Traumatic intracerebral hemorrhage

Definition

AKA Hemorrhagic contusion.

(TICH). The definition is not uniformly agreed upon. Often considered as hyperdensity areas on CT (some exclude areas <1 cm diameter).

Traumatic intracerebral hemorrhages is a traumatic intracranial hemorrhage result from either nonpenetrating or penetrating trauma to the head.

Examples:

Deep Brain Stimulation,......

see Frontal traumatic intracerebral hemorrhage.

Epidemiology

Traumatic intracerebral hemorrhage epidemiology.

Classification

see Delayed traumatic intracerebral hemorrhage.

Risk factors

The presence of APOE ε4, an elevated international normalized ratio, and a higher glucose level (≥ 10 mmol/L) are predictors of Progressive traumatic intracerebral hemorrhage. Additionally, APOE ε4 is not associated with traumatic coagulopathy and patient outcome.

Diagnosis

Computed tomography perfusion imaging (CTP) is a useful, fast, and appropriate method in evaluating perfusion of pericontusional hypodensity area that may help the treating physician to provide an appropriate treatment to the patient.

The development of brain CT allowed improvements in the diagnosis and characterization of traumatic intracerebral hemorrhage, and the traumatic brain injury field is being further revolutionized by the development and refinement of brain MRI.

Traumatic intracerebral hemorrhages frequently coexist with extracerebral hemorrhages. Frequently, clinical manifestations of traumatic intracerebral hemorrhage depend on the severity of traumatic brain injury.

Cerebral cortical contusions are one of the most common computed tomography (CT) findings in head injury.
It is a bruise of the brain tissue.

Like bruises in other tissues, cerebral contusion can be associated with multiple microhemorrhages, small blood vessel leaks into brain tissue.

The expression of caspase 3 and HAX-1 after cerebral contusion has time sequential regularity, which may provide new evidence for forensic diagnosis of cerebral contusion interval \(^6\).

Results revealed that at 2 hours after cerebral contusion and laceration injury, aquaporin 4 expression significantly increased, brain water content and blood-brain barrier permeability increased, and the number of pinocytotic vesicles in cerebral microvascular endothelial cells increased. In addition, the mitochondrial accumulation was observed. As contusion and laceration injury became aggravated, aquaporin 4 expression continued to increase, brain water content and blood-brain barrier permeability gradually increased, brain capillary endothelial cells and astrocytes swelled, and capillary basement membrane injury gradually increased. The above changes were most apparent at 12 hours after injury, after which they gradually attenuated. Aquaporin 4 expression positively correlated with brain water content and the blood-brain barrier index. This experimental findings indicate that increasing aquaporin 4 expression and blood-brain barrier permeability after cerebral contusion and laceration injury in humans is involved in the formation of brain edema \(^7\).

**Treatment**

see Traumatic intracerebral hemorrhage treatment.

**Outcome**

Traumatic intracerebral hemorrhage outcome.

**Case series**

Twenty-two patients with traumatic cerebral contusion (diagnosed on initial noncontrast head computed tomography [CT]) who initially did not require surgical intervention were enrolled in this study. Contrast-enhanced and perfusion CT scans were performed within 6 hours of injury, and follow-up noncontrast CT scans were performed at 24 hours and 72 hours.
In each noncontrast CT scan, the volumes of the contusion hemorrhage and edema were calculated using computerized planimetric techniques. The initial Glasgow Coma Scale, hemorrhage progression, clinical deterioration, and the need for subsequent surgery were recorded. The early radiologic findings were compared with these parameters and functional outcome at 6 months to identify predictive radiologic signs. CE was present in 9 of 22 patients (41%) and was highly associated with hemorrhage progression (p < 0.05), clinical deterioration (p < 0.01), and need for subsequent surgery (p < 0.01). In addition, patients with CE had a greater volume of edema at 24 hours (p < 0.01) and 72 hours (p < 0.01) than those who did not have CE. However, CE was not found to be associated with poor outcome.

