Disseminated intravascular coagulation following cranial trauma

Case report

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Cranial and spinal trauma resulted in disseminated intravascular coagulation (DIC) in a 78-year-old man, causing widespread bleeding and incoagulable blood. Traumatized brain tissue was found in the lumina of dural venous sinuses. The mechanisms of DIC are reviewed. It is suggested that intravascular release of potent cerebral thromboplastin contributed to the severity of DIC in this patient, by causing activation of the extrinsic clotting system. Intrasinus brain tissue in cases of human trauma has not previously been reported.

**KEY WORDS** - brain injury - skull fracture - sinus thrombosis - disseminated intravascular coagulation

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**Disseminated intravascular coagulation (DIC)** may be associated with various forms of trauma to the brain. Excessive widespread and massive bleeding followed severe cranial and spinal injury in the patient reported here. Traumatized brain tissue was found in the lateral and sagittal dural venous sinuses. A direct cerebral source of thromboplastin is therefore suggested as the exacerbating mechanism of DIC in this patient with incoagulable blood.

**Case Report**

This 78-year-old man fell from a 20-foot ladder, striking his occiput on the concrete pavement. The accident occurred at 4:00 p.m. He was comatose, and was promptly taken to a local hospital. An intravenous infusion of fluid was started, and a Foley catheter and endotracheal tube were placed. He arrived at Vanderbilt Hospital 2 hours later.

*Admission.* The patient was pale and unresponsive, his blood pressure was 138/90 mm Hg, pulse 60/min, and respirations 28/min and regular. A large subgaleal hematoma was present in the left frontoparietal region. Blood oozed from an open depressed skull fracture in the occiput. He was bleeding severely from the nose, oropharynx, right ear, and sites of venipuncture; extensive ecchymoses appeared on the upper extremities, and petechiae were seen on the legs and thorax. He had proptosis of the left eye, and the conjunctiva was chemotic and hemorrhagic. The right pupil was normally reactive, the left fixed and dilated. The upper and lower extremities were flaccid except for extensor movements of the shoulders. The abdomen was distended.

A subclavian line was placed. Venipunctures were made for blood analysis. Each puncture site bled profusely, resulting in a hematoma. The Foley catheter drained grossly bloody cerebrospinal fluid (CSF). Peritoneal tap also revealed bloody fluid containing 1700 white blood cells (WBC's) and 470,000 red cells per cu mm. Lidocaine was used to counteract premature ventricular contractions. Blood was not given.

X-ray examination of the skull revealed a diastatic fracture, with depression of the left parietal and occipital bone. Linear fractures extended anteriorly and bilaterally from the depression. A chest film was normal. The C-4 vertebra was dislocated on C-5. An intravenous pyelogram established that the kidneys were not functioning.

The blood count revealed 19,300 WBC's,
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hemoglobin 13.9%, and platelets 94,000/cu mm. Initial blood gases were pH 7.43, pCO₂ was 28 mm Hg, and pO₂ was 69 mm Hg. Repeated data 30 minutes later were 7.27, 38 mm Hg, and 62 mm Hg, respectively. The following blood studies were done: glucose 255 mg/100 ml, urea nitrogen 19 mg/100 ml, sodium 136 mEq/liter, potassium 3.7 mEq/liter, chloride 108 mEq/liter, CO₂ 19 mM/liter, total bilirubin 1.6 mg/dl. The serum amylase was 122 IU and the amylase in the peritoneal fluid was 3 IU. The blood sent for typing and crossmatch was incoagulable. Prothrombin time and partial thromboplastin time were greater than 800 seconds.

The patient became hypotensive 3½ hours after the accident, had a cardiopulmonary arrest, and died. The clinical impression was DIC secondary to severe cranial trauma.

Postmortem Examination. There was a comminuted left occipital skull fracture that coursed through the left temporal bone and into the left orbit. The fracture also extended across the torcular to the base of the skull on the right. A fragment of bone had lacerated the occipital cortex and the left lateral venous sinus. Subdural, subarachnoid, intraventricular, intracerebral, and upper pontine hemorrhages were found. Hemorrhagic congestion of the lungs and an upper gastrointestinal hemorrhage were present. The capsule of the liver and the renal pelvis had peteciae. A mesenteric hematoma was associated with 240 cc of serosanguinous fluid. There was an old posteroseptal myocardial infarct. The right coronary artery was thrombosed for 3 cm in its proximal course, beginning 0.5 cm distal to the point of origin.

