

injury with the needle during administration of tumescent anesthesia, and the thermal energy transfer with the laser probe that could cause degradation and perforation of the vein wall with arterial injury.

Very few cases of arteriovenous fistula in the external iliac system are reported in the literature, probably caused by the inadvertent advancement of the probe through the saphenous vein arch in the external iliac vein either with mechanical perforation through the vein into the artery or vessel wall damage due to activation of the laser while still within the external iliac vein. Lack of tumescent anesthesia at this level likely increases the risk of damaging the arterial wall. (4-5) Echo-Doppler and CT angiography are the non-invasive imaging methods to confirm the diagnosis, and allow for the characterization of the arteriovenous fistula (location, size and flow); an additional angiography is only required if an intervention is considered for marked lower limb edema, recurrent varicose veins, venous complications (phlebitis, ulceration), intermittent lower limb claudication from steal syndrome or heart failure. (2) The cases of external iliac arteriovenous fistula –including our case– were reported in the post-treatment of the internal or great saphenous vein, and all of them presented systo-diastolic murmur with thrill in the homolateral inguinal region to treatment. Diastolic heart failure was the most common clinical presentation, together with dyspnea, ascites, edema and lower limb heaviness with unilateral predominance, and occurred between 3 weeks and 2 years after surgery. (4, 5) Cases of heart failure and superficial femoral artery arteriovenous fistula have also been described after laser ablation of the saphenous vein. (6) All patients required either conventional surgery or endovascular treatment with covered or hybrid stent, depending on the anatomical proximity of the arteriovenous fistula to the inguinal ligament and the size of the fistula. In every case, symptoms were resolved, with disappearance of the murmur and inguinal thrill. (4, 5)

In summary, arteriovenous fistula is a rare complication of endovenous laser treatment, but it can develop high flows and cause heart failure in some patients. Therefore, the presence of iatrogenic arteriovenous fistulas should be ruled out in patients with signs and symptoms of heart failure and a recent or long-term history of saphenous vein failure treatment with laser ablation.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/ Supplementary material).

Ethical considerations

Not applicable

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Persistent ST-Segment Elevation Due to Myocardial Infiltration

In most cases, ST-segment elevation is due to acute coronary occlusion. However, atypical presentation or atypical evolution should make us consider other etiologies.

We present the case of a 71-year-old patient with persistent chest pain and shortness of breath. The electrocardiogram (ECG) showed ST-segment elevation in leads V2 through V4. A severe non-occlusive lesion was found in the distal left anterior descending artery and a stent was implanted, resulting in unsuccessful ST-segment normalization. Days later, symptoms recurred and a computed tomography (CT) scan revealed lung cancer with multiple metastases including the myocardium. ST-segment elevation in the ECG could indicate tumour invasion of the myocardium at the level of the left ventricle.

A 71-year-old man presented at the emergency department with persistent chest pain and shortness of breath. The ECG showed ST-segment elevation in leads V2 through V4 (Figure 1a). He had a history of arterial hypertension and smoking. He was referred to another hospital to undergo urgent revascularization. Coronary angiography showed critical narrowing at the distal segment of the left anterior descending artery (LAD), which was successfully treated with a drug-eluting stent (Figures 2a and 2b). Atypical evolution was observed, with persistent ST-segment elevation but without necrosis (no Q-waves [Figure 1b], no troponin el-

evation, no abnormalities in ECG). Symptoms recurred after 7 days (refractory chest pain, dyspnea and orthopnea). CT revealed primary lung cancer in the right upper lobe, myocardial interventricular septum infiltration (Figure 3) and metastases in bones and suprarenal glands. During this hospitalization, the ECG showed ST-segment elevations with a similar pattern, which was probably due to tumor invasion of the myocardium. Histopathological examination was not performed; however, CT findings suggested lung adenocarcinoma. Patient died one month after percutaneous coronary intervention (PCI), under palliative treatment.

