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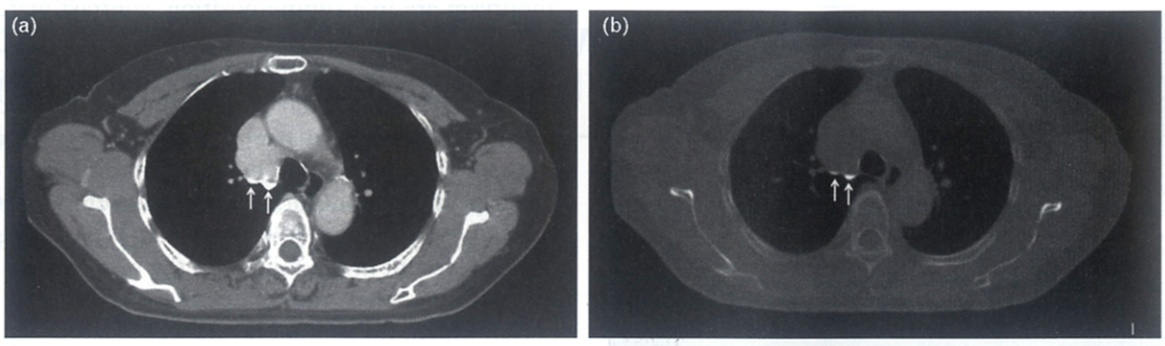
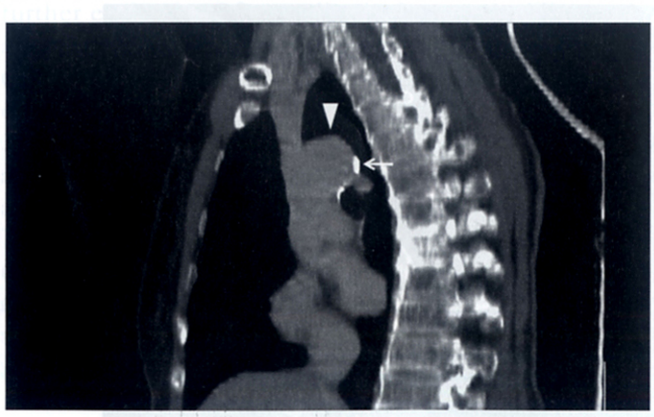
Fig. 3



(a) Late-phase postcontrast computed tomography shows the homogeneously enhanced mass with an attenuation of 161 Hounsfield units similar to that (166 Hounsfield units) of the superior vena cava. Collection of the injected contrast material with biconvex curvilinear contour (arrows) is demonstrated. (b) The same level imaged with bone window clearly reveals fluid–fluid layers (arrows) in the anterior aspect of the contrast material collection.

partial and total thrombus of idiopathic azygos vein aneurysm, which are associated with an increased risk of pulmonary embolism, have been reported in the literature.[3,7,8] Some patients have undergone surgical operation or endovascular treatment to prevent the theoretical risks of rupture and thrombus formation. In particular, intraluminal thrombus can flow into the pulmonary arteries causing pulmonary embolism. There is not enough information about natural history, but a case of aneurysm of the azygos vein has also been reported, which was managed conservatively for 4 years without any change in the size of the aneurysm or thrombus formation.[1] It was concluded that follow-up CT might be a reasonable approach for treating asymptomatic patients with nonthrombosed azygos vein aneurysms. In the present case, the patient was reluctant for further intervention and preferred conservative management.

Fig. 4



Sagittal image of maximum intensity projection of the right side of the mediastinum reveals an enhanced mass along the pathway of the azygos arch (arrow head), with a collection of injected contrast material presenting as a fluid–fluid layer (arrow).

CE-CT is a safe and appropriate choice for the diagnosis of azygos arch aneurysm. Contrast material collection with a fluid–fluid layer appearance may be an interesting CT finding of azygos arch aneurysm when it involves the azygos valve.