Microscopically, cerebral edema and hemorrhage were present. Sections of the left lateral and superior sagittal sinuses showed traumatized brain tissue within the lumina (Fig. 1). The lung revealed hemorrhagic congestion and edema. Pulmonary arterial fat emboli were present. The kidney contained fibrin thrombi within glomerular capillaries. There was evidence of acute renal tubular necrosis. The liver had mild fatty change. The bone marrow was normal. The myocardium of the posteroseptal left ventricular wall was ischemic. The right coronary artery was occluded by an acutely ruptured atheromatous plaque.

Discussion

Disseminated intravascular coagulation is a syndrome precipitated by various diseases resulting in a pathological activation of the hemostatic system. Fibrin thrombi are formed, clotting factors consumed, platelets decreased, and the fibrinolytic system activated. The DIC syndrome is initiated by at least three mechanisms: 1) endothelial cell injury activates Hageman's factor XII and the intrinsic clotting system; 2) tissue injury activates the extrinsic clotting system; and 3) erythrocyte, leukocyte, or platelet injury releases phospholipid, a component necessary for the intrinsic and extrinsic systems (Fig. 2). An interplay of all three mechanisms resulted in DIC in our patient.

Activation of the extrinsic system by cerebral thromboplastin incites pathological thrombin generation. Thrombin cleaves fibrinogen to fibrin and aggregates platelets. Thrombosis ensues with consumption of clotting factors such as fibrinogen, and loss of platelets. Intracerebral thrombosis was not encountered in our patient, but this absence was considered to be secondary to increased fibrinolysis. Thrombin also activates plasminogen, providing a link between the hemostatic and fibrinolytic systems. Fibrinolytic activity of the brain is low, but animal studies have shown enhancement after crush injury. Fibrinolysis results in formation of fibrin split products, which inhibit thrombin. Enhanced fibrinolysis, inhibition of thrombin, and consumption of coagulation factors then explain why the patient developed profuse hemorrhage rather than thrombosis.

The brain contains high concentrations of tissue thromboplastin. Cerebral damage has therefore been suggested by various authors as a cause of DIC in cases of cranial trauma, encephalomalacia, and other cerebral lesions. Intravenous injection of potent cerebral thromboplastin in experimental animals has caused immediate DIC. Goodnight, et al., offered evidence of defibrination in nine of 13 patients who had destruction of brain tissue confirmed by direct inspection. Defibrination, however, was not observed in 13 other patients after cerebral trauma but without destruction. These authors theorized that disruption of normal brain barriers allowed circulating blood to come in contact with cerebral thromboplastin after severe trauma causing laceration of brain tissue.

The laboratory diagnosis of DIC is based on three criteria: prolonged prothrombin time, decreased platelets, and low fibrinogen level. A fibrinogen level was not measured in this case because of the brief...
hospital course. The platelet count was 94,000/cu
mm, prothrombin time was greater than 800 seconds.
Prothrombin time measures factors in the extrinsic
system, and is prolonged only when factors are below
a critical level or when an anticoagulant, such as
heparin or fibrin split products, is circulating. Our patient did not have a history of a
bleeding disorder or severe liver disease to account for
reduced clotting factors, and heparin was not used.
Prolongation of prothrombin time, therefore, was
related to formation of fibrin split products and con-
sumption of clotting factors, both phenomena of DIC.
The fall of the patient was probably precipitated by
an acute thrombus in the right coronary artery. The
subsequent clinicopathological findings are consistent with DIC. Cerebral crush injury led to introduction of traumatized brain tissue into the lateral and sagittal
sinuses, and release of potent cerebral thromboplastin.
The patient then exhibited signs of a diffuse bleeding
Diathesis, oozing uncontrollably from traumatized
regions, the oropharynx, and venipuncture sites. Gross
hematuria developed, and an intravenous pyelogram
revealed that the kidneys were not functioning. Severe
hemorrhages also occurred in the brain, lungs, and up-
per gastrointestinal tract. Clinically, the patient was in
a state of consumption coagulopathy. These findings,
coupled with a decreased platelet count and prolonged
prothrombin time, strongly indicate DIC. The severity
of the clotting defect led to incoagulable blood.

This case is unique because of the direct demonstra-
tion of intrasinus brain tissue as a source for release of
cerebral thromboplastin into the blood stream. The
extrinsic clotting system was thereby pathologically
activated.

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