

At the “threshold” of certainty: Risk stratification and clinical assessment incongruity in the diagnosis of aortic dissection

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Maxim Zlatopolsky, Daniel Brancheau  and Marcel Zughuib

Abstract

The use of D-dimer assay to screen for aortic dissection has become routine in clinical practice. Although an uncommon disease entity, aortic dissection is rapidly fatal without prompt diagnosis, and biochemical detection in the proper clinical context is crucial. We performed a retrospective chart review of all aortic dissections presenting to a community tertiary care center between 2000 and 2016 and identified three patients in whom laboratory D-dimer testing was positive and a diagnosis of Stanford Type A aortic dissection was confirmed with computed tomography angiography based on clinical assessment and a newly validated risk stratification tool.

Keywords

Cardiovascular, D-dimer, aortic dissection, Stanford A, risk stratification, acute aortic syndrome, chronic aortic dissection

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Patients presenting to the emergency department (ED) with aortic dissection (AD) require prompt evaluation, diagnosis, and treatment. Despite multiple advances in these areas, there is no definitive biochemical assay to establish a diagnosis.¹

AD is the most common acute aortic syndrome, with an exceptionally high mortality rate—approaching 50% within the first 48 h if left untreated.² It also remains a rare clinical diagnosis with a highly variable clinical presentation.^{3,4} This necessitates accurate clinical assessment in conjunction with risk stratification and D-dimer assay.

The D-dimer assay detects products of complete fibrinolysis and has been used as a highly sensitive test for pulmonary embolism (PE). Elevated levels may be seen in a variety of clinical settings, including malignancy, immobility, connective tissue disease, and end-stage renal disease.⁵ Most importantly, D-dimer is not sufficient as a stand-alone screening tool.

D-dimer assays have previously been utilized in the assessment of patients with AD.⁶ In regard to acute AD, there is no definitive cut-off to “rule-in” the diagnosis. There have been several studies measuring the utility of D-dimer in diagnosing AD.^{7,8} This is reflected in current European Society of Cardiology (ESC) guidelines on the management of acute aortic syndromes, with a class IIa recommendation to use D-dimer to rule out AD in cases with low clinical suspicion.

Recently, risk stratification has emerged as a validated tool in the initial assessment of patients with suspected AD. Nazerian et al. in the Diagnostic Accuracy of the Aortic Dissection Detection Risk Score Plus D-Dimer for Acute Aortic Syndromes (ADvISED) study created a validated risk calculator with a minimal failure rate. The ADvISED investigators used the previously outlined Acute Aortic Dissection–Risk Calculator (AAD-RS) by Rogers et al. and supplemented the sensitive D-Dimer biomarker to form a diagnostic algorithm which will only miss 1 in 300 cases of AD.^{8,9}

We present three patient case series in which application of the aforementioned risk calculator yielded a conflicting result. All three patients were subsequently diagnosed with Stanford AAD using computed tomography (CT) angiography.

Case section

Case 1: A 61-year-old female with a history of hypertension presented to the ED with pain in her chest and right leg that

Providence Hospital Medical Center, Michigan State University, Southfield, MI, USA

Corresponding Author:

Maxim Zlatopolsky, 22512 Marter Rd. St. Clair Shores, MI 48080, USA.
Email: zlato87@gmail.com



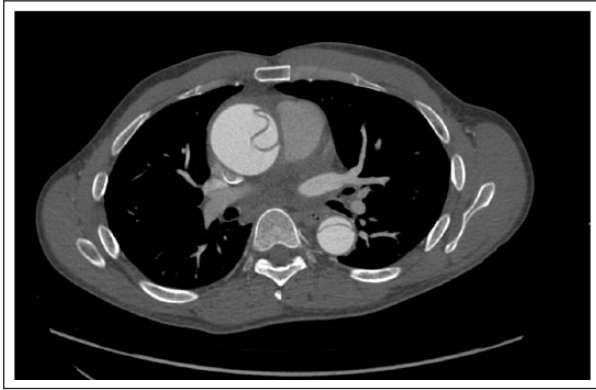


Figure 1. Axial CT of dissection flap.



