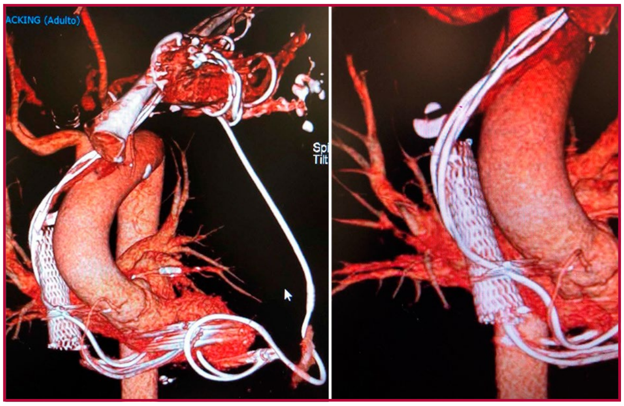

Fig. 2. Three-dimensional reconstruction of the multislice computed tomography scan, showing the different pacing leads and the stent implanted in the superior vena cava (right image: detail of the entrapped leads).

presented, compression of pacemaker leads by a stent in the SVC is exceptional and constitutes a challenge for extraction. Hybrid cardiac surgery with simultaneous percutaneous extraction was a safe option to treat this unusual and complex case. In view of these results, the complexity of extracting leads entrapped between a stent and the SVC wall should be considered in case of SVC syndrome requiring stent placement.

Conflicts of interest

None declared.

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

Ethical considerations

RAB is Director of the Argentine Journal of Cardiology.

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Complete Left Bundle Branch Block and Blunt Cardiac Injury: A Lesson Learned

Chest injury is the second leading cause of mortality after head injury, and accounts for 20-25% of all accidental deaths. (1) Even though the treatment of patients with polytrauma exceeds cardiological management, patients with closed chest trauma presenting with arrhythmias, elevated cardiac enzymes or pain may require our evaluation. These findings may indicate blunt cardiac injury, a condition in our specialty that causes high mortality. Most cardiac complications secondary to blunt cardiac injury due to closed chest trauma occur within the first 24 hours (65% are already present on admission), (2) and cardiologists should be alert to their clinical presentation and outcome.

We report the case of a 26-year-old male patient with no previous relevant history who was admitted after falling from a motorcycle due to frontal collision with a car. He presented polytrauma with closed-chest and right forearm trauma, encephalocranial trauma and posterior loss of consciousness. On admission, the electrocardiogram (ECG) showed complete atrioventricular block (AVB), QRS complex with complete right bundle branch block (RBBB) morphology and heat rate (HR) of 25 bpm (Fig. 1a). On physical examination, the patient presented an open forearm fracture (Gustilo I) and appeared lucid without signs suggestive of cerebral ischemia and with normal blood pressure. Some minutes later, 3:1 AVB with LBBB conduction developed that was not present on an ECG taken the previous year. After isoproterenol administration, the HR increased to 60 bpm (Fig. 1b). An echocardiogram was urgently performed, with normal results. The laboratory tests showed elevated troponin I level of 2.16 IU/L (normal value <0.02 IU/L). The patient presented complete AVB and complete LBBB during the first 24 hours after admission and was asymptomatic; then, the heart rhythm alternated between sinus rhythm and sinus arrest with persistent complete LBBB. The tests performed to evaluate polytrauma did not show costal fractures, but the computed tomography scan showed signs of pulmonary contusion, right pleural effusion and distal radial and cubital fracture. Another echocardiogram was performed 24 hours after admission which showed left ventricular hypertrophy (IVS 1.3 mm and LVPW 1.1 mm) with abnormal septal motion due to the complete LBBB). Cardiac magnetic resonance imaging was performed to evaluate the presence of edema, hematoma or fibrosis as a cause of rhythm disturbance, with normal results. Four days after the road accident, the patient recovered the sinus rhythm with a heart rate of 55 bpm but as the complete LBBB persisted and he had to undergo surgery due to the forearm fracture, an electrophysiology study was performed on day 14. The study showed normal AV conduction and prolonged HV interval (80 ms) in the context of a complete LBBB. A temporary pacemaker was placed before surgery. Six months later, the patient persists asymptomatic with complete LBBB.

