Traumatic Intracranial Hypertension

Traumatic Intracranial Hypertension may be due to any of the following (alone or in various combinations):

1. cerebral edema

2. hyperemia: the normal response to head injury.

Possibly due to vasomotor paralysis (loss of cerebral autoregulation). Maybe more significant than edema in raised ICP.

3. traumatically induced masses

a) epidural hematoma

b) subdural hematoma

c) intraparenchymal hemorrhage (hemorrhagic contusion)

d) foreign body(e.g.bullet)

e) depressed skull fracture

4. hydrocephalus due to obstruction of CSF absorption or circulation

5. hypoventilation (causing hypercarbia → vasodilatation)

6. systemic hypertension (HTN)

7. venous sinus thrombosis

8. increased muscle tone and Valsalva maneuver as a result of agitation or posturing → increased intrathoracic pressure → increased jugular venous pressure → reduced venous outflow from head

9. sustained posttraumatic seizures (status epilepticus).

A secondary increase in ICP is sometimes observed 3–10 days following the trauma, and may be associated with a worse prognosis.12 Possible causes include:

1. delayed hematoma formation

a) delayed epidural hematoma

b) delayed acute subdural hematoma

c) delayed traumatic intracerebral hemorrhage (or hemorrhagic contusions) with perilesional edema: usually in older patients, may cause sudden deterioration. May become severe enough to require evacuation

2. cerebral vasospasm

3. severe adult respiratory distress syndrome (ARDS) with hypoventilation

4. delayed edema formation: more common in pediatric patients

5. hyponatremia