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

MUG® in-situ fenestration expandable stent-graft was placed in both renal arteries and intentionally covering the origin of the left renal artery. Once this stent graft was expanded, the other stents placed in the SMA and right renal artery were expanded.

After the in-situ fenestration stent-graft was deployed, an 8 Fr OSCOR® guidewire was introduced through the right femoral artery and was positioned in the ostium of the left renal artery (Fig. 2A). A 0.035-inch straight Terumo® hydrophilic guidewire was advanced through the membrane of the SETA MUG into the left renal artery and was then exchanged for a 0.014-inch support guidewire to advance a 3.5 mm coronary angioplasty balloon catheter (Fig. 2B) and then a 5-mm balloon catheter to create a fenestration in the membrane. Two 6 x 22 mm SIGBI G SETA® PTFE-coated stents were introduced through this orifice, to redirect the blood through the TL into the left renal artery, closing the re-entry tear (Fig- 2C).

The final part of the procedure consisted in implanting two straight balloon expandable RIK SETA® branches for both common iliac arteries using the kissing stent technique, and a supplementary balloon expandable RIK F SETA® extension for the left external iliac artery, with a fenestration for the origin of the internal iliac artery, thus sealing the distal re-entry tear.

Mean blood pressure was strictly maintained at ≥ 80 mm Hg to avoid spinal hypoperfusion. Cerebrospinal fluid pressure did not vary during the procedure and the spinal drainage was removed 24 h later.

The procedure lasted 330 minutes, 420 cm3 of non-ionic contrast agent were used and mean fluoroscopy time was 93 minutes. The patient remained hospitalized for 4 days.

A computed tomography angiography was performed 30 days later. Both renal arteries and the SMA were patent without endoleaks and the FL was completely occluded, so all the blood flowed through the TL (Fig. 3).

About 20-40% of the patients with acute type B thoracoabdominal aortic dissection evolve with aneurysmal dilation of the FL during follow-up. (2) Conventional surgery and the novel hybrid surgery represent valid therapeutic options. Both techniques have high morbidity and mortality, even in experienced centers. (3)

Complete endovascular treatment of chronic aortic abdominal dissections with fenestrated or branched stent grafts has been, until now, limited. This is mostly due to the narrow working space in the TL (partially collapsed by the FL) and to the impossibility of sealing reentry tears of the FL because of the lower anatomical position between the CT and the renal artery. (4)

There are reports on the use of endovascular occluders to seal the reentry tear of the FL with poor outcomes, especially related with the persistence of endoleaks and lack of remodeling of the aortic lumen. (5)

In our case, the use of a SETA MUG® stent graft

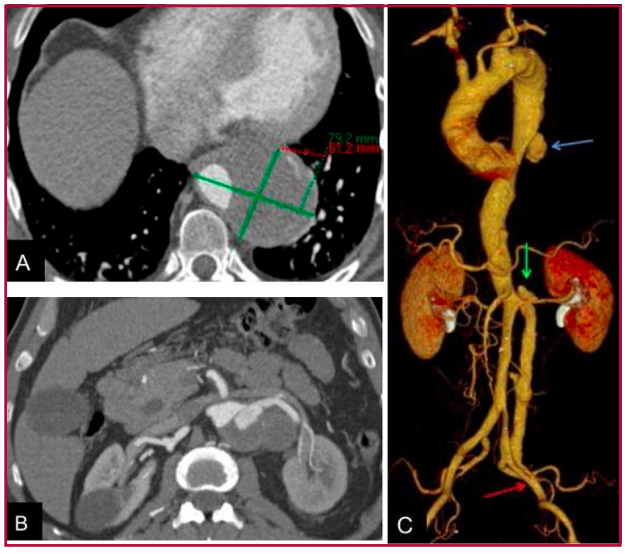

Fig. 1. A and **B**. Axial sections of computed tomography angiography. (A) Large periaortic intramural hematoma in the sigmoid aorta. (B) Left renal artery emerging from the false lumen (FL) associated with aneurysmal dilation of the aorta. **C.** Three-dimensional reconstruction showing an ulcer in the descending aorta (blue arrow), reentry tear of FL in the left renal artery (green arrow) and reentry tear of the FL in the left external iliac artery (red arrow).

