Use of cryoprecipitate coagulum to control tumor-bed bleeding

Case report

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The case is presented of a 44-year-old woman who underwent reoperation under cardiac standstill for a recurrent left sphenoid wing meningeal hemangiopericytoma. Because of persistent accumulations of clot with clinical deterioration and shift on computerized tomography scanning, the patient was returned to the operating suite twice. At the second reoperation, hemostasis was finally achieved through the instillation of admixed cryoprecipitate, calcium, and activated thrombin.

KEY WORDS □9 hemangiopericytoma □9 hemostasis □9 cryoprecipitate coagulum □9 thrombin

Acheying hemostasis in large operative brain wounds can be difficult. The problem can be compounded in patients with coagulopathies, previous exposure to radiation, chemotherapy, and thrombocytopenia. Cardiac standstill can be a useful management technique but it does not guarantee control of postoperative clot accumulation. The following case illustrates this difficult problem and offers a solution.

Case Report

This 36-year-old right-handed woman presented to the Neurosurgical Section in November, 1974, with symptoms of left-sided headaches, nausea, and vomiting. She was found to have a large vascular tumor attached to the floor of the middle fossa (Fig. 1). The major feeding vessels from the external carotid artery were embolized, and the external carotid artery on the left was ligated prior to an attempted total resection of the mass through a left temporal craniotomy. Pathological findings were reported as an atypical, very cellular meningioma. She did well postoperatively except for mild dementia and temporal lobe epilepsy, well controlled with Dilantin (phenytoin). In March, 1978, she presented with diplopia accompanied by a left facial burning pain, and in March, 1979, she was readmitted because of increased facial pain, decreased hearing on the left, decreased taste on the left side of her tongue, worsening diplopia, and dysnomia. A computerized tomography (CT) scan showed recurrent tumor in the left temporal fossa. Arteriography disclosed intense tumor vascularity. The tumor was judged inoperable at that time, and radiation along with cis-platinum chemotherapy was administered. Her symptoms abated.

In July, 1982, she was reexamined for a gradual recurrence of her symptoms with added mental difficulties. A CT scan at that time disclosed increased growth of the left temporal fossa tumor with accompanying hemispheric edema and encephalomalacia with shift across the midline (Fig. 2 left). In November, 1982, she was readmitted for an attempt at complete removal of the recurrent tumor under hypothermia and cardiac standstill.

First Operation. On November 22, 1982, the left temporal fossa was entered through the previous craniotomy site. There was no evidence of dural invasion. Four to 5 cm of the left temporal tip was resected and the tumor was found to occupy most of the temporal fossa. There was no evidence of invasion into the brain or base of the brain. The tumor appeared to arise from the middle third of the sphenoid ridge and did not involve the carotid artery. Conventional resection was employed until major bleeding vessels were encountered at the tumor bed. At this point the patient was
cooled, and placed on femoral-femoral cardiac bypass by a cardiothoracic surgeon. The resection was then quickly carried out with no cerebral circulation and the brain temperature registered 15°C by probe. The major vasculature supplying the tumor bed could not be visualized and was thought to be retracted into the parenchyma. With completion of tumor removal, the bypass pump was turned on and off so as to locate and coagulate bleeding vessels in the tumor bed. Bleeding from one artery in particular was persistent, despite clipping and coagulation. After 1½ hours of turning the bypass pump on and off in this fully anticoagulated patient, it was decided that she should be rewarmed and cardioplegia be discontinued. With considerable effort a reasonably dry tumor bed was achieved, and the situation was thought to be tolerable. The dura was then closed over the remaining temporal lobe leaving a small gap inferiorly where the temporal lobectomy had left a vacant space. The bone flap was not replaced because of the long duration of surgery, previous radiation therapy, the high probability of accumulation of a hematoma, and swelling. The scalp flap was reapproximated over a suction drain, which was left in the subdural and epidural spaces inferiorly.

Second Operation. Six hours later, CT scanning disclosed a significant accumulated clot with marked edema and shift across the midline of the left hemisphere (Fig. 2 right). The patient remained unresponsive except for semipurposeful movements of the left extremities to deep pain. She was returned to the operating room for clot evacuation. There was both epidural and subdural blood communicating with an intraparenchymal clot. The brain surface oozed diffusely.

Fig. 1. Selective left carotid arteriogram made in November, 1974, showing intense tumor blush and hypervascularity of the left sphenoid wing meningeal hemangiopericytoma.