Early parenchymal CE is associated with hemorrhage progression, cerebral edema, clinical deterioration, and need for subsequent surgery. These patients should be monitored closely, and early surgery may be needed if deterioration occurs. Further elucidation of the pathophysiology is needed to formulate effective treatment for these high-risk patients.

In severe traumatic brain injury (TBI), contusions often are worsened by contusion expansion, or “hemorrhagic progression of contusion” (HPC), which may double the original contusion volume and worsen outcome. In humans and rodents with contusion-TBI, sulfonylurea receptor 1 (SUR1) is upregulated in microvessels and astrocytes, and in rodent models, blockade of SUR1 with glibenclamide reduces HPC. SUR1 does not function by itself, but must co-assemble with either KIR6.2 or TRPM4 to form KATP (SUR1-KIR6.2) or SUR1-TRPM4 channels, with the two having opposite effects on membrane potential. Both KIR6.2 and TRPM4 are reportedly upregulated in TBI, especially in astrocytes, but the identity and function of SUR1-regulated channels post-TBI is unknown. Here, we analyzed human and rat brain tissues after contusion-TBI to characterize SUR1, TRPM4 and KIR6.2 expression and, in the rat model, to examine the effects on HPC of inhibiting expression of the three subunits using intravenous antisense oligodeoxynucleotides (AS-ODN). GFAP immunoreactivity was used to operationally define core versus penumbral tissues. In humans and rats, GFAP-negative core tissues contained microvessels that expressed SUR1 and TRPM4, whereas GFAP-positive penumbral tissues contained astrocytes that expressed all three subunits. Förster resonance energy transfer imaging demonstrated SUR1-TRPM4 heteromers in endothelium, and SUR1-TRPM4 and SUR1-KIR6.2 heteromers in astrocytes. In rats, glibenclamide as well as AS-ODN targeting SUR1 and TRPM4, but not KIR6.2, reduced HPC at 24 hours post-TBI. Our findings demonstrate upregulation of SUR1-TRPM4 and KATP after contusion-TBI, identify SUR1-TRPM4 as the primary molecular mechanism that accounts for HPC, and indicate that SUR1-TRPM4 is a crucial target of glibenclamide.

Case reports

2015

Petrela et al. report a complication of catheter ablation that, to their knowledge, has never been previously reported. A 63-year-old man had undergone successful transvenous catheter thermoablation for atrial fibrillation. The patient remained well until 3 days prior to further admission when he noticed itching in the right frontal area of his scalp. On palpating his scalp, he discovered a metallic body projecting out of it and he proceeded to extract 20 cm of wire from his head. The following day a progressive left hemiplegia developed, and the patient experienced a deteriorating level of consciousness. A CT scan of the brain showed a right frontotemporal intraparenchymal hemorrhage and revealed a metallic structure in the middle of the hematoma. The hematoma was evacuated and a decompressive craniectomy was performed. The guidewire was identified, but it was only possible to extract part of it. It was covered by fibrous tissue, secondary to inflammatory
reaction. To the authors' knowledge, this is the first report of guidewire-induced brain hemorrhage. The guidewire apparently had not been removed and had spontaneously migrated from the heart to the brain and beyond to the scalp where it then exited the patient's head. The patient had been well before he attempted to pull out the wire. Earlier identification of the iatrogenic complication of a retained guidewire might have prevented the fatal outcome in this case.

**Forensic medicine**

In the forensic medicine, objective and, if possible, the most accurate determination of the age - the time of the brain contusion, has practical significance. In our previous work, we discussed the importance of the neuron cytoskeleton proteins - neurofilaments, in this area. The purpose of this paper is to present the possibilities of using the phenomenon of angiogenesis in the brain contusions, to determine its age, on the basis of previous studies in animal models and in human biological material. The current review of the literature showed no conclusive data that would allow use morphological changes in angiogenesis to determine the age of the brain contusion in forensic medical practice. For these reasons, it is reasonable to take a broader research on the human material.