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Cardiac memory on Memorial Day: a nice coincidence

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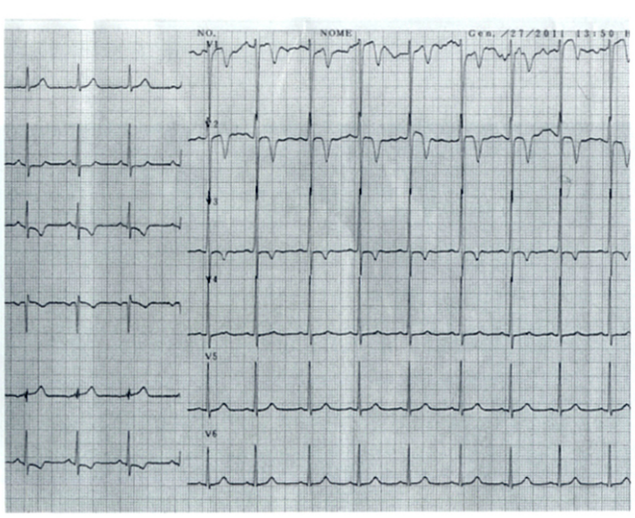
To the Editor

Memorial Day (27 January) commemorates worldwide the victims of the Holocaust. Curiously, on the same day this year we observed an unusual case that emphasizes the importance of being aware of the memory.

On that day (27 January 2011), a 23-year-old man came to our consulting room. He suffered from fleeting episodes of precordial pain, not correlated with physical activity. He had no coronary risk factor. Surprisingly, his resting ECG showed deep negative T waves (TW) in V1–V3, D3 and aVF (Fig. 1). Physical examination, echocardiogram and serum markers were normal. Although younger colleagues pushed for coronary angiography, a less youthful cardiologist played the card of memory. After a small increase of sinus rate produced by hyperventilation, phase 3 left bundle branch block (LBBB) appeared, with good concordance between TW negativity during normal conduction and polarity of QRS during LBBB (Fig. 2). This simple manoeuvre disclosed the mystery of negative TW, demonstrating that they were due to cardiac memory. We didn't perform any invasive examination in our young patient who, as a first hypothesis, could have an initial Lenègre's disease.

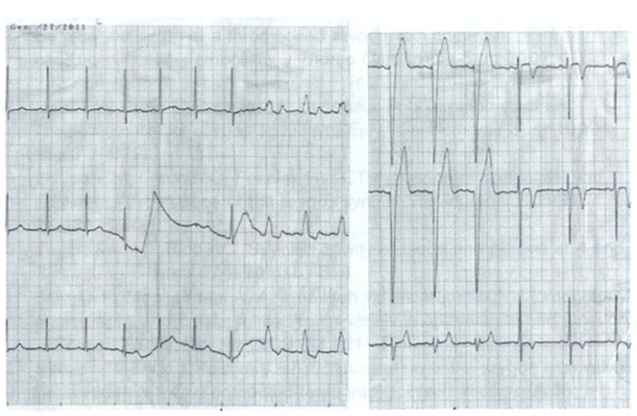
For too many years it has been dogmatically accepted that TW changes are either primary or secondary in nature. Thus, when in the late 1960s some reports about intermittent LBBB with negative TW during normal conduction appeared, the general opinion was that these TW alterations were ischemic in nature.[1] In 1978, a degree thesis from a young student of Pavia University (the author of this letter) pointed out that perhaps the negative TW in intermittent LBBB were not ischemic, but related to abnormal activation during LBBB.[2] Some years later, Rosenbaum *et al.*[1] described cardiac memory. They postulated that a change in ventricular activation, such as the one induced by LBBB (or by ventricular pacing or by ventricular pre-excitation), caused subsequent occurrence of pseudoprimary TW changes, whose direction was concordant with the QRS forces of LBBB. Furthermore, they stated that TW changes develop in a gradual cumulative fashion, suggesting that the heart in some way 'learns', and seems to keep a memory of the previous

Fig. 1



Cardiac memory on Memorial Day, 27 January 2011. On the left: bipolar (D1, D2, D3) and unipolar (aVR,aVL,aVF) limb leads. On the right: unipolar precordial leads (V1–V6). The ECG showed normal conduction and deep negative T waves in right precordial leads.

Fig. 2



On the left: during hyperventilation (leads V4–V6) a slight shortening of sinus cycle was evident, and phase 3 left bundle branch block (LBBB) appeared. On the right: during registration of V1–V3, LBBB spontaneously disappeared, in concomitance with a slight increase of sinus cycle. Notice the good concordance between T-wave negativity during normal conduction and negative polarity of QRS during LBBB. The same behaviour was evident in D3 (not shown).

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electrical activity.[3] Today, cardiac memory has no more secrets, its molecular mechanisms have been pointed out,[4] and the likelihood that memory represents a monogenically determinate function is speculated.[5] But beyond its physiological mechanisms, the recognition of cardiac memory has obvious diagnostic and therapeutic implications, and serious mistakes may often derive from the inability to recognize it. So, don't forget the memory!

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