A Case Report: Acquired Factor V inhibition after bovine thrombin exposure in a pediatric cardiac patient – the anesthetic implications.

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Introduction: The use of topical preparations of bovine thrombin is a widely acceptable practice for surgical hemostasis in many surgical procedures including cardiovascular, orthopedic, neurological and gynecologic procedures. At least 1 million patients in the United States are exposed to topical preparations of thrombin each year. Streiff and Ness reviewed published reports of factor V (FV) inhibitors in adults and found 40-66% of cardiac surgery patients and 20% of neurosurgery patients develop FV antibodies after bovine thrombin exposure. Surprisingly, the reported pediatric cases are rare and there are only twelve documented cases of factor inhibition after bovine thrombin exposure. We report a case of a child who presented with an intracranial bleed after developing FV inhibition from bovine thrombin exposure. We discuss the anesthetic implications and the perioperative management of this acquired coagulopathy.

Case: A 5 year old, 12 kg female former 33-week twin B infant with a history of tricuspid atresia, pulmonary atresia, nonrestrictive ventricular septal defect and transposition of the great arteries presented with two epidural hematomas after a fall. She had undergone previous surgeries including right Blalock Taussig shunt, PDA ligation, right bi-directional Glenn anastomosis and shunt takedown, and two weeks prior an extracardiac non-fenestrated Fontan at which time she was exposed to bovine thrombin without complications. Upon admission, CT of the head showed two epidural hematomas and her coagulation profile revealed PT 17, INR 1.4, PTT 48, Factor 5 level 7%. Surgery was delayed because of concern for the difficulty in correcting the coagulopathy due to factor inhibition with blood products alone. After consultation with hematoloy, the patient was started on intravenous immunoglobulin (IVIG), corticosteroids, fresh frozen plasma (FFP) drip, and scheduled platelet transfusions. Despite these measures, she developed a sudden change in mental status, exhibiting bilateral extensor posturing and was intubated. A repeat CT of the head revealed expanding hematomas with mass effect. She was taken to the OR emergently for evacuation of the hematomas. After induction of general anesthesia, central venous line and arterial line were placed. The FFP drip was maintained and packed red blood cells were transfused. A right parietal craniotomy and right suboccipital craniotomy with evacuation of the epidural hematomas was performed. The patient was extubated four hours later after hemostasis was assured and stable hemodynamics. She was discharged without neurological sequela ten days later. Postoperatively, she received 2 days of IVIG, daily FFP transfusions for 2 weeks and weaned from steroids in 3 weeks. Her Factor V levels were greater than 50% at 12 days after presentation and normal at 3 weeks.

Conclusion: Topical bovine thrombin can induce inhibitors to coagulation factors including FV. The inhibitors usually develop 7-10 days after a primary exposure and can last for several weeks to months. As more pediatric cardiac patients survive longer and undergo multiple exposures from bovine thrombin, we may see a rise in the number of patients with acquired factor inhibitors. Transfusion medicine specialists and hematologist serve an important role in the management of coagulopathies associated with thrombin exposures. Preparation for emergent cases of acquired factor inhibitors requires communication with blood bank and transfusion specialists, and potential treatment with IVIG, high dose steroids, and plasmapheresis. As newer commercial fibrin sealants become available,
education of the surgeon and medical community regarding the complications of bovine thrombin exposure may reduce the use of sealants and therefore, the incidence of FV inhibitors.

References: