A Case of Disseminated Intravascular Coagulation During Repair of Aortic Dissection

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DISSEMINATED INTRAVASCULAR COAGULATION DURING REPAIR OF AORTIC DISSECTION

CASE SUMMARY:

A 62 year old male presented to the emergency department with chest pain and was found to have a type A aortic dissection. Past medical history was significant for hypertension and alcohol liver disease. He was brought to the OR emergently for repair.

Preoperative CT angiography revealed a Stanford A aortic dissection from the noncoronary cusp to the distal aorta just distal to the origin of the inferior mesenteric artery with involvement of the left renal artery and associated left renal infarct. Diffuse hepatic steatosis was also noted on imaging.

On arrival, minimal history was obtained due to the emergent nature of the case. A pre-induction arterial line was placed with local anesthetic infiltration over the radial artery. Rapid sequence induction was performed with high dose fentanyl, Propofol and succinylcholine. The patient was endotracheally intubated, a transesophageal echocardiography probe was placed and right internal jugular vein cannulated for central venous access.

With minimal delay, a median sternotomy was performed and the patient was placed on cardiopulmonary bypass with the left subclavian artery as the arterial cannulation point and the right atrium as the venous cannulation point for cardiopulmonary bypass. Approximately 400 cc of hemopericardium was evacuated. The patient was placed on cardiopulmonary bypass (CPB). The entry point of dissection was identified just proximal to the noncoronary cusp. The patient was cooled to 22 degrees celcius, a Propofol bolus was administered to attain a BIS of 0. The dissection was repaired with BioGlue and dacron graft and CPB was restarted. Once the repair was complete, we separated from CPB and protamine was administered. The patient was noted to be significantly coagulopathic, ACT read > 999. Patient was transfusd with cryoprecipitate & FFP. Surgical sites were reassessed, no obvious surgical bleeding was noted but there was continuous oozing from all surfaces. On TEE, there was good contractility but an underfilled ventricle despite activation of massive transfusion protocol and volume resuscitation ongoing with a rapid insfuser. Repeat coagulation & TEG were sent to the lab. INR was elevated to 1.6, TEG showed increased K and decreased alpha angle for which additional cryoprecipitate was given. ACT improved to 700, the maximum amplitude was low â€” platelets were given as part of the massive transfusion protocol. Activated factor 7 was also given.

As resuscitation was continued in the OR, the volume status of the ventricle on TEE improved, however, the patient was still requiring pressors. The decision was made to pack the
chest and take the patient to ICU to continue resuscitation and come back for a second look later to reassess bleeding and do a delayed closure of the chest.

Resuscitation continued overnight, coagulation profile was corrected, volume status improved and the patient returned to the OR on POD1 for delayed closure which was uneventful. On postoperative day 6, the patient had progressed to the point of being considered for extubation. A fiberoptic bronchoscopy and bronchoalveolar lavage was performed for pulmonary toilet prior to extubation. After this procedure, the patient was noted to have falling blood pressures and increased chest tube output. A chest X ray was performed which showed increased left pleural effusion. An emergent CTA chest was performed which showed pneumothorax, and a large hemomediastinum. The patient was taken emergently back to the OR. The chest was reopened and hemomediastinum and hemothorax left greater than right was found and evacuated. An area of bleeding was identified in the region of the proximal suture line ad repaired.

DISCUSSION:
DIC AND AORTIC DISSECTION

Disseminated intravascular coagulation (DIC) is a rare but well recognized complication of aortic dissection. DIC occurs due to the endothelial disruption and exposure of blood to tissue factor in the nonendothelialized false lumen which causes release of cytokines which in turn activate the coagulation cascade. This results in formation of thrombin.

Time to intervention is inversely related to the fibrinogen level which explains why fibrinogen may be normal in the preoperative investigations. 1 study done in China and published in Journal of Cardiothoracic Surgery in June 2017 demonstrated that at the time of induction of anesthesia, Thromboelastogram (TEG) was shown to have a short R time which is indication of coagulation factor consumption in the pre-intervention period which proves that cardiopulmonary bypass is not the sole determinant of development of DIC in patients with aortic dissection. In spite of full heparinization, there is increased thrombin formation while on cardiopulmonary bypass. This ongoing consumption eventually leads to the coagulopathy observed in fulminant DIC. As part of the consumptive coagulopathy, thrombocytopenia is observed but the platelet function remains intact.

DIC related to aortic dissection may be present at the time of diagnosis, though it was a late finding in the index case. Up to 40 % of patients with aortic dissection will demonstrate elevated levels of fibrin degradation products but only 4% will experience significant bleeding and other derangement in lab values in keeping with DIC.

DIAGNOSIS OF DIC / CLINICAL PICTURE:

DIC is a syndrome diagnosed by combination of clinical and laboratory data.

Diagnosis of DIC is usually an assessment of:

- Platelet count (reduced)
- Fibrin Degradation products (increased)
- PT & APTT (Prolonged)
Fibrinogen (low)

Other abnormalities that may be observed include:

- Micropangiopathic changes on blood smear
- Shistocytes, helmet cells
- Prolonged thrombin time
- Reduced levels of coagulation factors VII, X, V and II (prothrombin)
- Reduced level of intrinsic anticoagulants antithrombin, protein C and protein S

In addition to non-surgical bleeding / “oozing” that is commonly associated with DIC, other organ systems may be affected due to thrombosis, hemorrhage or hypoperfusion. Associated organ dysfunction which may be noted include renal failure, liver failure, acute lung injury, adrenal failure and neurologic symptoms including delirium, coma or intracerebral hemorrhage.

TEG IN DIC:

Thromboelastogram will differ depending on the stage of DIC. Initially, there will be a procoagulant state with a short R time due to the release of coagulation factors. As these factors are consumed there will be a hypocoagulable state.

MANAGEMENT OF DIC:

The mainstay of treatment of DIC is to remove the inciting factor for continued coagulation and fibrinolysis.

- **Platelet transfusion:**
  
  For platelet counts of 50,000 x 10^9 or below with active bleeding, platelet transfusion is indicated. For patient who are not actively bleeding, or in a non-operative setting, a threshold value of 10-20,000 x 10^9 is usually the value for platelet transfusion.

- **Cryoprecipitate:**
  
  Transfusion of cryoprecipitate at a dose of 15-30 ml/kg is indicated for replacement of fibrinogen when the measured value is below 100. Replacement of 3 g (2 cryoprecipitate pools) of fibrinogen can be expected to raise plasma levels by 1g/l.

- **Fresh Frozen plasma**
  
  Useful for replacement of the consumed coagulation factors and contributes to volume replacement during brisk hemorrhaging.

- **Other agents**
In specific situations, agents such as recombinant factor VIIa, prothrombin complex concentrates, gabexate and antithrombin may be used in the setting of DIC. The evidence for these agents is variable and certain contraindications must be borne in mind for each agent.

References:
1. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4531585/