Cardiology

KEYWORDS: direct thrombin inhibition; embolism; thrombosis

CO-OCCURRENCE OF PULMONARY ARTERIAL EMBOLISM AND CORONARY THROMBOSIS IN A PATIENT ON CHRONIC ANTICOAGULANT TREATMENT WITH DIRECT THROMBIN INHIBITION

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ABSTRACT:

We present the case of an 86-year-old man with permanent atrial fibrillation on chronic treatment with a direct oral thrombin inhibitor (dabigatran 110 mg bid) with evidence of segmental pulmonary embolism on thoracic CT, immediately followed by ST segment elevation myocardial infarction. Blood analysis revealed reduced renal function and high plasmatic level of dabigatran.

Case Report

An 86-year-old man with hypertension and permanent atrial fibrillation (AF) on direct oral anticoagulation treatment (dabigatran 110 mg bid) was admitted to our emergency care unit suffering from dizziness, sweating, dyspnoea ad oppressive chest pain. Systemic blood pressure was 110/90 mmHg, heart rate (HR) 40 bpm arrhythmic. The electrocardiogram showed AF with slow ventricular response and no evidence of ischemic alterations. Hypoxia and hypocapnia were both present at arterial blood gas analysis. Transthoracic echocardiography revealed mild dilation of the right ventricle and normal left ventricular segmental and global kinesis. Biochemical blood analysis showed normal cross-linked fibrin degradation product (XDP), serum creatinine of 2,51 mg/dl (estimated glomerular filtration rate of 23 ml/min, according to Cockroft-Gault formula), troponin T 35,4 ng/L (reference limit < 14 ng/L), plasmatic level of dabigatran 235 ng/ml (upper reference level for our laboratory 200 ng/mL). We decided to perform a thoracic CT with contrast dye that resulted positive for segmental pulmonary embolism (Panel A, B; red arrows). Then, unfractionated heparin was immediately started. During the observation time in the emergency care unit, patient reported abrupt worsening of symptoms with new electrocardiographic finding of inferior ST-elevation myocardial infarction (STEMI) (Panel C), that was immediately treated by primary percutaneous angioplasty and drug eluting stent implantation of the proximal tract of right coronary artery (Panel D).

The co-occurrence of pulmonary arterial embolism and coronary thrombosis in a patient on chronic anticoagulant treatment is a rare clinical condition. In this specific case, the patient was assuming a direct thrombin inhibitor (DTI), dabigatran 110 mg bid, according to his glomerular filtration rate at the time of prescription. Before dabigatran therapy, the patient had been on warfarin for seven years without embolic and/or thrombotic complications. The switch from vitamin K antagonist to DTI had been pursued according to patient wish to accomplish a simpler therapeutic scheme. He had no family history suggesting a higher thrombotic risk. Moreover, patient's compliance to therapies was referred optimal as it was evident by the blood analysis assessing plasmatic level of dabigatran that resulted higher than the upper reference level, in this case also due to an acute worsening of glomerular filtration rate. Direct oral anticoagulants (DOACs) have been shown to be not inferior to vitamin K antagonists in reducing thrombo-embolic events in patients with non-valvular AF and venous thrombo-embolism1. However, thrombin presents crucial anti-coagulant and pro-fibrinolytic activities, which play an essential role in haemostasis control2. This could represent the "back side of the coin" of iatrogenic thrombin inhibition3. There is a rationale for presuming that direct thrombin inhibition could not only prevent thrombosis in certain predisposing conditions - such as atrial fibrillation - but also favour thrombosis in other predisposing venues - such as arterial unstable plaques. Considering the pleiotropic functions of thrombin in the haemostasis cascade and regulation, it is possible that direct thrombin inhibition may represent an unsafe target for pharmacological antagonism3. Conversely, oral DOACs acting at an earlier step along the coagulation cascade, by not acting directly on thrombin but inhibiting activated factor X, leave "untouched" the most relevant thrombin-dependent anti-coagulant mechanism. For this reason, Xa factor inhibitors could have a safer profile of action in the management of patients with atrial fibrillation and high athero-thrombotic profile.

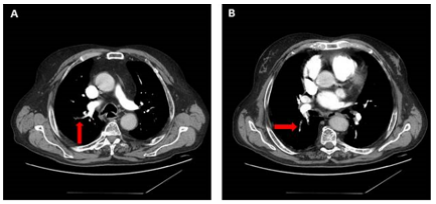
CONFLICT OF INTEREST DECLARATION: The authors declare that there is no conflict of interest regarding the publication of this paper.

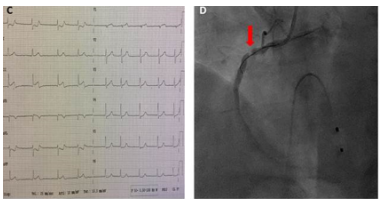
Figures legend.

Panel A and B: thoracic CT showing segmental pulmonary artery embolism (red arrows).

Panel C: electrocardiographic evidence of inferior ST-elevation myocardial infarction

Panel D: Guidewire angioplasty crossing the thrombotic lesion at the proximal tract of right coronary artery.





REFERENCES

1. Camm AJ, Lip GY, De Caterina R, Savelieva I, Atar D, Hohnloser SH, et al. 2012 focused update of the ESC Guidelines for the management of atrial fibrillation. Eur Heart J. 2012; 33: 2719-47.
2. Di Cera E. Thrombin as procoagulant and anticoagulant. J Thromb Haemost. 2007; 5 (Suppl. 1): 196-202.
3. Fragasso G, Corti A, Loiacono F, Margonato A, D'Angelo A. Oral direct thrombin inhibition: a double-edged sword?. Heart Lung Vessel. 2015;7(3):191-7. Review.