Although acute myocardial infarction (AMI) is the most frequent cause of ST-segment elevation, it could occur in other circumstances, as in tumor with myocardial involvement. (1)

The most common underlying malignancies with secondary cardiac involvement include carcinoma of the lung, breast, esophagus, stomach, and kidneys, as well as melanoma, lymphoma and leukemia. Primary lung carcinomas account for around one third of cardiac metastases. (2, 3)

Myocardial metastasis from neoplastic disease is of

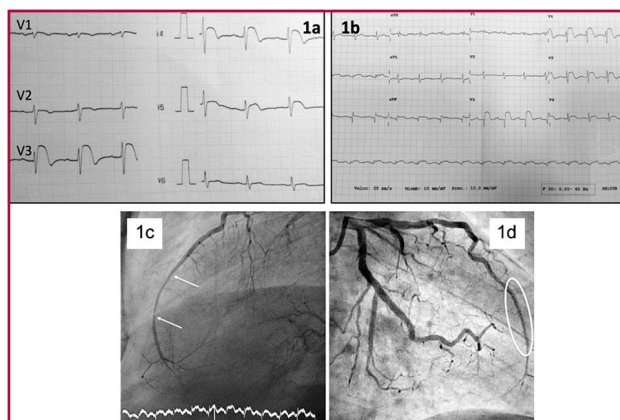


Fig. 1. **1a.** Baseline ECG: ST-segment elevation in leads V2 to V4. **1b.** Discharge ECG: ST-segment elevation in leads V3 to V6. Q-wave in V3. Minimal ST-segment elevation in leads D2-D3-avF. **1c.** Left anterior descending artery angiography before treatment. **1d.** Image following stent implantation.

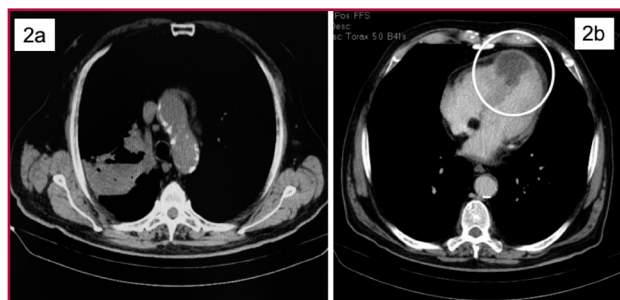


Fig. 1. Computed tomography. **2a.** Lung cancer in the right upper lobe. **2b.** Myocardial interventricular septum and apex infiltration.

ten clinically unapparent, and very difficult to diagnose. Of 151 consecutive autopsies of lung cancer patients, cardiac metastasis was found in 67 patients (44.4%), while myocardial metastasis was found in 11.9% of patients. ECG of patients with myocardial metastasis revealed ST-segment and T wave abnormalities and various types of arrhythmia. These alterations were observed in 4 patients with myocardial metastasis, and in 6 without this pathology (pericardial metastasis alone). (2) Case reports of ST-segment elevation without coronary occlusion have been described in different ECG leads depending on the infiltrated area of the heart. (2, 4, 5)

Patients with cancer and normal ECGs are unlikely to have cardiac metastasis. The ECG finding of myocardial ischemia or injury has high specificity (96%, $p < 0.000001$) for cardiac metastasis. (4) On the other hand, ST-segment and T wave abnormalities are an unspecific finding of myocardial metastasis. (2)

Myocardial involvement by direct lung cancer invasion is unusual and is often clinically silent, although it can cause malignant pericardial effusion with or without symptoms of pericarditis, arrhythmias, heart failure, and rarely AMI. (2) AMI might be caused by tumor embolization or direct compression of the coronary arteries. (5)

The present case was initially misdiagnosed as acute coronary syndrome with ST-segment elevation due to the presence of cardiac pain and ST-segment elevation on ECG. However, after fatal evolution of the patient in one month, we should conclude that the abnormal ST-segment elevation in this patient was not a manifestation of AMI, but possibly caused by an alteration in the myocardial electrical properties associated with tumor invasion. Moreover, the presence of normal cardiac enzymes was also an important evidence that the ECG abnormalities were not associated with either AMI or myocarditis. (6)

Conflicts of interest

None declared.

