Usefulness of point-of-care ultrasound in the atypical presentation of aortic dissection: a case series and brief review of literature

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Abstract

Acute aortic dissection represents a surgical emergency that, in most cases, may present an atypical and non-specific clinic, making the diagnosis difficult and late. Computed tomography angiography represents the gold standard for diagnosis, but different ultrasound techniques are able to provide valuable additional information related to diagnosis, and optimize therapy and prognostic assessment.

In this case series, we describe different atypical patterns of aortic dissection, associated to non-diagnostic D-Dimer levels. These atypical conditions escape recognized diagnostic algorithms for the diagnosis of aortic dissection, although the incidence of such events is far from negligible. A clinical approach in the emergency setting that provides for a systematic and standardized use of bedside ultrasound could help reduce the incidence of errors and diagnostic delay, addressing the gold standard instrumental diagnostics for the reference pathology.

Introduction

Acute aortic dissection represents a surgical emergency that can be easily suspected and quickly diagnosed in the case of a typical presentation.\(^1\) In particular, lacerating and acute chest pain, anteriorly, migrating, with posterior irradiation, associated with asymmetry of the peripheral arterial wrists, represent rather suggestive findings. However, in most cases, the presentation of this serious condition is very subtle, and the symptoms are rather unspecific, so as to make the diagnosis difficult and sometimes late.\(^2,3\) In confirmation of this, a high incidence of missed or incorrect diagnoses has been reported in the literature which weigh on the correct and timely management of acute aortic syndromes.\(^4,5\) Current imaging techniques, of which computed tomography angiography (CTA) represents the gold standard, can recognize or
exclude alterations of the aortic wall with great efficacy; however the patient needs adequate selection. 

In the ADvISED Prospective Multicenter Study, a multicenter prospective observational study involving 6 hospitals in 4 countries from 2014 to 2016, the Authors assessed the diagnostic accuracy obtained by adding the D-Dimer dosage to the aortic dissection detection (ADD) risk score (ADD-RS), suggesting a low failure rate for both the ADD-RS = 0/ DD− and ADD-RS ≤1/ DD− strategies. 

Different ultrasound techniques, with particular reference to transthoracic echocardiography and vascular ultrasound, but also transesophageal echocardiography, intravascular ultrasound and contrast-enhanced ultrasonography, are able to provide valuable additional information related to diagnosis, and optimize therapy and prognostic evaluation. In the emergency department setting, the bedside ultrasound assessment is able to provide a rapid clinical and diagnostic framework, particularly in the critically ill patient.

**Aim**

In this case series, we describe different atypical patterns of aortic dissection, associated to non-diagnostic D-Dimer levels, and the role of bedside thoracic /abdominal ultrasound in suggesting this suspicion and addressing the diagnosis (Table 1).

**Case Reports**

The first patient, A, was an 81-year-old male patient who entered the emergency room for the onset of palpitations for about 4 days, apparently not associated with angina or dyspnea, and without hemodynamic deterioration at the presentation.

Regarding his clinical history, the patient reported an essential hypertension in adequate pharmacological control with lacidipine. No other significant morbid conditions were found.
At the emergency room visit, the patient was alert and cooperative. On the electrocardiogram we found a sinus tachycardia with an average rate of 130 bpm, and no signs of myocardial ischemia. Arterial pressure and arterial saturation in ambient air were within normal values. Hematric tests showed only a slight increase in highly sensitive troponin, and values at the upper limits of the norm for pro-BNP; blood gas analysis showed a normocapnic respiratory failure, requiring low flow oxygen therapy, and a mild hyperlactacidemia.

