Aortic dissection type 1, windsock sign, neglected lead, and Kounis syndrome: What a coincidence!

To the Editor;

In the very interesting and important e-page original image published in this month's issue of Anatolian Journal of Cardiology (1), the authors have presented a 61-year-old obese and hypertensive male patient, a smoker, who developed sudden-onset retrosternal chest pain associated with ST and T wave electrocardiographic abnormalities. Computerized tomography showed Stanford type 1 acute aortic dissection spreading from the origin of the coronary arteries to the descending aorta and appearing as intimo-intimal intussusception with windsock linear or curvilinear filling defects in the aortic arch.

Although the electrocardiographic abnormalities were not specified, this case seems of paramount clinical and diagnostic importance, especially in the casualty department in terms of the accurate therapeutic measures employed.

In the patient mentioned above, the dissection had started at the origin of coronary arteries spreading to the descending aorta and probably disturbing and pressing the coronary ostia. Similarly, type 1 dissecting aneurysm affecting the ascending aorta can also expand and press the left main artery and the coronary ostia. In such instances, the electrocardiographic changes may mimic acute myocardial infarction. Until recent years, the AVR lead was regarded as neglected limb lead (2). Indeed, recent reports have shown that ST-segment elevation of more than 1.0 mm in lead aVR associated with widespread ST-segment depression in inferolateral leads, denotes severe left main or three-vessel disease with 80% sensitivity and 93% specificity (3). However, the same electrocardiographic changes can be present in type 1 dissecting aneurysm affecting the ascending aorta that expands and presses the left main artery and the coronary ostia. Although the clinical picture is of acute myocardial infarction, the treatment is completely different and includes emergency surgery and avoidance of anti-platelets, aspirin, and heparin. Such a dilemma is easily solved using point-of-care transthoracic echocardiography in the emergency room to mitigate the risk of inappropriate percutaneous coronary intervention (which might delay implementation of aortic repair surgery) and inappropriate use of aspirin, anti-platelets, heparin, or thrombolysis (which might precipitate hemorrhagic cardiac tamponade (4). A similar electrocardiogram showing the unique sign of ST elevation in lead aVR with reciprocal ST depression in the majority of other leads has been reported recently (5). It concerned a 50-year-old woman who developed bradycardia and hypotension after intravenous sugammadex administration during anesthesia for transabdominal hysterectomy and right salpingo-oophorectomy that was attributed to Kounis syndrome manifestation (6). In another patient with Stanford type 1 circumferential aortic dissection with widespread ST segment depression and intussusception of the aortic intimal flap into the aortic valve, transthoracic echocardiography showed a "heart in heart" picture because of the heart-shaped dissected flap prolapsing into the left ventricle! (7). The physicians in the emergency department wisely did not treat this patient (1) with chest pain as a myocardial case and correctly proceeded to cardiac computed tomography as the emergency outpatient electrocardiogram had been proven inconclusive owing to the patient's obesity.

Nicholas G. Kounis, Virginia Mplani[1], Ioanna Koniari[2]

Department of Cardiology, University of Patras Medical School, Patras-*Greece*

[1]Intensive Care Unit, University of Patras Hospital, Patras-*Greece*

[2]Department of Cardiology, University Hospital of South Manchester NHS Foundation Trust; Manchester-*UK*

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Address for Correspondence: Nicholas G. Kounis, MD, Department of Cardiology, University of Patras Medical School, Queen Olgas Square, 7 Aratou Street, Patras-*Greece*
E-mail: ngkounis@otenet.gr

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Author`s Reply

To the Editor;

We have read with a great interest, the above letter to the editor regarding our e-page original image (1) titled, "Aortic dissection type 1, windsock sign, the neglected lead, and Kounis syndrome: What a coincidence!". We would like to thank them for their contribution about differential diagnosis and management. However, we would like to point out that our diagnosis is obvious and confirmed by surgery without any suspicion. Skeptical ST segment and T wave changes were seen on electrocardiography in our patient, which differentiated it from the case that the authors mentioned. Widespread ST-segment depression in inferolateral leads and ST-segment elevation of more than 1.0 mm in lead aVR could not be seen. Asymmetrical T wave inversion with minimal ST depression could be seen in the inferolateral leads, which might be associated with hypertension and left ventricular hypertrophy. Furthermore, our patient had uncontrolled hypertension. As the echocardiographic examination was suboptimal because of the patient's obesity, we performed cardiac computed tomography (CCT) to rule out acute coronary and aortic syndromes (double rule-out) for this patient. We did not see any pressure on the left main coronary artery and the coronary ostia on CCT, which was also confirmed by surgery. The patient was discharged uneventfully.

In our routine clinical practice, we follow National Institute for Health and Care Excellence (NICE) and European Society of Cardiology (ESC) guidelines for diagnosis and management of chest pain, and we have experienced ESC/EACVI level 3 certified cardiologist and radiologist at our center. We have tremendous experience with CCT with a high workload (over 9000 patients in the last 5 years).

That's why we use CT imaging so often especially in patients in whom echocardiographic imaging is suboptimal.

İbrahim Altun*, Özcan Başaran*, Oğuzhan Çelik*, İlknur Altun, Buğra Harmandar*****

Departments of *Cardiology, and **Radiology, ***Cardiovascular Surgery, Faculty of Medicine, Muğla Sıtkı Koçman University; Muğla-*Turkey*

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Address for Correspondence: Dr. İbrahim Altun,
Muğla Sıtkı Koçman Üniversitesi Tıp Fakültesi,
Kardiyoloji Anabilim Dalı, 48000 Muğla-*Türkiye*
Phone: +90 252 211 51 74
E-mail: ibrahim_altun@yahoo.com