**Introduction:**
Disseminated Intravascular Coagulation, DIC, is poorly understood, difficult to manage, and associated with high mortality rates of 31-86%. Causes associated with higher survival rates are the better understood etiologies of septic abortion, neisseria infection, and acute promyelocytic leukemia.

**Learning Objectives:**
1) Management of DIC when the cause is unknown
2) Discussing if there should be limits on aggressive resuscitation of DIC with extensive use of blood products at the risks of high cost and significant resource depletion

**Case:**
51 yo male who recently traveled to North Carolina, presented with pain, urinary retention, and penile and scrotal swelling after coitus.

**Labs:**
Hb, INR, aPTT, fibrinogen, D-dimer, and platelets suggested severe anemia, thrombocytopenia, and DIC.

**Hospital course:**
During his 5-day hospitalization, the patient was given:
- 7 units PRBC (goal hemoglobin >10) (Figure 1)
- 7 units of platelets (goal platelet count >50,000) (Figure 2)
- 210 units cryoprecipitate (goal fibrinogen >150) (Figure 3)
- 16 jumbo units FFP to correct his INR and aPTT (Figure 4)
- Low dose heparin, 5mg/kg/hr to interrupt the coagulation cascade. (shown to benefit patients in DIC with severe sepsis)
- Antithrombin concentrate when antithrombin III levels were low
- Fibrinogen, antithrombin III, hemoglobin, and platelet count normalized, but INR, aPTT, and D-dimer remained elevated.

- Had hematuria and signs of thrombosis, including tender ecchymoses, mottled skin, blisters, and necrotic tissue throughout his extremities (Figures 5 and 6).
- Developed multiorgan failure with acute renal failure, confusion, intubation for airway protection, and heartblock with periods of sustained asystole.
- On HD5, his family decided on comfort care.

**Discussion:**
- Most research indicates that effective treatment of DIC is to treat its underlying cause.
- The patient was treated for presumed vibrio infection (blood cultures were negative) with antibiotics, this did not control his overwhelming systemic inflammatory response already underway that caused the hemorrhagic and thrombotic sequelae of DIC.
- DIC was treated with PRBCs, platelets, FFP, cryoprecipitate, low dose heparin, and antithrombin. Other treatment considerations were activated protein C and factor VIIa.

**Conclusion:**
- Despite medical advances, DIC remains difficult to treat, and research is ongoing to understand its pathophysiology, the effectiveness of blood products in its management, and other treatments that may decrease morbidity and mortality.
- The patient required at least 110 units of cryoprecipitate to bring his fibrinogen level above 150, and he would have likely required more FFP to correct the INR and aPTT if he was not made comfort care, but it is unclear if he would have survived even with these interventions.

**For further discussion:**
1) Since mortality in most causes of DIC is so high, should there be limits on aggressive resuscitation of DIC with extensive use of blood products at the risks of high cost and significant resource depletion?
2) Should families be allowed to switch to comfort care after starting down this road of aggressive resuscitation in DIC?

**References:**

**DIC – The Unknown**
Gunjan Patangay, MD, Tonya Fancher, MD, Jerry Powell, MD
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