Early spontaneous brainstem hemorrhage following widespread brainstem ischemia

Sir,

Hemorrhagic transformation (HT) is a well-known complication from ischemic stroke, usually occurring in the first 24 h after an infarct for parenchymal hematomas. Known risk factors for HT include size of the infarct, embolic etiology, early hypodensity on computed tomographic (CT) scan, use of reperfusion therapy, and early use of anticoagulation. Hemorrhagic transformation, however, has only rarely been reported involving the brainstem.

A 76-year-old man with a past medical history of hypertension, coronary artery disease, a permanent pacemaker, and chronic renal insufficiency presented to the hospital with coma. The patient had no history of stroke, transient ischemic attack, or intracerebral hemorrhage (ICH). He presented to the hospital at 2 ½ hours after onset with BP 180/72 mm Hg, HR 64 per min, RR 16 per min. His neurological examination revealed a fixed dilated left pupil, a prosthetic eye on the right, equivocal oculo-cephalic response, no corneal reflexes, and a present gag reflex. He had minimal symmetric flexor withdrawal response in the arms to pain, triple flexion in the legs, and had no response to verbal commands. He was intubated for airway protection shortly after arrival. CT scan of the head [Figure 1a and b] performed at 3 h after onset revealed no infarct or hemorrhage, though retrospective review may indicate a hyper-dense basilar artery. Laboratory values were normal, including urine toxicity except for a creatinine of 2.0 mg/dL. He was treated with 325 mg of aspirin in the emergency room. He received no other anti-thrombotic treatment. Magnetic resonance imaging was not attempted secondary to the patient’s pacemaker. The CT angiography of the head and intra-arterial thrombolysis were not attempted given a non-focal examination, unclear circumstances of the event as it occurred during sleep, and elevated creatinine value. The differential diagnosis included hypoxic-ischemic injury from unwitnessed cardiac arrest, toxin ingestion, and non-convulsive status epilepticus. Neurological examination remained unchanged. A CT scan of the head was performed 8 h later to rule out evolving ischemic changes [Figure 1c and d]. There was interval evolution of pontine and mesencephalic hemorrhages. Subsequent CT scan performed at 24 h [Figure 1e and f] indicated extension of the hemorrhage into the third and lateral ventricles, with significant edema and herniation of the cerebellum. The neurological examination remained unchanged, including an intact gag reflex and spontaneous irregular respirations, presumably from intact medullary function. Based on advanced directives, comfort care measures were initiated and he died eight days after the initial insult. The family did not consent to autopsy.

The mechanism by which HT occurs has been studied in animal models of arterial occlusion and cardioembolic stroke in humans. During a period of arterial occlusion, there is an initial accumulation of anaerobic metabolites and free radicals, causing local tissue damage. This is followed by an inflammatory phase, in which there is expression of selectin leukocyte adhesion molecules by the vascular endothelium, plasmin-mediated loss of laminin and fibronectin, and release of matrix metalloproteinases. This leads to a degradation of basal lamina components of the cerebral microvasculature, breakdown of the blood brain barrier, and transmigration of leukocytes and erythrocytes into the infracted area. With restoration of blood flow, which typically leads to hyperperfusion, it is thought that there is a further acceleration of this process due to formation of reactive oxygen species, allowing for greater extravasation of...
blood products into the affected tissue.\cite{5}

In the brain the most susceptible regions to ischemic injury are the hippocampus, Purkinje cell layer in the cerebellum, basal ganglia and thalamus. These areas have been previously described as developing HT.\cite{6} Previous reports have described HT in the brainstem, however, extension beyond the pons, including into the ventricular system has not been described other than in hemorrhages from AVM or hypertension.\cite{7}

Though most brainstem hemorrhages are primary hypertensive hemorrhages, the differential diagnosis should include the possibility of HT. The patient’s initial symptoms were likely related to brainstem ischemia with subsequent HT as there is no other clear explanation for the interval appearance of the patient’s ICH. Spontaneous HT is a rare, but potential complication of vertebro-basilar territory ischemia. Clinicians should suspect it when a patient with an ischemic infarct in this region has a sudden deterioration.

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References


Sudden appearance of idioventricular rhythm during inhalational induction with halothane in a child with congenital cataract

Sir,

An 11-year-old American Society of Anesthesiologists I female child with bilateral congenital cataract was scheduled for lens extraction under general anesthesia. Inhalational induction was done with halothane, oxygen, nitrous oxide using a semiclosed circle system (fresh gas flows of 6L/min). The child was asked to take deep breaths and halothane was