

Who is the Main Culprit of D-Dimer Elevation?

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Case Study

A 67-year-old man came to the emergency department of our hospital for "dyspnea for more than half a month". Physical examination at that time: sanguine, nephrotic facial features, mild anemia, lung breathing sound thick, right middle lungs can be heard and scattered in wet rales. The patient had undergone two endovascular aortic dissection operations in our hospital 2 years ago because of "descending thoracic aortic dissecting aneurysm". Others have no abnormalities. Patients were admitted to hospital for diagnosis: 1) Chronic kidney disease stage 5 Renal anemia, 2) Pulmonary infection, 3) Thoracic descending aortic dissecting aneurysm, 4) High risk group of hypertension grade 3.

Test

Complete blood count: GRAN% 81.0% H, LYM% 10.8% L, total RBC 1.88 T/L, Hb 53 g/L PL, HCT 15.6% L, RDW 17.7% H, PLT 112*10^9/L, PDW 9.50%. APTT 34.4s H, D-Dimer 4.71 mg/L H. CRP 63.9 mg/L H and PCT 0.35 ng/ml H. On the day of admission, the highest temperature was 38.3°C. Sulphur was given anti-infective treatment, however, fever again appeared, the peak temperature rose, and CRP was higher than before. Moxifloxacin was added for anti-infective treatment, covering G+ve bacteria and rare pathogens. There was no obvious abnormality in echocardiography. CT findings: chronic bronchitis; emphysema, multiple alveoli in both lungs; 2.

Two days after admission, gingival bleeding and multiple bleeding tendencies were observed. Clopidogrel and other long-term anticoagulants were suspended. In order to improve the coagulation function and prevent bleeding, 200 ml of frozen plasma of the same type was transfused.

However, 10 days later, there were repeated abnormalities in the four coagulation and D-dimer, positive 3P test, APTT 43.8s H, plasma fibrinogen FIB 3.78 g/L H and D-Dimer 88.00 mg/L H for activation of partial thromboplastin by four coagulation and D-dimer. Anti-infection, plasma transfusion, Regular hemodialysis, hypotension and other symptomatic treatment, the patient has not seen active systemic hemorrhage, the current condition slightly improved, but still need to be vigilant about DIC.

In this case, what is the cause of elevated D-dimer?

We performed thoracic triple CT and double-source thoracic pain triad screening: 1. After stent implantation of descending aortic aneurysm, a little contrast medium exudation was found in

slight inflammation in the middle lobe of the right lung (Figure 1).

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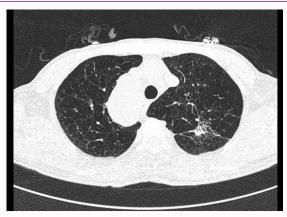


Figure 1: Slight inflammation in the middle lobe of the right lung



Figure 2: Right subclavian artery dissection involving the right vertebral artery orifice.



Figure 3: Right axillary artery dissection involving the right vertebral artery orifice.

the upper end of stent; 2. Right subclavian artery and axillary artery dissection involving the right vertebral artery orifice (Figure 2 and 3); excluding the possibility of pulmonary embolism. A little contrast medium exudation was found at the upper end of the stent.

Final interventional surgery findings that the increase of D-Dimer is caused by the formation of blood clots in the pseudolumen caused by dissection of arteries. Finally, clinicians should be aware of this potentially lethal complication of D-dimer elevation and the therapeutic options, because early diagnosis can improve prognosis. Routine triple CT assessments should be performed in patients with high D-Dimer presenting with hemodynamic instability.

Authors' Contribution

CH wrote the manuscript. All authors read and approved the final manuscript.