INTRODUCTION

Acute aortic dissection (AAD) is the most common catastrophic event affecting the aorta, with an estimated annual incidence approximately 5-30 per million. Ascending AAD occurs most commonly between 50 and 60 years of age. In the 2010 guidelines, the American Heart Association and the American College of Cardiology introduced the aortic dissection detection risk score (ADDRS) as a simple and systematic bedside tool guiding the diagnostic approach to suspected AAD that was revised in 2017 including the D-dimer assay.

Hereby, we present a 64-year-old patient with a typical atrial fibrillation (AF)—a condition actually nonincluded into the ADDRS—masking an AAD Stanford type-A extended into the right coronary artery (RCA) originating from the aortic false lumen, axillary artery aneurysm, and thrombosis. We finally present our considerations on the fact that supraventricular arrhythmias may be secondary to a perfusion deficit of the sinoatrial node artery.

CASE REPORT

The patient was admitted to our emergency department with right arm weakness and pain started 2 hours before admission. The ECG showed AF with rapid ventricular response and mild ST-segment depression in lower-lateral leads that
could also be interpreted as an underlying overload and/or ischemia (Figure 1). The patient was currently followed at our hospital for essential arterial hypertension with a satisfactory control of blood pressure by lacidipine; from the clinical records, previous supraventricular arrhythmias were excluded.

On the examination, the right arm was cold and radial and humeral pulses were absent. Color-Doppler echocardiography of the right arm showed aneurysmal dilatation of the right axillary artery. Computed tomography (CT) scan demonstrated an AAD Stanford type-A dissection of the ascending aorta with a suspect intimo-intimal intussusception (Figure 2) and the patient was promptly transferred to the cardio-thoracic unit.

3 | DISCUSSION

As far as we know, this is the second case reported in the literature of AAD Stanford type-A presenting with AF,4 and the presence of this arrhythmia was not helpful, on the contrary, it has contributed to confusing, at least, the initial clinical picture, although the peculiar symptomatology has addressed correctly to the execution of a contrast-enhanced CT that was consistent for the final diagnosis. Contrast-enhanced CT scanning,5 magnetic resonance imaging, and Transesophageal echocardiography are accurate techniques that are useful. In our case, the irregular appearance of the intimal flap at axial contrast-enhanced CT was firstly misinterpreted as an intimo-intimal intussusception; however, the absence of circumferential dissection and the so-called toe sign helped us in the differential diagnosis from a common intimal flap on volume rendering images as shown in the figure panels. Furthermore, AAD is characterized by a spectrum of clinical presentations that can be misleading; data from the International Registry of Acute Aortic Dissection1 support that, among the electrocardiographic abnormalities, nonspecific ST segment change is the most common, with an incidence of acute myocardial infarction of 4.8%, while AF is not mentioned. In our case, AF is possibly an epiphenomenon of the acute occlusion of the artery that supplies the sinus node (SN), an event to be fully featured within the AF etiological factors. An ischemic damage of the SN, that in a large percentage of subjects is vascularized by the SN artery originated from the RC, is a well-known cause of cardiac arrhythmias.6 Indeed, not infrequently, the ischemic hypothesis7 of AF usually is not taken into great consideration, even if it could be an epiphenomenon appearing during acute hypoxia, pneumonia, pulmonary embolism.6,7 Finally, it should be said that even the acute thrombosis of the SN artery has been implied into sinus node dysfunction, in the context of coronary artery disease.8
CONCLUSIONS

Aside the peculiar symptomatology, that in our case contributed, in a crucial way, to direct toward the solution of the clinical puzzle, it should be remembered that, even supraventricular AR, can sometimes be “only” epiphenomena of hypoxia. Indeed, AF is not rare in AAD,7 we suppose that the reduction of the oxygen supply to the sinoatrial nodal artery, that in 80% of the people origin from the right coronary,6 could induce arrhythmias. Finally, in order to reduce the time from diagnosis to surgery, that it is known to cut down the mortality of 50% in the first 48 hours,9 in our opinion, it is important to consider the supraventricular arrhythmias, alone with the D-dimer that has been recently included, to the ADDRS to assess the susceptibility to develop arrhythmias.10

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this manuscript and accompanying images. A copy of the written consent is available for review by the First Author of this report.

CONFLICT OF INTEREST

The authors have no conflict of interests to declare.

AUTHORSHIP

MMC, PV, and FS: planned the case, revised the literature, and drafted the manuscript. AL: contributed to the diagnosis by imaging. UC and AT: provided critical feedback on the surgical approach. All authors: discussed the results, contributed to the final manuscript, and approved the final version.

ORCID

Michele M. Ciulla http://orcid.org/0000-0002-6717-6065

REFERENCES

1. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA. 2000;283(7):897-903.
2. Nazerian P, Mueller C, Soeiro AM, et al. Diagnostic Accuracy of the aortic dissection detection risk score Plus D-Dimer for acute aortic syndromes: the ADvISED prospective multicenter study. Circulation. 2018;137(3):250-258.
3. Tang L, Hu XQ, Zhou SH. AcuteStanford type A aortic dissection mimicking acute myocardial infarction: a hidden catastrophe which should prompt greater vigilance. Acta Cardiol Sin. 2014;30(5):493-496.
4. Lemos AA, Pezzullo JC, Fasani P, et al. Can the unenhanced phase be eliminated from dual-phase CT angiography for
5. Ciulla MM, Astuti M, Carugo S. The atherosclerosis of the sinus node artery is associated with an increased history of supraventricular arrhythmias: a retrospective study on 541 standard coronary angiograms. *PeerJ*. 2015;3:e1156.

6. Ciulla MM, Astuti M, Vivona P, Gallazzi E, Meazza R, Lombardi F. The revascularization of the atrio-ventricular node artery in a case of right coronary artery occlusion promptly discontinues the atrio-ventricular block. *Hellenic J Cardiol*. 2017;58(1):77-79.

7. Koracevic G, Djordjevic D, Glasnovic J. Is significance of atrial fibrillation in acute aortic dissection underestimated? *J Emerg Med*. 2009;37(2):168-171.

8. Ando’ G, Gaspardone A, Proietti I. Acute thrombosis of the sinus node artery: arrhythmological implications. *Heart*. 2003;89(2):E5.

9. Pepper J. Differential aspects of the disease and treatment of Thoracic Acute Aortic Dissection (TAAD)-the European experience. *Ann Cardiothorac Surg*. 2016;5(4):360-367.

10. Erbel R, Alfonso F, Boileau C, et al. Diagnosis and management of aortic dissection. *Eur Heart J*. 2001;22(18):1642-1681.

How to cite this article: Ciulla MM, Vivona P, Lemos A, Sozzi F, Cioffi U, Testori A. Atrial fibrillation, an epiphenomenon of acute Stanford type-A aortic dissection with suspected intimo-intimal intussusception. *Clin Case Rep*. 2018;00:1–4. [https://doi.org/10.1002/ccr3.1701](https://doi.org/10.1002/ccr3.1701)