One distinct artery was found to be bleeding in the depth of the wound; this was clipped and coagulated. Minimal further brain was resected from the temporal lobe, no other difficulties were noted, and the wound was closed with a Hemovac drain in place.

The patient was returned to the intensive care unit in stable condition. She now exhibited equal and reactive pupils, semipurposeful movements on the left side in response to deep pain, and random right-sided movements. Intracranial pressure (ICP) measured via intraventricular monitoring was approximately 30 torr, and a Pentothal drip was initiated. A repeat CT scan in the morning once again revealed a reaccumulation of the hematoma with shift. The ICP remained elevated and required frequent ventricular drainage. Her platelet count had dropped to 63,000/cu mm, with the following coagulation profile: prothrombin time 14 seconds (normal level 11 to 13 seconds); partial prothrombin time 32 seconds (normal level 20 to 30 seconds); thrombin time 9 seconds (normal level 9 to 12 seconds); bleeding time 14 minutes (normal level 2.5 to 7.5 minutes); fibrinogen 165 mg/dl (normal level 200 to 400 mg/dl); and fibrin split products normal at less than 40 µg/dl. She received two units of fresh frozen plasma, one unit of cryoprecipitate, one eight-pack of platelets, and two units of packed red blood cells.

Third Operation. She was returned to the operating room for the second time for clot evacuation. Intraoperative findings were essentially the same as with the first clot. The bleeding sites were once again diffuse in nature. The wound was repeatedly irrigated with lactated Ringer's solution, and Gelfoam and Surgicel were applied. At this point, a mixture of cryoprecipitate solution, 1 cc of thrombin, and 0.5 cc of calcium was admixed and poured into the wound. Immediate coagulation of the material ensued with good adherence to the irregular brain surfaces. The brain remained...
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relaxed. Irrigation solution remained clear from that time on, and the wound was closed.

Postoperative Course. There was no further problem with bleeding. The patient had a prolonged period of unresponsiveness, right hemiplegia, and elevated ICP that could be controlled medically. She went on to develop a marked communicating hydrocephalus 1 month postoperatively, necessitating placement of a ventriculostriatal shunt. She was discharged alert but aphasic, with a right hemiplegia. Follow-up CT scans disclosed complete resolution of the cryoprecipitate coagulum and a large left frontotemporoparietal defect (Fig. 3).

Discussion

One unit of cryoprecipitate contains 145 units of Factor VIII and 250 mg of fibrinogen. Fibrinogen is an elongated ellipsoid molecule containing three pairs of polypeptide chains linked by disulfide bonds: αA (molecular weight (MW) 63,500); βB (MW 56,000); and gamma chain (MW 47,000). Each chain is an oligosaccharide group linked through asparagine residues. In the conversion of fibrinogen to fibrin, thrombin hydrolyzes one specific arginine-glycine bond in each αA and βB chain to release fibrinopeptides A and B from the NH2-terminal ends of the αA and βB chains. This reaction is catalyzed by calcium and produces a fibrin monomer. Fibrin monomers aggregate to form a soft soluble fibrin clot. The final step in the production of the insoluble fibrin clot requires Factor XIII fibrin stabilizing factor, which is present on platelets and plasma. Platelet fibrin stabilizing factor is activated by thrombin and occurs much sooner than plasma fibrin stabilizing factor, which is activated through a process that requires calcium. The net result is the formation of covalent cross links between the fibrin subunit polypeptide chains.3

Cryoprecipitate coagulum has been developed by urologists as an extractable cast of the collecting system in coagulum pyelolithotomy. Fischer, et al.,1,2 have found that cryoprecipitate when mixed with thrombin and calcium at a ratio of 1 cc cryoprecipitate:2 units thrombin:1 mg calcium chloride provides enough strength to successfully extract stones from the renal pelvis. The above clot precursors are warmed to 37°C and are admixed prior to instillation. Estimates of the necessary volume of coagulum are based on the volume of saline required to distend the renal pelvis when the ureter is occluded at the ureteropelvic junction. Once

the thrombin-calcium solution is mixed with the cryoprecipitate, coagulation begins within 30 seconds, so instillation must be immediate. When firm, the coagulum is opalescent and can be visualized by intraoperative radiographs.

The remarkable hemostasis achieved in the above case in a large diffusely oozing brain wound has prompted us to begin investigations using cat brain models. We hope this new modality to achieve hemostasis in particular situations will prove to be a useful addition to the neurosurgical armamentarium.

References


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