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The Challenge of Anticoagulation in Pulmonary Embolism Associated with Disseminated Intravascular Coagulation in the Same Patient: A Case Report

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ABSTRACT

The combination of pulmonary embolism and disseminated intravascular coagulation in the same patient poses an enormous challenge in therapeutic management because of the risk of thrombosis and risk of hemorrhage at the same time. We discuss this topic through a clinical case of an 85-year-old patient who is hospitalized with pulmonary embolism complicated by disseminated intravascular coagulation.

KEYWORDS

Pulmonary embolism; Disseminated intravascular coagulation; Anticoagulation

INTRODUCTION

One of the first case reports citing pulmonary embolism (PE) as an inducing factor for disseminated intravascular coagulation (DIC) was published in the 1980s, followed by a few more in the following two decades [1]. Pulmonary embolism (PE), most commonly caused by deep vein thrombosis of the legs, ranges from asymptomatic disease to massive embolism causing cardiac arrest and death. In massive pulmonary embolism, anatomical obstruction is the most important cause of compromised physiology [2].

Disseminated intravascular coagulation (DIC) is a serious clinical-pathological condition that complicates a range of diseases. It is characterized by a systemic activation of coagulation, leading to the formation of fibrin clots, the concomitant consumption of platelets and coagulation factors. Depletion of coagulation factors and thrombocytopenia can lead to bleeding, while fibrin clots and microvascular damage can lead to organ failure [2].

Consumption coagulopathy resembling disseminated intravascular coagulation (DIC) has been observed in patients with massive pulmonary embolism (PE) [2].

CASE REPORT

Mrs. FL, 85-year-old, operated on 2 weeks before her hospitalization for a placement of an intramedullary nail on a pertrochanteric fracture in the right lower limb, admitted to the cardiology department for a pulmonary embolism at high intermediate risk, put on anticoagulation based on low molecular weight heparin at a curative dose, complicated by a hematoma at the operative site after two days of treatment.

On admission, she was a NYHA stage III dyspneic patient, with BP 100/60 mmHg, Fc = 106 bpm , Arterial oxygen saturation = 70% in the open, under a mask at

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high concentration at 96%, with an ecchymosis and a hematoma on the level of the internal face of the hip opposite the surgical site (Figure 1). On the ECG, we note the presence of sinus tachycardia at 110 b/min, with an appearance of S1Q3, and negative T waves in V1, V2 and V3.

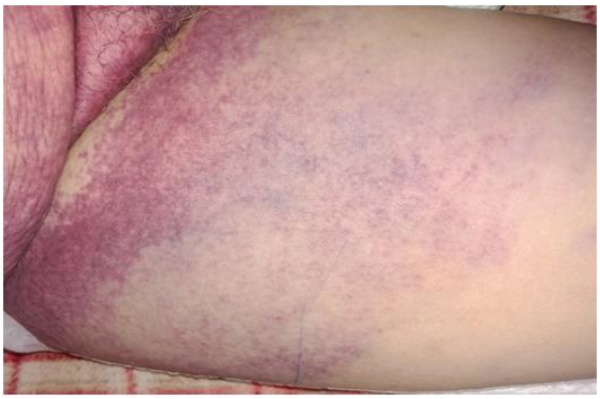

Figure 1: Hematoma of the right thigh.

The transthoracic echocardiography showed a conserved systolic and diastolic function of the left ventricle, LVEF = 50%, a dilated right ventricle in systolic dysfunction, a severe tricuspid insufficiency with a V_{max} of the TI at 4m/s, (Figure 2) systolic pulmonary arterial pressure at 67 mmHg, an IVC dilated to 28 mm.

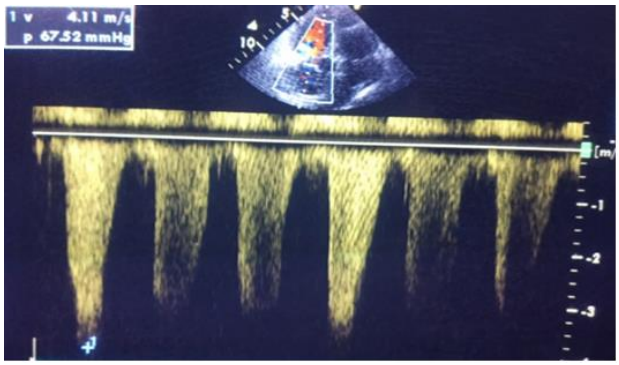

Figure 2: Systolic pulmonary pressure at 67 mmHg.

A thoracic CT angiography confirmed a bilateral pulmonary embolism, massive on the right with foci of middle lobar parenchymal infarction, Qanadli Score estimated at 35% (Figure 3).