Figure 2. Sagittal view of dissection propagating from aortic root toward renal arteries.

began approximately 30 min prior to arrival. A D-dimer was immediately performed and revealed a value >5000 ng/mL. This patient's AAD-RS=2 (high risk) was based on abrupt onset of sharp chest pain, hypotension, and D-dimer >500 ng/mL. The patient soon became hypotensive and a CT scan confirmed the diagnosis of a Stanford Type A dissection extending to the right iliac artery (Figure 1). The patient was taken emergently to the operating room where repair of the dissection was performed. The patient had an extended hospital stay but was discharged on postoperative day 10.

Case 2: A 57-year-old African American male with a history of uncontrolled hypertension and recent transient ischemic attack on clopidogrel presented to the ED following the acute onset of non-tearing chest pain several hours prior to arrival. A D-dimer was performed and revealed a value of 281 ng/mL. His AAD-RS=1 (low risk) was based on acute onset of sharp chest pain with radiation to the back, normal blood pressure, lack of perfusion deficit, and a D-dimer <500 ng/mL. CT angiography revealed a Stanford A



Figure 3. Coronal view of dissection flap.

AD with diffuse extension proximal to the origin of the mesenteric arteries (Figure 2). The patient was taken emergently to the operating room for repair of his dissection. Unfortunately, the patient developed hypotension secondary to perfusion deficits and expired shortly thereafter. After further discussion with family members, it was determined that the patient's onset of symptoms was actually 1 week prior to initial medical contact. This would explain the aberrantly low D-dimer value, and it still falls within the diagnostic definition of acute AD.

Case 3: A 63-year-old African American male with a history of hypertension and a 30 pack-year smoking history presented to the emergency room with intermittent sharp chest pain with extension to his right arm. An initial D-dimer level of 1024 ng/mL was preceded by CT angiography which revealed a Stanford A dissection (Figure 3). His AAD-RS=2 (high risk) was based on acute onset of sharp chest pain, pulse deficit, and D-dimer >500 ng/mL. He underwent repair of the dissection and improved postoperatively. He was discharged on postoperative day 9 without further complications.

Discussion

In cases 1 and 3, clinical presentation, positive results of D-dimer, and overall clinical trajectory are consistent with AD. The ADvISED investigators found that 66.9% of their study participants with AAD-RS scores ≥ 1 and D-dimer level ≥ 500 ng/mL were diagnosed with AD. The presence of attention-deficit disorder (ADD)-RS score ≥ 1 carried a sensitivity of 95% and a specificity of 26.4%, while a D-dimer of ≥ 500 ng/mL had a sensitivity of 96.7% and a specificity of 64%. Both cases had ADD-RS score ≥ 1 and a positive D-dimer and were appropriately diagnosed with acute AD.

However, case 2 outlines a more atypical presentation of acute AD with AAD-RS score ≤ 1 and negative D-dimer (≤ 500 ng/mL). The integration of the ADD-RS score ≤ 1 with a negative D-dimer was found to miss only three cases out of

924 patients with a failure rate of 0.3% by the ADvISED investigators.¹⁰ This case stresses the importance of the underlying clinical examination as well as thoughtful history-taking when assessing patients with suspected acute aortic syndromes.

In the most recent 2014 ESC Guidelines on the management of acute aortic syndromes, the most common explanation offered for low D-dimer values is that intramural hematoma (IMH) or penetrating aortic ulcers (PAU) may be present, or symptom onset was greater than 24 h prior to initial medical contact. Further studies are needed to evaluate the appropriate D-dimer level based on timing to symptom onset. This remains a difficult objective due to the low incidence of AD, the variability in clinical manifestations of this disease, and a lack of time-course studies evaluating D-dimer levels after symptom onset.

Conclusion

Our case series provides an example of the successes and failures of the most recent risk stratification tool available for the screening of AD. Although the failure rate is low, misdiagnosis remains a possibility. A better understanding of the precise temporal decrement in D-dimer values would allow for better clinical recognition and management of patients with subacute presentations or with possible alternative diagnoses. One of the limitations of our study is the low number of cases presented in this series. Irrespective, we feel that the diagnosis of such a rare and potentially fatal disease entity can still be identified with correct clinical evaluation when there appears to be a contradictory assessment on initial risk stratification.

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Declaration of conflicting interests

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Ethics approval

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Informed consent

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ORCID iD

Daniel Brancheau  <https://orcid.org/0000-0003-2048-0358>

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