Cardiac injury can be due to penetrating or blunt chest trauma. These types of lesions are completely different in their etiology, clinical presentation, implementation of diagnostic methods, treatment and prognosis. Blunt cardiac injury secondary to closed chest trauma can be caused by the sudden compression of the heart between the sternum and the spine, or by acceleration/deceleration movements, and can affect the free wall, interventricular septum, heart valves, subvalvular apparatus, the conduction system, or the coronary vessels.

There are no updated reports on the incidence of cardiac involvement in chest trauma, probably due to the lack of systematic search, the difficulty in interpreting symptoms and, occasionally, because electric abnormalities may be temporary. (3) An ECG and troponin levels should systematically be obtained on admission because of their 100% negative predictive value. (4)

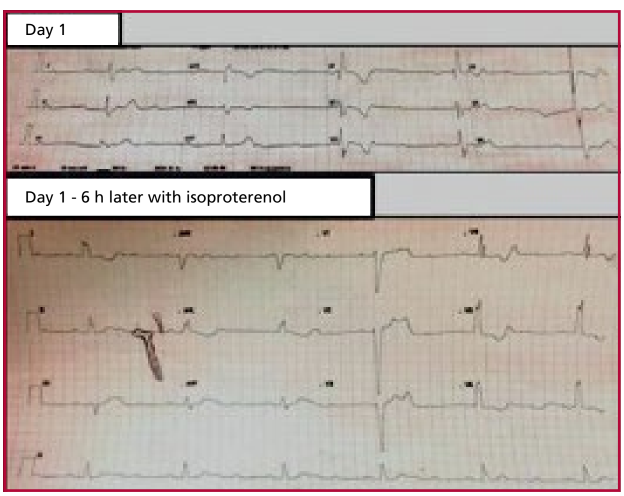

Fig. 2. 1a. ECG showing complete atrioventricular block with ventricular escape rhythm with complete RBBB morphology and heart rate (HR) of 25 bpm
1b. ECG at 6 h, with isoproterenol infusion: 3:1 AVB with LBBB conduction.

Table 1. Classification of chest injury. American Association for the Surgery of Trauma

Grade	Heart injury scale. American Association for the Surgery of Trauma Injury description
I	Blunt cardiac injury with minor ECG abnormality Pericardial wound without cardiac injury, cardiac tamponade, or cardiac herniation
II	Blunt cardiac injury with heart block or ischemic changes without heart failure
III	Blunt cardiac injury with sustained or multifocal ventricular extrasystoles. Cardiac injury with septal rupture, pulmonary or tricuspid valve incompetence, papillary muscle dysfunction and coronary artery occlusion without heart failure. Blunt cardiac injury with pericardial laceration with cardiac herniation. Blunt cardiac injury with heart failure. Penetrating tangential myocardial wound without affecting the endocardium, endocardium, but with cardiac tamponade.
IV	Cardiac injury with septal rupture, pulmonary or tricuspid valve incompetence, papillary muscle dysfunction or coronary artery occlusion with signs of heart failure. Cardiac injury with mitral or aortic valve incompetence. Cardiac injury involving the right ventricle or one of the two atria.
V	Cardiac injury with proximal coronary arterial occlusion. Cardiac injury with left ventricular perforation. Stellate wound with <50% tissue loss of the right ventricle, right atrium, or left atrium.
VI	Blunt injury with cardiac avulsion or penetrating wound producing > 50% tissue loss of a chamber. Advance one grade for multiple wounds to a single chamber or multiple chamber involvement.