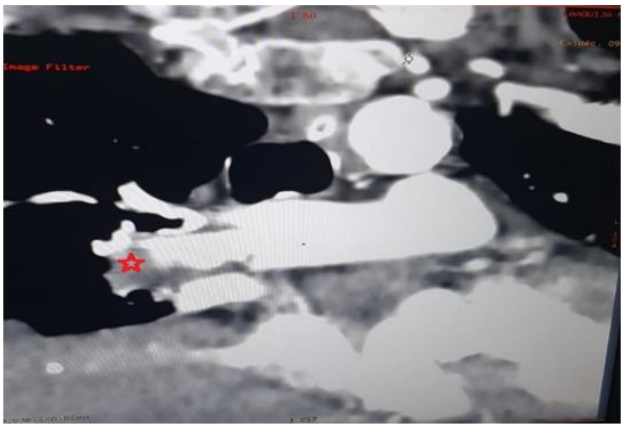

Figure 3: Thoracic CT angiography showing pulmonary embolism.

Routine laboratory tests revealed an anemia with hemoglobin level at 6.8 g/dl, thrombocytopenia at 36000/µL, blood cell count at 10120/µL C-reactive protein at 295 mg/l, low prothrombin level at 28%, activated partial thromboplastin time prolonged to 59 s, fibrinogen level at 0.5 g/l and D-dimers at 40,000 µg/l, Plasma creatinine at 6 mg/l and hepatic cytolysis with aspartate aminotransferase at 591 UI/L and alanine aminotransferase at 158 UI/L.

At this stage, we are basing ourselves on the diagnostic algorithm for "decompensated" DIC according to the International society for thrombosis and hemostasis (SCORE ISTH): The score is 6, we have retained the diagnosis of a clinical decompensated DIC and biological.

The patient received a transfusion of 3 blood pouches, 4 pouch platelets and fresh frozen plasma. We have opted for a therapeutic window to stop anticoagulant therapy.

One week after the transfusion and the end of the anticoagulant treatment, an improvement in the laboratory tests was noted: Hb at 10.6 g/dl, Platelets at 144000 µL, prothrombin level at 56%, activated partial thromboplastin time at 30 seconds.

Resumption of anticoagulation with low molecular weight heparin is restored.

DISCUSSION

The first score, based on current tests (platelet count, prothrombin time, fibrin degradation products, fibrinogen), was developed under the aegis of the Japanese Ministry of Health in the 1980s (Japanese Ministry of Health score and welfare, JMWH), a new version of this score having been reissued in 2005 (Japanese association for acute medicine score, JAAM). Using data from this early work, the International society for thrombosis and hemostasis (ISTH) proposed in 2001 a new scoring model that has since been the most evaluated. The expert committee responsible for establishing this new score wanted to:

- Emphasize the need to establish the presence of an underlying pathology known to be responsible for a DIC before calculating the score;
- Establish diagnostic criteria for two entities that are part of a continuum: "Compensated" or beginner DIC and "decompensated" or manifest DIC. The criteria establishing the diagnosis of “compensated” DIC had to be as sensitive as possible, while the diagnosis of “decompensated” DIC had to be more specific.
- A score based on widely used tests, identical to those used by the Japanese scores, although these lack the sensitivity to detect abnormalities that could lead to the diagnosis of DIC being considered "compensated" [3].

Diagnostic algorithm for "decompensated" DIC according to the International society for thrombosis and hemostasis (ISTH) [3]:

- Risk assessment: Does the patient have a pathology known to be associated with the presence of a DIC.
- If yes: Carry out the test; otherwise do not use this algorithm.
- Carry out tests for the overall evaluation of coagulation (platelet count, prothrombin time, fibrinogen, markers of fibrin degradation: Fibrin degradation products; D-dimers; soluble fibrin monomers).
- Evaluate test results.

Platelets (>100 = 0; <100 = 1; <50 = 2)

Markers of fibrin degradation (No increase: 0; Moderate increase: 2, Sharp increase: 3)

Prolongation of the prothrombin time (<3 seconds = 0; >3 seconds but 6 seconds = 2)

Fibrinogen level (>1 g/l = 0; <1 g/l = 1)

- Calculate the score.

If score ≥5: Compatible with a “Decompensated” DIC; repeat the score daily.

If score <5: Evokes without affirmation a DIC, repeat in 24 hours to 48 hours.

The use of the ISTH scoring system makes it possible to make an early diagnosis of DIC, even at the compensated stage, thus making it possible to identify high-risk populations in intensive care settings. These patients should then be closely monitored and benefit from specific additional diagnostic and therapeutic procedures [4].

In our case, the bleeding at the surgical site provided clinical evidence of coagulopathy and the CIVD score of >5 based on low platelet counts, low fibrinogen levels and decreased PT and increased D-dimers very well supported the presence of an overt DIC complicating a PE.