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Ethical considerations

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Short QT Syndrome Associated with Noncompacted Myocardium

Cardiomyopathies are a heterogeneous group of myocardial diseases associated with mechanical or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilation and are due to a variety of frequently genetic causes. Cardiomyopathies are confined to the heart or are part of generalized systemic disorders, which may lead to cardiovascular death or progressive heart failure-related disability. (1)

Noncompacted myocardium is a primary genetic cardiomyopathy first described in 1984 by Engberding, characterized by myocardial hypertrabeculation with sinusoids in communication with the ventricular cavity, resulting from a sudden arrest in the normal embryogenesis prior to compaction, and involving predominantly the distal portion of the left ventricle. (2)

Short QT syndrome (SQTS), introduced as an association with sudden death (SD) in 1993 by Algra, but first described by Ghusack in 2000, who reported 6 unrelated family cases with atrial fibrillation as the form of presentation, is a primary cardiomyopathy described within the channelopathies generating electrical dysfunction. (3) Diagnosing SQTS is not easy, since in a first instance, numerous studies associated corrected QT interval (QTc) <300 ms with SD, but then, the limit point proposed was two standard deviations from the normal QTc (350 ms), so the cut-off point to consider a short QT interval would be 320 ms. In 2011, Gollob et al. proposed a diagnostic score based on QTc, the JT interval, the genotype, and the patient's medical record and his/her family history, assigning points to each category. An overall score of 4 points or greater indicates a high-probability of SQTS, 3 points an intermediate probability and 2 points or less a low probability of SQTS. (4) In 2015, the European Society of Cardiology (ESC) standardized criteria and determined that the presence of QTc <340 ms (Class I, level of evidence C), or a QTc <360 ms with at

least one associated genetic mutation, family history of SQTS, family history of SD before the age of 40, or survival from an episode of ventricular fibrillation (VF) or documented ventricular tachycardia (VT) in the absence of structural heart disease (Class IIa C) constitute diagnosis of SQTS.

A 19-year-old male patient, without known personal pathological history, with a history of a 4th degree relative (cousin) who died of SD at the age of 1 year, and a 2nd degree relative (maternal uncle) who also died of SD at the age of 15 months, consulted for paroxysmal palpitations and dyspnea in FC II-III associated with an episode of chest pain. Sinus bradycardia at 40 bpm was detected, with adequate chronotropic response.

The ECG showed absolute QTc of 320 ms, and corrected by Bazett's formula of 292 ms (Figure 1). The echocardiogram revealed myocardial hypertrabeculation in the mid-apical region of the low and lateral walls, suggestive of noncompacted myocardium (Figure 2). The MRI reported preserved left ventricular size and function and normal myocardial wall thickness. Within the intraventricular cavity, abundant trabeculae in the lateral-apical region and apical segment were observed (Figure 1), with a relation between noncompacted and compacted areas of 2.4 (noncompacted area 16.5 mm, and compacted area 6.8 mm in a short axis view), meeting the diagnostic criteria for noncompacted myocardium (Figure 2).

The 24-hour Holter evidenced average HR of 45 bpm, minimum 33 bpm, maximum 94 bpm, without ventricular arrhythmia, maximum and minimum QTc of 375 ms and 276 ms respectively, and minimum absolute QT of 360 ms, with scarce daily variability despite high HR variability.

No familial phenotypes have been found in the screening for familial SQTS.

With the diagnosis of SQTS and the patient's consent, a cardioverter defibrillator (ICD) was implanted.

So far, SQTS associated with noncompacted myocardium has not been described.

Short QT syndrome was diagnosed according to the 2016 ESC guidelines for ventricular arrhythmias and prevention of SD due to QTc <340 ms (class IC). Based on the Gollob score, we have a patient with high probability of SQTS (4 points QTc <330 ms, and second-degree family history of SD) without performing genetic testing. The yield of genetic testing for SQTS is low (14%). (4)

So far, noncompacted myocardium has been described in association with other conditions, such as long QT syndrome; recently, a novel entity has been introduced, which identified the mutation in the HCN4 gene that is a phenotypic manifestation of sinus node dysfunction, noncompacted myocardium, mitral valve prolapse, and aortic dilation. Of all the family cases analyzed, no patients with short QT have been reported. (5)

Moreover, SQTS has been associated with Brugada syndrome and early repolarization syndrome, with sig-