

Implantation of an Andrastent XL in an adult with advanced chronic heart failure due to coarctation of the aorta

Implantacja Andrastentu XL u osoby dorosłej z zaawansowaną niewydolnością serca w przebiegu koarktacji aorty

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Abstract

We report the case of a 49 year-old-man with congenital coarctation of the aorta (CoA), admitted in a critical clinical condition due to advanced secondary cardiomyopathy and chronic heart failure. An Andrastent XL was implanted successfully in the CoA. The procedure resulted in an almost completely resolved CoA and prompt clinical improvement in the patient.

Key words: coarctation of the aorta, heart failure, stent implantation, Andrastent XL

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INTRODUCTION

Patients with congenital coarctation of the aorta (CoA) usually die due to left ventricular failure caused by a sudden collapse of myocardial function [1]. Surgical treatment of CoA has been performed for many years with good results, although the risk of surgery is much higher in adults than in children [2]. Transcatheter stent implantation seems to be a valuable alternative to surgical treatment.

We describe the case of a 49 year-old man with severe CoA, whose clinical condition at hospital admission was critical, with signs of overt and very advanced heart failure (HF). The patient had not been previously treated, as he had refused to give consent to any invasive procedure. The use of an expandable aortic stent effectively widened the narrowed

aorta, and proved to be a life-saving procedure in this clinical setting.

CASE REPORT

A 49 year-old man was admitted to the intensive care unit (ICU) of our centre with end-stage chronic HF caused by secondary cardiomyopathy due to the presence of CoA. Four years previously, he had been diagnosed with congenital CoA. Computer tomography performed at that time revealed tight CoA in a typical location (Fig. 1) with a short section of hypoplastic vessel below the stenosis. The patient had been offered two therapeutic options — a cardiac surgical procedure or a transcatheter expansion of CoA using an expandable aortic stent. The patient was then in NYHA class II and his left

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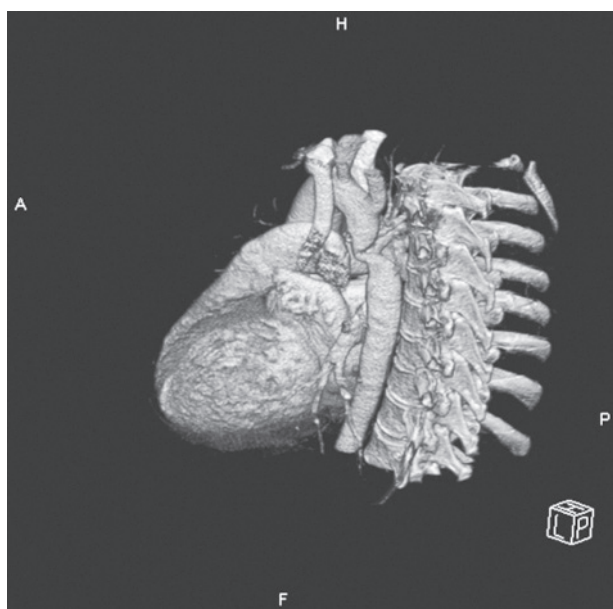


Figure 1. Computer tomography scan showing tight coarctation of the aorta in the typical location

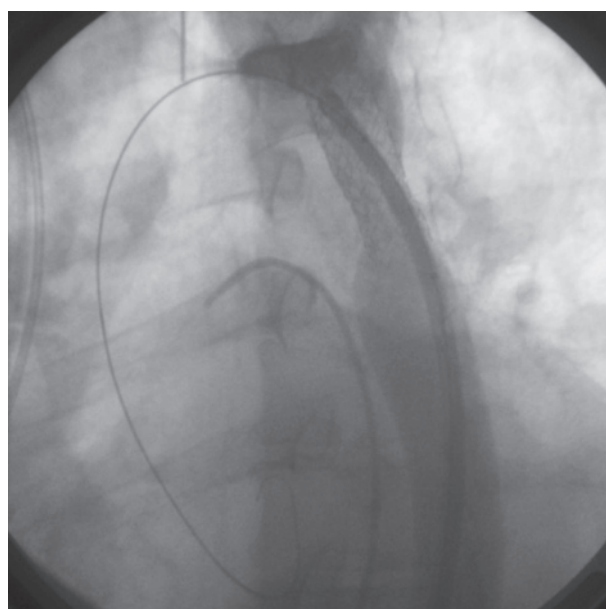


Figure 2. Control aortography (manual contrast injection) in LAO view after stent implantation

ventricular ejection fraction was good. Unfortunately, at this time he would not give his consent to any form of invasive treatment.

Since then, the patient's clinical condition had gradually deteriorated. From February 2011 onwards, the patient was repeatedly hospitalised due to rapidly increasing symptoms of HF, but he still refused invasive treatment. In July 2011, he was referred to our centre in a critical condition.

On admission, the patient presented with NYHA class IV, with evidence of active sepsis and deep purulent ulcerations of the left lower limb, associated with massive ascites and pleural effusions. His left ventricular ejection fraction was only 15%. Physical examination revealed typical signs of end-stage chronic HF, very severe dyspnoea and lack of femoral pulses. Oxygen saturation was 88% and the patient was administered oxygen via a face mask. At this stage, the patient finally gave his consent to invasive treatment, in the form either of a cardiac surgical procedure or an aortic stent implantation. Shortly after his admission to the ICU, he suffered sudden cardiac arrest due to ventricular fibrillation. Resuscitation procedures were successful and sinus rhythm was restored after 20 min. However, the patient required artificial ventilation with 100% oxygen and very high infusion doses of adrenaline (0.5–0.8 mcg/kg/min) to provide satisfactory blood pressure and efficient cardiac output.

