Hetastarch Coagulopathy in a Neurosurgical Patient

To the Editor:—We wish to report a patient who developed a coagulopathy postoperatively, which we believe was most likely due to hetastarch. The patient, a 36-yr-old, 60-kg woman, underwent a subtemporal craniotomy for removal of a recurrent epidermoid cyst, the initial removal having been performed uneventfully 2 yr earlier. She had had two other operations, a cholecystectomy and a vaginal hysterectomy, within the past 5 yr without complications. Her only medication was cimetidine for a probable peptic ulcer. Her preoperative laboratory values, including a prothrombin time (PT), partial thromboplastin time (PTT), and platelet count, were normal. Anesthesia for the craniotomy consisted of nitrous oxide 60%, thiopental infusion 2100 mg iv, meperidine 100 mg iv, and a nondepolarizing muscle relaxant iv. Fluid replacement during the 7-h operation consisted of lactated Ringer’s solution, 700 ml, and hetastarch 6%, 2000 ml iv.

Near the end of the operation, the surgeons noted unusual difficulty obtaining hemostasis. Coagulation studies showed a PTT 46 s (normal 34 s) with a normal PT and platelet count. Despite the administration of three units of fresh frozen plasma (FFP), the PTT increased to 56 s. Additional coagulation abnormalities included an increase in fibrin split products to 20 mcg/ml (normal 10) and a shortened thrombin time (12 s). During the night, the patient developed a right hemiparesis and anosmia. An emergency head CT scan showed a large hematoma in the left temporal lobe with a moderate mass effect. Because of the coagulopathy, the decision was made not to evacuate the clot. After administration of a total of 15 units of FFP over the next 2 days, the PTT returned to normal. The patient was discharged 9 days postoperatively, with her only disorder being a mild expressive dysphasias.

Hetastarch 6%, a heterogenous mixture of synthetic polysaccharides resembling glycogen, produces effective, prolonged intravascular volume expansion (24–48 h), which is clinically equivalent to, but considerably less expensive than, albumin. Because of these effects, we have chosen to administer hetastarch for volume expansion in selected neurosurgical patients, where we believe that the administration of crystalloid solution might precipitate or exacerbate cerebral edema. Studies in human volunteers indicate that hetastarch may prolong the PTT, in association with a decrease in Factor VIII coagulant activity and related antigen, and a decrease in von Willebrand factor activity. We believe our patient’s course corresponds well with the known effects of hetastarch on coagulation: a prolongation of PTT, a decreased thrombin time, and evidence of accelerated fibrinolysis. While we cannot prove conclusively that the coagulopathy was due to hetastarch rather than some other coincidental cause, such as primary fibrinolysis or the excess release of brain phospholipids, we would like to alert anesthesiologists to this possibility. While our communication was under review, Symington, a consulting hematologist to a neurosurgery service, reported in a letter to the editor two neurosurgical patients who received hetastarch and developed bleeding complications. As a consequence of both our and Symington’s experiences, we are initiating a study of the effects of hetastarch on coagulation in neurosurgical patients.

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REFERENCES

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