt the blood brain barrier resulting in vasogenic edema.

The third phase of edema sets in with the lysis of RBC in the intracerebral clot. Hemoglobin breakdown products activate reactive oxygen species, trigger cytokines (esp IL6 & IL10) and activate the complement system (esp C 3d & C9)[9].

Cerebral edema jeopardizes the revival of potentially salvageable neurons. The mass effect of the contused mass results in an impairment of cerebral blood flow, adding to the neurological insult. Cerebral blood flow gets impaired due to many causes. The rise in ICP by the expanding contused mass results in a fall of the cerebral perfusion pressure head in a setting of failed cerebral autoregulation (trauma induced)[10]. This hypoperfusion is also due to microvascular distortion resulting in further hypoxic cytotoxic insult. Shifts of brain matter compound the insult by vascular and axonal distortions and obstruction to the CSF circulation. The management of intracranial pressure therefore is important to prevent second injury.

MODALITIES OF ICP MANAGEMENT IN CEREBRAL CONTUSIONS

Position: Raising the head end of the bed by 30° and ensuring optimal venous drainage assists in resorption of interstitial fluid into the venous system.

Oxygenation: Maintaining the PCO_2 between 30 to 35 mm of Hg and oxygenation at 100% (PaO_2 100 mm of Hg) optimises vascular diameters and oxygen delivery. Moderate hyporcarbia of (PCO_2 28-30mm of Hg) may be beneficial over short stints, but hyporcarbia to < 25mm Hg PCO_2 results in vasospasm and ischemia[10].

Hyperosmolar therapy: Mannitol is the most commonly used hyperosmolar agent. In the first few minutes, mannitol acts by improving the blood viscosity. There is improvement in cerebral blood flow and cerebral perfusion pressure is normalized. There is a resultant decrease in vessel diameter. The osmotic effects of mannitol, however, sets in after 30 minutess and lasts upto six hours. Deleterious effects of Mannitol include its diuretic effects, with the resultant depletion of circulating blood volume. There is also a potential for mannitol to escape into the extravasculqar compartment where it can worsen cerebral edema.

Pulsed mannitol therapy minimizes the extravascular extravasation and is superior to continuous mannitol infusion. Serum osmolality of more than 320 mosml cannot be achieved using mannitol[11]. The risk of acute tubular necrosis worsens, if the osmolarity crosses 320 mosml with mannitol therapy.

Hypertonic saline used for hyper osmolar therapy has been shown to result in superior outcomes. Serum osmolarity of 360 mosmol has been achieved with hypertonic saline and the diuresis and vascular depletion of mannitol has been obviated to a great extent[12]. In addition to its osmotic effects, mannitol also modulates CSF production and resoption, promotes tissue oxygen delivery and modulates inflammatory and neurohormonal responses.

The high brain tissue osmotic pressures in the centre of an area of contusion(around 390 mosml) would result in an edema relatively resistant to osmotic therapy.Surgical evacuation of these contusions would appear optimal, but the risk of surgical trauma inducing further cellular disruption and increasing the load of idiogenic osmoles has to be weighed against the benefits of removal of broken down cells and hematoma.

Antiepileptic drugs: These drugs prevent early onset seizures, but do not influence the potential for epileptogenesis. Seizures in the setting of increased ICP may be catastrophic and may lead to irreversible neurologic sequelae. In neurotrauma management units with intensive monitoring and ICP control, the addition of phenytion may not significantly contribute to seizure prevention even in the acute phase[13].

Neuroprotection: Both barbiturate coma and hypothermia have been used with the aim of decreasing the cerebral metabolic rate. Hypothermia has been explored as a means of neuroprotection and neuronal onservation in TBN[14]. Mild and moderate hypothermia may impede the inflammatory cascades which lead on to necrotic cell death[13]. Barbiturate coma (burst suppression on EEG) often results in significant hypotension which may impede cerebral perfusion. Effective and safe protocols for hypothermic neuroprotection are still being evolved.

CSF Drainage: An intraventricular catheter may be used for ICP monitoring. If ICP rises catastrophically, drainage of a few cc of CSF often help abort the potential ravages of sustained intracranial hypertension.

SURGICAL MANAGEMENT

(a) The role of decompressive craniectomy

Decompressive craniectomy is an effective tool in the reduction of ICP[14]. The procedure aims at negating the pressure volume relationship of the closed cranial cavity. A large bone flap is removed and the dura is expanded with the help of autologus or artificial tissue. Decompressive craniectomy per se does not tackle the pathological brain swelling. However, increasing the size of the container it alleviates the effects of raised intracranial pressure. Decompressive craniectomy is indicated in a patient with a GCS of 13 or less with a midline shift of more than 5mm. a bifrontal decompressive craniectomy may be used to decrease ICP In cases with generalized edema and central herniation[15].

(b) Surgical evacuation of hematoma and hemorrhagic contusions

The theoretical benefit of removing the contused brain include the removal of the edema producing osmotic load and the abolition of necrotic and apoptotic cascades triggered off by the products of blood degradation. Surgical excision of a contusion is best done conservatively with minimal or no trauma to surrounding brain tissue. The surgical evacuation of a brain contusion is ideally done through a limited and optimally placed cortical incision. The accumulation of blood and debus is dominantly subcortical and a limited cortisectomy suffices for optimal decompression. Hemorrhagic necrotic brain tissue may be sucked out through an appropriately placed pial-cortical window. Conservative contusectomies are best combined with a decompressive craniectomy.

There is only a limited role of intracranial pressure monitoring after a decompressive craniectomy . Bulge of the swollen brain is easily appreciable clinically. This is adequate for following the cause of brain swelling post operatively. Postcraniectomy hydrocephalus and subdural hygromas are potential complications.

CONCLUSION

There is an osmotic load in an area of brain contusion caused by membrane and cellular structural disintegration products. This is unlikely to respond to standard hyperosmolar therapy. Rational treatment of mass-producing hemorrhagic contusions consist of conservative contusectomy along with abrogation of the volume induced pressure rise by a decompressive craniectomy.

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