Chest radiography showed a mild bilateral pleural effusion with increased representation of the interstitium. Despite the administration of oxygen therapy and intravenous diuretic therapy, the heart rate remained high, and hyperlactacidemia did not decrease. We performed a bed-side thoracic ultrasound evaluation with finding some B lines at the bases, bilaterally, a slight pleural effusion, and, above all, a not hemodynamically significant pericardial effusion, without kinetic disturbances of cardiac walls. We extended evaluation to abdominal aorta, discovering a diffuse atherosclerotic involvement and aspecifica haemodynamic alterations on pulse wave velocity assessment. After the specialist cardiological evaluation, for the suspicion of an alteration of the ascending aorta including the valve, the patient was subjected to CT angiography of the thoracic aorta. The instrumental examination showed a type A dissection, involving aortic valve, with indication for cardiac surgery correction. No involvement of carotid and subclavian arteries was found. On blood tests, D Dimer values were slightly altered, but not significantly if corrected for the patient's age.

The second patient, B, was a young man of 40 years, with no significant pathologies in history, who entered the DEA for pain in the right hypochondrium, suspected of biliary colic and persistence of high blood pressure, resistant to therapy with calcium antagonists. At the blood tests we found an isolated elevation of creatine phosphokinase, with a slight D-Dimer level elevation. There was no hyperlactacidemia or troponin alteration. Electrocardiogram was normal, with heart rate at the upper limits. At the bedside ultrasound evaluation of the
abdominal aorta, we found a demodulation of the hemodynamic flow, with alteration of the vascular wall. Subsequent CT angiography of the aorta revealed a type B dissection (type III b DeBakey classification) involving the distal aorta and the right external iliac.

The third patient, C, was a young man with sudden isolated left flank pain associated with urinary retention. At the clinical evaluation we found pain on percussion of the left side; arterial pulses were symmetrical and normosphigmic. In the blood tests we had found marked neutrophilic leukocytosis, elevation of the C reactive protein and creatine phosphokinase; elevated lactacidemia was found on blood gas analysis. The patient underwent bedside ultrasound in suspicion of left renal colic; however, the ultrasound evaluation ruled out a dilation of the renal pelvis, showing a hyperechogenicity of the renal cortex with almost no arterial flow detectable in the left renal artery. Moreover, the hemodynamic arterial profile was asymmetric at the level of the external iliacs, with a reduced systolic peak on the left side, affected by the suspected dissection.

The patient underwent CT angiography with dissection of the abdominal aorta in the prerenal tract, complete exclusion of the left renal artery and ongoing renal infarction.

**Discussion**

Aortic dissection represents a serious pathology whose treatment and prognosis depends both on the type of vascular lesion, as well as on the precocity of the diagnosis. In particular, the mortality rate secondary to thoracic aortic dissection is high and above all time-dependent, increasing from 1% to 1.4% for each hour of delay in treatment, and in any case up to 68% in the first 48 hours.

The most frequent clinical presentation consists of chest pain - pulses. However, dissection can occur anywhere in the aorta, making clinical manifestation potentially unpredictable. Indeed, cases of atypical presentation are frequently described and associated with variable
outcomes (Table 2). Acute painless aortic dissection has been described in 5% of patients and may occur in approximately 5% of patients; in about 8% of cases it was reported as painless and associated with syncope. Acute paraplegia has been reported rarely in 4.2% of 1805 patients with aortic dissection. Numerous other clinical reports describe a myriad of clinical presentations, potentially making the physician's job in diagnosing the disorder very difficult. The importance of the rapid and certain diagnosis of aortic dissection is even more true, especially when considering the differential diagnosis with acute coronary syndromes, whose therapy is potentially harmful. Literature data support the use of computed tomography for diagnosis in cases showing an indicative or suspicious clinical presentation (Table 2) escape the usual diagnostic algorithms, and the suspicion is formulated only after multiple imaging studies are performed, with considerable delay.

For this reason, the clinician in the emergency department must keep the suspicion of possible dissection of the aorta high, and must have access to a versatile and quick-to-use diagnostic method that allows a multisystem approach to the patient. In this regard, the role of point of care ultrasound as an ally of the emergency physician is well known. In this case report, the three patients showed palpitations, pain typical of biliary colic, and renal colic, respectively. A systematic approach for the management of the thorax and abdomen with bedside ultrasound method allowed the diagnosis to be delayed to a minimum.

