CLINICAL VIGNETTE

COAGULOPATHY IN THE HOSPITALIZED PATIENT

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A 30-year-old male with multiple medical problems was admitted to the hospital due to shortness of breath. His history was complicated by lupus, end-stage renal disease (ESRD) on hemodialysis, cardiomyopathy with an ejection fraction of 25-30%, and multiple ICU admissions for complications of all these issues. His last admission was one month prior where he was medically optimized for congestive heart failure (CHF). Hematology was consulted due to a significant coagulopathy with INR=1.8 and PTT=50.

Review of his medications showed that he was not on any anticoagulants, including heparin or Coumadin. He reported anorexia but stated that he ate a well-balanced diet. He had no bleeding problems. On exam, his vital signs were unremarkable. He appeared older than his stated age, but otherwise, his exam was fairly unremarkable beyond some mild scleral icterus. His laboratories were consistent with his known renal disease. His liver function tests showed elevations of his transaminases, total bilirubin, and alkaline phosphatase. Review of his labs from his prior hospitalization showed that these same labs had been trending up during the end of his last visit as well and were much higher.

The exact etiology of a coagulopathy can be difficult to determine in the hospital patient due to multiple, concurrent medical issues. It is appropriate to start with a mixing study to determine if the diathesis is a factor deficiency versus an inhibitor issue. With a mixing study, the patient's plasma is combined 1:1 with normal, donor plasma and the coagulation tests are re-performed. Factor deficiencies easily correct due to the presence of adequate coagulation proteins in the donor blood. Conversely, inhibitors will affect both patient and donor plasma and the PT and/or PTT continue to be abnormal. In the patient above, there was correction of the PT and PTT with mixing, indicating a deficiency.

The clotting cascade is complex with the PT and PTT affected by different proteins. However, coagulation does converge on a common pathway (Factors X, V, II, and fibrinogen). Thus, since both the PT and PTT were abnormal, we assume that there was either a deficiency along the common pathway or a process affecting many coagulation factors. Deficiencies of individual factors along the common pathway are quite rare. However, acquired disorders affecting multiple proteins are not uncommon with the most frequent etiologies including Vitamin K deficiency, disseminated intravascular coagulation (DIC), and liver failure, all of which were feasible in this ill, hospital patient. It becomes easier to tease out the exact cause by remembering which factors are affected by these three disease states[1,2].

Vitamin K deficiency- Factors II, VII, IX, and X require Vitamin K for synthesis. Liver disease-All factors are produced by the liver except Factor VIII. DIC-All factors are consumed fairly evenly.

Thus, a good screening protocol is to check Factor V, VII, and VIII levels. Factor V is independent of Vitamin K, so low levels can be attributed to liver disease or DIC. Factor VIII is normal/high in liver disease and also independent of Vitamin K, so low levels are consistent with DIC. Low Factor VII and normal V and VIII support Vitamin K deficiency.

In the above patient, Factor V=15%, Factor VII=21%, and Factor 8=150%. These lab results supported a diagnosis of liver failure related to his cardiomyopathy and CHF. He received further dialysis with improvement in his LFTs and INR decreased to 1.4 and PTT to 40 by discharge.

REFERENCES

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