Leitner et al., [1] reported 100% hospital mortality in patients with massive PE complicated by DIC and

cardiopulmonary arrest. There are no well-defined guidelines for the management of patients with simultaneous massive PE and DIC. Massive PE leading to cardiopulmonary collapse becomes an indication for thrombolytic therapy, but the institution of IV heparin in a controlled environment leads to dramatic improvement and has been shown to save lives. We need further studies to explain the exact mechanisms leading to DIC in these patients and to guide appropriate therapy.

Classification (Symptom)			Treatment for Underlying Disease	Anticoagulation Therapy					Antifibrinolytic Therapy	Fibrinolytic Therapy	Blood Transfusion	
				UFH	LMWH	DS	SPI	AT			FFP	Platelet Concentrates
General			O	C	B_2	C	B_2	B_1[b]	D	D	O[c]	O[c]
Asymptomatic	Blood Transfusion	Not Necessary	O	C	B_2	C	B_2	B_2[b]	D	D		
		Necessary	O	C	B_2	C	B_2	B_2[b]	D	D	B_2[c]	B_2[c]
Bleeding	Minor		O	C	B_2	C	B_2	B_2[b]	D	D		
	Severe		O	D	D	D	B_1	B_2[b]	C[e]	D	O[c]	O[c]
Organ Failure			O	C	B_2	C	B_2	B_1[b]	D	D[d]		
Complication	Major Thrombosis		O	B_2	B_1	B_2	C	B_2[b]	D			
	TTP		O	C	B_2	C	B_2	B_2[b]	D	D	O	D
	HIT		O	D	D	D	B_2	B_2[b]	D	D		D
Recommendation Levels (Modified Kish Guide)[g]												
Recommendation												
Consensus				Treatment does not have a high quality of evidence, but it should be carried out as common sense								
A				Treatment has high quality of evidence, and the clinical usefulness is clear								
B_1				Treatment has moderately high quality of evidence, or it has high quality of evidence but the clinical usefulness is not significant								
B_2				Treatment does not have a high quality of evidence, but it has few deleterious effects and it is carried out clinically								
C				Treatment does not have a high quality of evidence or the clinical usefulness is not clear								
D				Treatment has high quality of evidence, and it has deleterious effects								

Table 1: Recommendations for the treatment of DIC.

AT: Antithrombin; DIC: Disseminated Intravascular Coagulation; DS: Danaparoid Sodium; FFP: Fresh-Frozen Plasma; HIT: Heparin-induced Thrombocytopenia; LMWH: Low Molecular Weight Heparin; SPI: Serine Protease Inhibitor; TTP: Thrombotic Thrombocytopenic Purpura; UFH: Unfractionated Heparin

[a]O: Denotes Consensus.

[b]Limited in patients with less than 70% of AT.

[c]According to the guideline for blood transfusion.

[d]Consultation with specialist for fibrinolytic therapy.

[f]Consultation with specialist for antifibrinolytic therapy.

[g]Recommendation levels determined according to "the guidelines of DIC treatment preparation committee" according to the evidence level and medical care of Japan. Physician should decide on adequate treatment, not only according to the guidelines but also according to the condition of each patient and institute.

The fact that severe DIC can cause PE has been discussed previously: An autopsy study by Katsumura et al. to examine the incidence of thromboembolism in DICs, compared 87 patients whose disease was complicated by DIC and 64 patients who had no blood clotting abnormalities in their terminal disease. While in the control group, macroscopic thromboembolism was identified in 20 patients (31.3%), it was found in 51 (58.6%) of CIVD 4 patients.

Disseminated intravascular coagulation is therefore classified as follows: Asymptomatic type (no clinical symptoms of DIC but biology confirms DIC), that is, a type of marked bleeding and organ failure. Appropriate treatment differs and is based on the type of DIC. These treatments are summarized in Table 1 [5].

In patients with overt DIC, platelet transfusion is recommended to maintain a platelet count of at least 50,000/L for patients with active bleeding or 20,000/L for patients without signs of bleeding. For patients with acute deep vein thrombosis or pulmonary embolism with overt CID and bleeding, the use of an inferior vena cava filter has been recommended, with anticoagulant therapy started after the bleeding has stopped [5-7].

4. CONCLUSION

The rationale for treatment of DIC is based on the pathogenic mechanism and the phase of the syndrome but currently lacks evidence from clinical trials. Although data from phase III clinical trials on the treatment of DIC are not available, the subgroup analysis of studies presented in a review 6 proposes a rational therapeutic strategy for DIC according to these principles:

- ✓ Hypercoagulability treatment.
- ✓ By the administration of rapid-acting antithrombotics mainly LMWH or UFH when renal function is compromised.
- ✓ And by the administration of concentrates of natural coagulation inhibitors (AT, rhAPC, rTFPI and rTM) in order to restore the effectiveness of the natural pathways of anticoagulants.
- ✓ Restoration of coagulation factor deficiencies when plasma levels are very low, and the risk of bleeding is high or active bleeding is present [5].

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