Conclusions

Dissection of the aorta represents a hidden possibility in the shadow of multiple, apparently non-specific clinical pictures. These conditions escape recognized diagnostic algorithms for the diagnosis of aortic dissection, although the incidence of such events is far from negligible. A clinical approach in the emergency setting that provides for a systematic and standardized use of bedside ultrasound could help reduce the incidence of errors and diagnostic delay,
addressing the gold standard instrumental diagnostics for the reference pathology.

References


Table 1. Clinical characteristics and ultrasound findings of patients.

<table>
<thead>
<tr>
<th></th>
<th>Patient A</th>
<th>Patient B</th>
<th>Patient C</th>
</tr>
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<tbody>
<tr>
<td>Age, y.o.</td>
<td>74</td>
<td>40</td>
<td>36</td>
</tr>
<tr>
<td>Sex</td>
<td>Male</td>
<td>Male</td>
<td>Male</td>
</tr>
<tr>
<td>Initial symptoms</td>
<td>Palpitations</td>
<td>Right hypochondrium pain</td>
<td>Left flank pain</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>120</td>
<td>160</td>
<td>150</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>80</td>
<td>100</td>
<td>90</td>
</tr>
<tr>
<td>Frequency rate, bpm</td>
<td>130, sinus tachycardia</td>
<td>100, sinus tachycardia</td>
<td>90, sinus tachycardia</td>
</tr>
<tr>
<td>D Dimer, mcg/L</td>
<td>750</td>
<td>470</td>
<td>510</td>
</tr>
<tr>
<td>CPK, U/L</td>
<td>260</td>
<td>180</td>
<td>630</td>
</tr>
<tr>
<td>Lactate levels</td>
<td>1.7</td>
<td>0.7</td>
<td>2.9</td>
</tr>
<tr>
<td>TT US</td>
<td>Pericardial effusion, basal B lines, mild pleural effusion</td>
<td>No relevant findings</td>
<td>No relevant findings</td>
</tr>
<tr>
<td>TA US</td>
<td>Atheromatous abdominal aorta, aspecific halmodinamic alterations on pulse wave</td>
<td>Demodulation of the hemodynamic flow, associated to alteration of the vascular wall, on pre-renal aorta</td>
<td>No flow detectable on left renal artery, asymmetrical flow on iliac arteries</td>
</tr>
<tr>
<td>Type of lesion</td>
<td>Stanford A, DeBakey II</td>
<td>Stanford B, DeBakey IIIb</td>
<td>Stanford B, DeBakey IIIa</td>
</tr>
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SBP, systolic blood pressure; DBP, diastolic blood pressure; bpm, beat per minute; TT US, trans-thoracic ultrasound; TA US, trans-abdominal ultrasound.
Table 2. Atypical presentation of aortic dissection.

<table>
<thead>
<tr>
<th>Atypical presentation</th>
<th>Author</th>
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<tbody>
<tr>
<td>Dull pain deep to the suprasternal notch</td>
<td>Saunders T and Suzuki T, 2018(^{14})</td>
</tr>
<tr>
<td>Pain mimicking appendicitis</td>
<td>Azharuddin M \textit{et al.}, 2018(^{25})</td>
</tr>
<tr>
<td>Fever</td>
<td>Yuan, 2017(^{26})</td>
</tr>
<tr>
<td>Painless</td>
<td>Siddiqui \textit{et al.} 2018(^{27}), Ayric \textit{et al.} 2006(^{28}), Young \textit{et al.}, 2002(^{16}), Rosen \textit{et al.}, 1988(^{17}), Zull \textit{et al.}, 1988(^{18})</td>
</tr>
<tr>
<td>Neurological presentation at the onset</td>
<td>Bekele \textit{et al.} 2017(^{29}), Rosen \textit{et al.} 1988(^{17}), Zull \textit{et al.} 1988(^{18})</td>
</tr>
<tr>
<td>Pleuritic chest pain</td>
<td>Mohamed-Yassin \textit{et al.} 2019(^{30})</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Xiuhua \textit{et al.} 2016(^{31})</td>
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