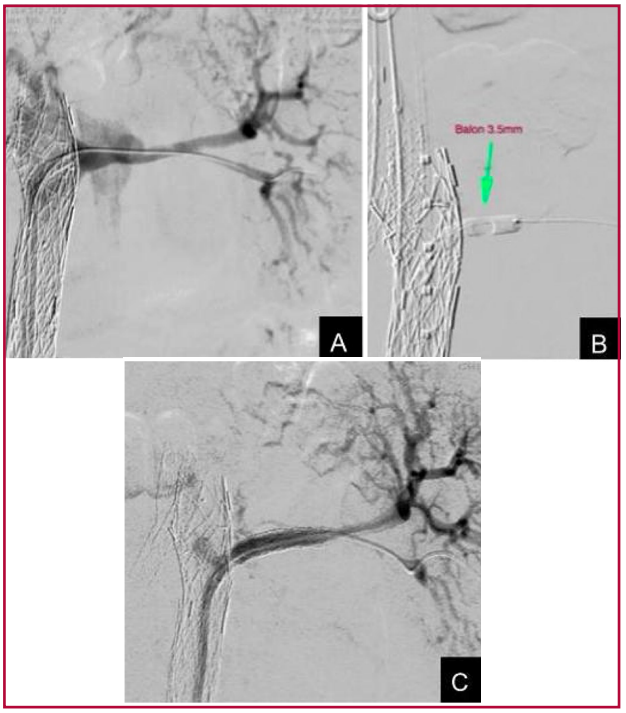

Fig. 2. A. Angiographic control through the guidewire showing patent MUG graft and false lumen (FL). **B.** Balloon angioplasty for in situ fenestration. **C.** Angiographic control showing blood flow through the true lumen with complete sealing of the FL

with in situ fenestration gave us the possibility of sealing the FL reentry tear at the level of the left renal artery ensuring blood flow into the kidney during the entire procedure. The high porosity of the device allows the placement of covered stents through its meshes. We did not find any publication in the international literature about the use of a stent with these characteristics.

We decided to use coated stents for the SMA and the right renal artery (chimney technique) to ensure blood flow to both arteries and because the risk of endoleak during follow-up is lower due to the small diameter of the TL. In a recent study, Oikonomou et al. reported that the use of covered stents in the visceral branches for the treatment of post-dissection thoracoabdominal aortic aneurysm is feasible. (6)

The distal reentry tear in the left external iliac artery was sealed with a straight balloon expandable stent graft with a fenestration towards the internal iliac artery, which allowed blood flow into the vessel. There was no change in creatinine levels immediately after the intervention and in the last control before the computed tomography scan.

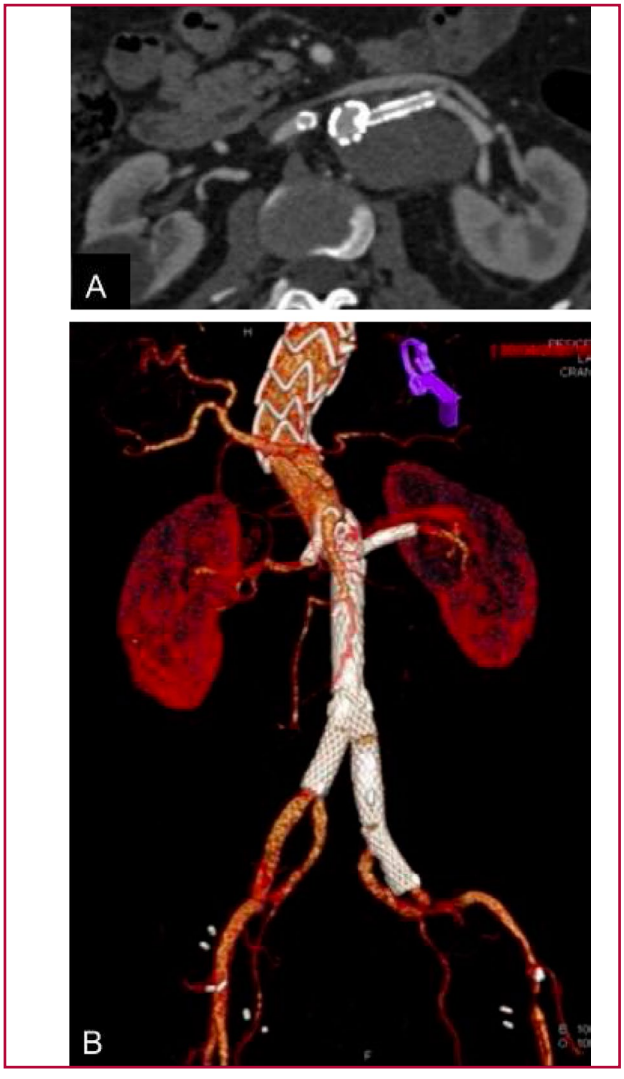

Fig. 3. A. Computed tomography angiography (axial section) showing the patent stent connected with the left renal artery with complete thrombosis of the aneurysmal dilation.
B. Three-dimensional reconstruction showing patent visceral arteries, normal flow through the true lumen and patent internal iliac arteries

While the rate of cardiovascular and renal complications is relatively low, the risk of spinal cord ischemia during endovascular repair of thoracoabdominal aneurysms remains significantly high, hence the importance of performing these procedures in two stages.

We treated the thoracic aorta three months before repairing the abdominal aorta to reduce the risk of paraplegia. Nevertheless, during the repair of the abdominal aorta, we placed a spinal drainage catheter, maintained a stable mean blood pressure not <80 mm Hg, and the left subclavian artery and both internal iliac arteries remained patent.

