Case Report

B-type Natriuretic Peptide May be Unsuitable for Diagnosing Central Acute Pulmonary Embolism

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Abstract

We describe a case of a 90-year-old male admitted to the emergency department with deep vein thrombosis and central acute pulmonary embolism. Despite a remarkably increased value of D-dimer and a modestly elevated concentration of cardiac troponin I, the value of B-type natriuretic peptide was found to be non-diagnostic. Limited to this single case report, our evidence suggests that the measurement of natriuretic peptides is questionable for diagnosing central acute pulmonary embolism in the emergency department. **[Indian J Chest Dis Allied Sci 2014;56:253-254]**

Key words: B-type natriuretic peptide; Pulmonary embolism; Venous thromboembolism.

Introduction

The measurement of natriuretic peptides, including B-type natriuretic peptide (BNP), pro-BNP and N-terminal pro-BNP (NT-Pro-BNP), is currently considered a cornerstone for supporting the diagnosis and prognostication of patients with heart failure.[1] Recent evidence has also been provided that in patients with pulmonary embolism (PE) the levels of NT-Pro-BNP are nearly four-fold higher in the presence of central thrombi than in patients with peripheral thrombi.[2] The practical usefulness of these biomarkers has, however, been recently disputed by Panigrahi,[3] who argued that natriuretic peptides may not be specific markers of PE, and it may also be unjustified to spend up to 20 crucial minutes for these measurements in a critically ill patient. In partial support of this conclusion, we describe the case of a patient admitted to the emergency department (ED) with deep vein thrombosis (DVT) associated with central acute PE and non-diagnostic values of BNP.

Case Report

A 90-year-old male patient was referred to the local ED for worsening dyspnoea, with onset approximately 40 days before admission. Clinical examination revealed dyspnoea at rest, heart rate 95 beats per minute, respiratory rate 32 per minute, mild hypertension (150/90 mmHg) and hypoxaemia [arterial oxygen tension (PaO_2)=59 mmHg). He had experienced a previous episode of DVT associated with PE, which had occurred about six years ago for which he had been treated with anticoagulant therapy (i.e., warfarin) for six months.

Upon admission to the ED, the Revised Geneva Score (RGS)[4] of the patient was 13, indicating a high pre-test probability of PE. Thus, biochemical tests were done along with contrast-enhanced computed tomography (CT) of the chest. The test results (Table), showed a remarkably elevated D-dimer levels, along with a mild elevation of cardiac troponin I (cTnI) and a non-diagnostic BNP. All the other parameters were within the respective reference range.

Table. Laboratory data of the 90-year-old male patient admitted to the emergency department with deep vein thrombosis and central acute pulmonary embolism

Laboratory Parameter	Value	Reference Range
White blood cell count ($x10^9$)	9.4	4.0–10.0
Red blood cell count ($x10^{12}$)	5.16	4.30–5.90
Platelet count ($x10^9$)	232	150–400
Prothrombin time (sec)	9.1	8.2–10.6
Activated partial thromboplastin time (sec)	27.7	26.0–30.2
Fibrinogen (g/L)	1.8	1.5–4.0
D-dimer (ng/mL)	1551*	<245
Serum creatinine (mg/dL)	0.8	0.5–1.4
B-type natriuretic peptide (pg/mL)	80	<100
Cardiac Troponin I (ng/mL)	0.06*	<0.04

* *Abnormal values*

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Chest CT was consistent with central acute PE. More specifically, a "saddle embolus" was detected at the bifurcation of the left pulmonary artery along with substantial thrombus burden in lobar branches of right pulmonary artery (Figure) and, partially, also in lobar branches of left pulmonary artery. The major diameter of thrombus in the pulmonary artery was 33 mm (six years earlier, when previous episode of PE occurred, the diameter was 29 mm). Filling defects were also clearly visible downward the obstruction.

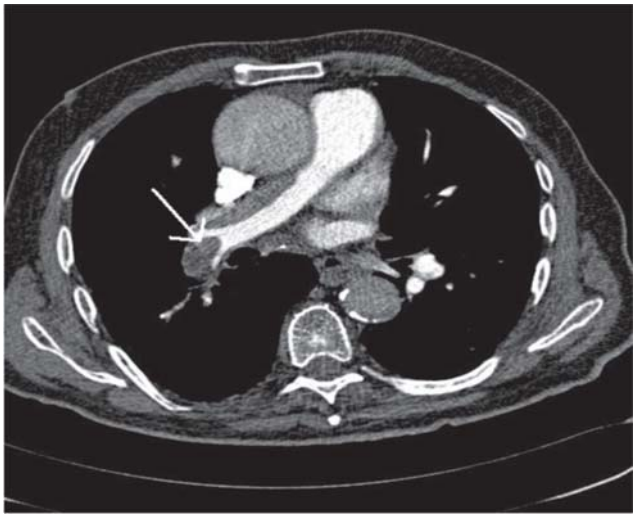

Figure. Computerised enhanced computed tomography chest showing saddle embolus at the bifurcation of the left pulmonary artery (arrow).

A venous doppler study of the legs revealed the presence of acute DVT in the left leg, extending from the popliteal to the common iliac vein. Thrombolytic treatment was not administered, and the patient was treated with standard anticoagulant therapy with oral anticoagulation (i.e., warfarin) associated with low molecular weight heparin until the international normalised ratio (INR) was greater than 2. The patient was discharged 7 days after the acute episode of venour thromboembolism, for home management with anticoagulant therapy, as per our hospital protocol.

Discussion

The assessment of natriuretic peptides is common for differential diagnosis of dyspnoea and shortness-of-breath in the ED.[5] In patients with PE, substantially increased values of BNP are significantly associated with all-cause mortality, PE-related mortality and serious adverse events.[6] Recent evidence also suggests that higher pro-BNP levels may indicate a greater probability of central thrombus location in PE.[2] Nevertheless, the present case report seems to contradict this notion. In our patient, despite the presence of acute central PE, the concentration of BNP was non-diagnostic upon presentation, and remained so for the remaining days of hospital stay (range between 80 and 91 pg/mL). This is noteworthy, considering that the concentration of BNP typically increases with ageing and a diagnostic cut-off greater than 100pg/mL would have probably been more appropriate in this 90-year-old patient.[7] Therefore, the evidence limited to this single case report is in support of the recent conclusion of Panigrahi,[3] who suggested that increased levels of natriuretic peptides may be indicative, but are not adequately sensitive nor specific for effective diagnosis of centrally located thrombi in the ED. Therefore, CT angiography should always be performed whatever the value of natriuretic peptides upon patient admission to the ED.

Despite the remarkable increased value of D-dimer, which was rather predictable considering the severity of thrombosis,[8] we also observed a value of cTnI that modestly exceeded the diagnostic threshold of the assay. This finding is compatible with the presence of a mild myocardial injury due to PE since all other potential causes of troponin elevation were excluded (i.e., atrial fibrillation, renal impairment, myocarditis).[9] This is not really surprising, since an increase of cardio-specific troponins may often occur after an episode of severe PE, as a result of myocardial ischaemia and micro-infarction due to alterations in oxygen supply and demands of the right ventricle.[10] Hence, it is noteworthy that the diagnostic value of cTnI was even greater than that of BNP in this case of central acute PE.

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