Disseminated intravascular coagulation in a patient with congestive heart failure

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Summary

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Disseminated intravascular coagulation (DIC) is a syndrome characterized by generalized excessive activation of coagulation pathways followed by their consumption, with activation of anticoagulation and fibrinolysis. It is a common syndrome associated with systemic inflammatory conditions. Its development in the context of severe heart failure has been scarcely reported. We here report the case of an 80-year-old man, previously diagnosed with dilated cardiomyopathy, was admitted with severe congestive heart failure and cardiac cachexia. In this context he developed disseminated intravascular coagulation. Laboratory alterations followed a course parallel to the clinical features of heart failure decompensation. No other cause for DIC was identified, so it is assumed that it developed in relation with marked impairment of organ perfusion. This case shows the association between congestive heart failure and disseminated intravascular coagulation. To our knowledge, only 7 similar cases have been reported.

Keywords: Congestive heart failure, Disseminated intravascular coagulation, Cardiac cachexia

Introduction

Disseminated intravascular coagulation (DIC) is defined by the systemic activation of blood coagulation, that results in the deposition of fibrin and the formation of microvascular thrombi in various organs. After a first thrombotic phase, able to cause multiorgan dysfunction, ongoing activation of the coagulation cascade leads to consumption and exhaustion of clotting factors and platelets, and, secondarily, to enhanced fibrinolysis. Depending on the nature of the triggering factor, its clinical presentation may span from an explosive, catastrophic process combining thrombotic and hemorrhagic features and multiorgan failure, to just a relatively mild, subclinical disorder, only detected by laboratory alterations.
The most frequent causes are systemic inflammatory conditions, such as obstetric disorders, metastatic neoplasms, massive traumatisms and sepsis [5,7] but there are other uncommon causes, as that reported below.

PRESENTATION OF CASE
An 80 year-old man presented with intense asthenia, progressive breathlessness and orthopnea since two months before admission. He also complained swelling of both legs and hypotension. Previous medical problems included the presence of dilated cardiomyopathy since at least 6 years before, and also an acute coronary event about five years prior to this admission. He also reported constipation in the last months, with some episodes of hematochezia. On physical examination he had hypotension (72/37 mmHg), jugular vein distension, crackles in the auscultation and edema on both legs, that progressively became worse during the hospital stay leading to anasarca, despite intensive therapy. The patient was also severely malnourished. Laboratory evaluation at admission showed mild normocytic anemia (Hb 11 g/dL, mean corpuscular volume (MCV) 94 fl), initially normal platelet count (208000/mm³); prothrombin time 84%; fibrinogen 492 mg/dL; serum creatinine 1.3 mg/dL, and raised N-terminal brain natriuretic peptide (NT-proBNP) levels (12700 pg/mL). Fourteen days after admission, platelet count fell down to 48000/mm³, prothrombin time to 40%, and fibrinogen to 283 mg/dL, afterwards got down to 283. In addition, these alterations were accompanied by a raised D-dimer levels (7064 ng/mL at the 19th day after hospital admission). All these data are consistent with DIC. Interestingly, the development of DIC matched the moment of maximum decompensation of heart failure, and also the analytical disorder improved with high doses of furosemide, as the clinical features of decompensated heart failure also did. Indeed, both the clinical manifestations of heart failure and the described alterations of clotting factors and fibrinolysis fluctuated in a parallel fashion during the hospital stay. One month after admission, the patient developed a nosocomial pneumonia, which killed him a few days later. Because of the reported episodes of hematochezia, a colonoscopy was performed, showing a non-stenosing colorectal neoplasia, 25 cm away from the anal margin, 15 mm of maximum diameter. We also performed a CT scan that revealed no metastasis, but severe heart failure, with cardiomegaly, interstitial and alveolar edema, pleural effusions, coronary atherosclerosis and indirect signs of low cardiac output. No alterations were detected by CT in the colon wall.

Discussion
The patient was admitted because of decompensation of severe congestive heart failure as the main problem, and a mild anemia, probably related either to the heart failure itself and/or the small intestinal neoplasm, which was only detected by colonoscopy, but not by CT due to its small size. During the hospital stay he developed a DIC, hardly attributable to any of the common causes of this condition: the neoplasia was small and localized, with no metastases, and no trauma or sepsis were present; the pneumonia that killed him was a final event. In addition, biochemical alterations consistent with DIC followed a course parallel to the clinical features of heart failure decompensation. For these reasons the most likely cause of DIC was the severe heart failure. This association is uncommon. Only 7 cases have been documented, and only 2 survived [1]. It should be mentioned that in all these 7 cases the patients had an intra-cardiac thrombus, and 5 of them had had an ischemic heart attack. In the CT of the case here reported, during hospital stay, there was no intra-cardiac thrombus, but it was indeed documented in an echocardiography performed 2 months before admission.

The association of DIC in the context of CHF is an uncommon phenomenon, with an obscure pathogenesis. Some authors [6] state that an intracardiac thrombus is a necessary condition for this syndrome to develop. It has been also hypothesized that coagulation might activate spontaneously, in a similar way as it happens in aortic aneurysms or hemangiomas. Possibly, endothelial hypoperfusion and hepatic congestion may trigger the clotting cascade and promote DIC syndrome [4]. The presence in this case of intense impairment of organ perfusion observed in the CT scan lend support to the hypothesis of a spontaneous activation of coagulation due to endothelial damage related to a very low cardiac output.

Conclusion
In conclusion we present a case of a patient with congestive heart failure associated with disseminated intravascular coagulation for which no other condition was identified. The development of this last syndrome may be explained by endothelial damage because of hypoperfusion, and spontaneous activation of coagulation. This case reinforces the possibility that both entities are related to each other, adding to the few other cases reported in the medical literature.

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