The promising immediate outcome in this patient demonstrates that the use of this new stent graft in-situ fenestration is feasible and could introduce a new concept in the endovascular approach of these patients with post-dissection thoracoabdominal aneurysms. However, further studies with larger number of patients and longer follow-up will be essential to evaluate the mid and long-term effectiveness of this technique, as well as the need for eventual secondary procedures.

Conflicts of interest

None declared.

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Ethical considerations

Not applicable

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Severe Pericardial Calcification: Does it Still Exist?

Constrictive pericarditis is the final phase of a pericardial inflammatory process with progression to chronic involvement producing pericardial constriction and severe diastolic dysfunction. Only 1.8% of inflammatory pericarditis progress to constriction: postoperative pericarditis ranks first, followed by radiation-induced pericarditis and those caused by autoimmune diseases and infections.

Severe circumferential fibrosis and calcification are markers of adverse outcome and make the etiologic diagnosis difficult.

We describe the clinical and pathological findings of a case of pericardial constriction and cardiac osseous metaplasia in an immunocompetent patient.

A 58-year-old male patient with a history of hypertension and non-insulin-requiring diabetes mellitus visited the outpatient clinic due to edema of the lower extremities and class III dyspnea during the previous month. He was born in the province of Chaco in northeastern Argentina where he lived with his family in a poor home in overcrowded conditions.

On physical examination, his general status was poor, with cachexia and evident signs of advanced heart failure.

The electrocardiogram showed complete right bundle branch block with no other abnormalities. The laboratory tests revealed anemia, elevated liver enzymes and hypoalbuminemia. Serology tests for viral hepatitis B and C and HIV were negative, with positive tests for Chagas disease and a PPD test <5 mm.

The chest X-ray showed severe circumferential pericardial calcification (Figure 1 A). The patient underwent Doppler echocardiography which confirmed the presence of pericardial calcification and signs of constriction. The chest computed tomography scan also

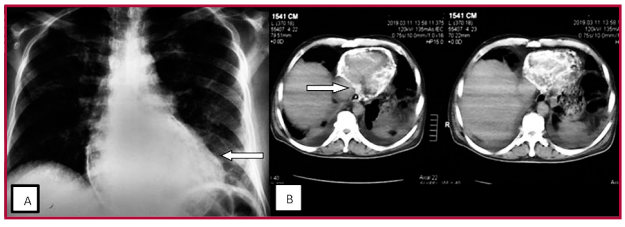

Fig. 1. A) Chest-X ray. B) Chest computed tomography scan with diffuse pericardial calcification (white arrows)

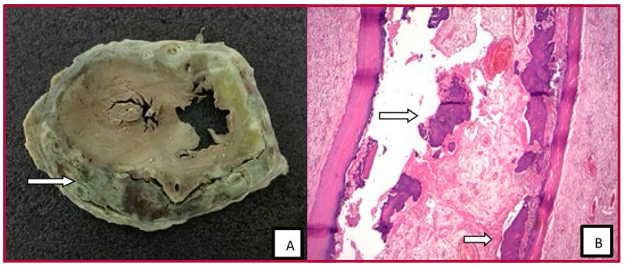

Fig. 2. A) Gross examination of the explanted heart with severe pericardial calcification. B) Optical microscopy image showing inflammatory infiltrate without granuloma and osseous metaplasia.

confirmed circumferential calcification of the pericardium with an "egg-shell" pattern (Figure 1B), and the presence of normal size calcified mediastinal, parahiliar, axillary and mesenteric lymph nodes.

A diagnosis of constrictive pericarditis was made, and after discussing the case in an interdisciplinary meeting, pericardiectomy was decided after improving the patient's general status, in terms of nutrition and heart failure.

Ten days after hospitalization the patient presented signs of multiple organ failure secondary to sepsis caused by a probable respiratory infection, and he finally died.

The autopsy report was chronic constrictive pericarditis, active and purulent, with pericardial areas of dystrophic calcification and osseous metaplasia (Figure 2).

Chronic constrictive pericarditis is rare and represents 1.8% of inflammatory pericarditis. (1) In Argentina, tuberculosis (TB) is the most common cause of pericardial constriction, but is a rare form of extrapulmonary TB, with a mortality rate of 60% when not treated. (2)

Pericardial involvement usually occurs from lymphatic spread of Mycobacterium tuberculosis, while hematogenous or contiguous spread from a primary pulmonary focus is uncommon.

The presence of fibrosis, pericardial calcification and osseous metaplasia reflect chronic and irreversible constriction. (3) The absence of active inflammation parameters (normal erythrocyte sedimentation rate and C-reactive protein and absence of leukocytosis) predict the lack of response to medical treatment.

Although these patients are candidates for surgical treatment (4), the main obstacle is the inability to remove calcium from the parietal pericardium, resulting in extremely high mortality (40%) (5) associated with several factors: the poor general condition of these patients at the time of surgery, advanced age, advanced functional class (III-IV), presence of moderate to severe pericardial effusion and ascites, many of which were present in this patient.

Tuberculosis was the most likely etiology in our patient due to his living conditions and epidemiological environment. In our setting, TB is still a common cause of constrictive pericarditis and pericardial cal-