Commotio cordis describes sudden cardiac death resulting from blunt-force trauma to the chest causing ventricular fibrillation. Excluding commotio cordis, the American Association for the Surgery of Trauma (5) has described six grades of cardiac injury (Table 1), ranging from nonspecific ECG abnormality (grade I) to cardiac perforation (grade V) and even avulsion of the heart involving >50% of cardiac tissue (grade VI). According to this scale, our patient belonged to grade II (heart block). Conduction abnormalities include complete RBBB, LBBB, bifascicular blocks or complete AVB; (6) complete RBBB is more common (7) than complete AVB and LBBB due to its anterior location and its proximity with the sternum. Atrioventricular block is rare and has been reported in only 50 cases according to a systematic review. (8) Complete AVB occurred within 72 hours of injury in 80% of the patients, and 1:1 AV conduction was restored within 7–10 days in about half of survivors probably after edema resolution, as it is one of the probable mechanisms of conduction disturbances. Permanent pacemaker implantation was indicated in about 50% of the patients due to recurrent or permanent complete AVB and mortality rate was 20%. (8) In our case, the patient presented complete AVB with QRS morphology of complete RBBB. Once the sinus rhythm was restored, the conduction abnormality was a complete LBBB because the left branch was the fascicle injured by the blunt cardiac injury, and persists even 6 months after the accident, which turns the case exceptional. Given the lack of specific recommendations on the need for pacemaker, we believe that reporting this type of case will allow the possibility of making recommendations in the future based on the knowledge of the natural history of patients with cardiac injury after a closed chest injury.

Conflicts of interest

None declared.

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

Ethical considerations

Not applicable

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Chronic Dissection of the Abdominal Aorta. Endovascular Therapy with Novel Stent-Graft in-situ Fenestration

Although thoracic endovascular aortic repair has become a promising treatment for complicated acute type B dissection, its role in treating chronic post-dissection thoraco-abdominal aortic aneurysm is still limited owing to persistent retrograde flow into the false lumen (FL) through abdominal and/or iliac re-entry tears. (1) Aortic dilation is the main factor to determine long-term survival in these patients.

The aim of this study is to demonstrate the feasibility of endovascular treatment using in situ stent-graft fenestration for the left renal artery, sealing the re-entry tear and completely redirecting the blood flow into the true lumen (TL).

This approach was used in a 62-year-old male patient with dilation of the abdominal aorta discovered in an abdominal ultrasound during the preoperative assessment before an elective cholecystectomy. Patient's risk factors were hypertension and chronic smoking.

Physical examination revealed a pulsating aortic beat, and femoral and popliteal arterial pulses were normal. The computed tomography angiography showed a large ulcer in the descending thoracic aorta associated with an intramural hematoma, with a transverse aortic diameter of 79 mm (Fig. 1A). A chronic dissection with a patent FL and an aneurysmal dilation of the abdominal aorta were also observed with a transverse diameter of 59 mm (Fig. 1B). It was also possible to visualize the origin of the celiac trunk (CT), the superior mesenteric artery (SMA) and the right renal artery emerging from the TL and the left renal artery from the FL, with a re-entry tear in this sector. A compression of the TL was observed in the infrarenal abdominal aorta and there was a distal re-entry tear in the left external iliac artery (Fig. 1C).

A two-stage endovascular repair was decided due to the complexity of the case. Firstly, the giant ulcer in the descending aorta was treated by implanting two self-expandable Hercules™ stent grafts. Three months later, endovascular repair of the abdominal aorta was performed in the catheterization laboratory under general anesthesia and with invasive blood pressure monitoring. A spinal drainage catheter was inserted to monitor cerebrospinal fluid (CSF) pressure and both femoral arteries and the right subclavian artery were incised. Two 70-cm length multipurpose type introducers (8 Fr and a 7 F Flexor®) were introduced through the subclavian arteriotomy for selective cauterization of the SMA and the right renal artery, respectively. An PTFE-coated SIGBI G SETA® stent-graft was positioned at 3 cm of each vessel (one measuring 8 x 38 mm in the SMA and another 7 x 38 mm in the right renal artery; chimney technique) to allow blood flow in these vessels. Then, a 25 x 80 mm RAKB SETA® balloon-expandable full stented graft was introduced in the abdominal aorta via the right femoral artery, and a 25 x 50 mm SETA