Another three successfully treated incidents of cardiac arrest occurred. Thoracic ultrasound revealed massive pleural effusions and 2,000 mL of clear fluid was drained from the right pleural cavity, which resulted in a marked improvement in oxygenation. It was decided that the only therapeutic option

in such a critical condition was a percutaneous stent implantation into the CoA as a medical emergency.

The patient was transferred to the cath lab intubated, artificially ventilated, and on a high-dose adrenaline infusion. The time between the first incident of cardiac arrest and the start of the procedure was only 2 h. Cardiac catheterisation was performed via the left femoral artery. The CoA was crossed using a hydrophilic guidewire and multipurpose catheter. The pressure measured in the ascending aorta was 106/70 (82) mm Hg, while in the descending aorta it was 80/62 (69 mm Hg), meaning systolic gradient was 26 mm Hg. The aortographic image revealed a tight CoA in a typical location (1.5 cm below the left subclavian artery) and a short hypoplastic section of the aorta below the stenosis with a diameter of 7–8 mm. The diameter of the aorta was 20 mm above the CoA, and 25 mm at the level of the diaphragm. Extrastiff guidewire (0.035 × 260 cm long) was inserted into the ascending aorta. The short 6 F sheath was replaced with a 12 F long Mullins sheath. An Andrastent 48 XL (Andramed GmbH, Germany) was mounted on the balloon catheter Maxi LD 16 mm × 4 cm (Cordis Comp.). The proper location of the stent was confirmed angiographically and the stent was expanded (Fig. 2). The overall time of the procedure was 55 min, with the fluoroscopy lasting 4.5 min.

Immediately after the procedure, the systolic gradient through the CoA had diminished to 4 mm Hg and a clear pulse had appeared on the femoral arteries. A dramatic improvement in the patient's general condition was also noted. Catecholamine doses were gradually reduced. Over the subsequent days of treatment in the ICU, the left pleural cavity

and the abdominal cavity were drained with resulting volumes of 1,200 mL and 5,000 mL, respectively. The patient was gradually weaned off the ventilator and was extubated 72 h after undergoing the procedure.

In his subsequent time in ICU, the patient presented very high levels of procalcitonin and signs of sepsis. Blood cultures revealed the presence of *Meticillin-Susceptible Staphylococcus Aureus*. He was therefore given high-dose intravenous penicillin, which yielded a gradual improvement. Heparin-induced thrombocytopenia was also suspected (due to the presence of characteristic skin lesions and low platelet count). When this was definitely confirmed via laboratory tests, low-molecular weight heparin was immediately converted to subcutaneous fondaparinux (Arixtra, GlaxoSmithKline), with a rapid resolution of skin lesions and an improvement in the patient's general condition. After 12 days of ICU treatment, the patient was transferred to the general cardiology ward in a good and stable condition. He remained in hospital for the next nine days, mainly due to the need for surgical management of a deep wound to the lower leg and the necessity of taking high doses of intravenous antibiotics. Eventually, he was discharged home after 21 days of treatment and currently remains in a NYHA II/III functional class.

DISCUSSION

In the natural history of CoA, the appearance of HF has two distinct peaks. The first peak relates to about 5% of neonates with CoA, and is often associated with the presence of accompanying heart defects. In most adult patients with congenital CoA however, the heart defect usually remains asymptomatic until the patient is in his or her thirties, with a second peak therefore occurring only after the age of 40. This is usually associated with concomitant aortic stenosis, coronary artery disease or cardiomyopathy, which increases the existing arterial hypertension [1].

Our previous studies, as well as multicentre studies by other researchers, have shown very encouraging results of stent implantation in CoA over both short- and long-term observations [3, 4]. It is worth mentioning that in the reports of interventional treatment of CoA, the procedures have always been performed by interventional cardiologists (not by interventional radiologists, as proposed by the Polish health-care payer).

In the presented case, we performed a procedure of aortic stenting without balloon catheter predilatation, as previously proposed by Bulbul et al. [5]. This was because the extremely serious condition of our patient before and during the procedure meant that we needed to keep intervention time to a minimum. This also reduced the possibility of stent migration. We applied a new type of cobalt-chromium balloon-

-expandable stent (namely Andrastent XL). We have found this type of stent is very useful in widening congenital and acquired stenoses of large vessels [6, 7].

We believe two important conclusions may be drawn from this case report.

Firstly, that CoA should be treated as soon as a severe form of this defect is established. Performing this procedure a few years earlier would have avoided the disastrous consequences of CoA.

Secondly, that it is never too late for this procedure, as our patient had no other treatment option on admission to our centre. Similar observations were made by Alcibar et al. [8]. The fundamental mechanism that contributed to the improvement following the procedure was a reduction of afterload, when the obstruction of bloodflow (caused by the CoA) was suddenly removed. These actions were associated with timely and effective procedures performed in the setting of the intensive care unit by a multidisciplinary team, with the profound involvement of anaesthesiologists and intensivists.

We believe that transcatheter aortic stent implantation in an adult with a severe CoA is the treatment of choice, taking into account the critical symptoms of HF and the extremely serious clinical condition of the patient.

Conflict of interest: none declared

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