Cor et Vasa

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/crvasa

Kasuistika | Case report

Successful endovascular treatment of type B aortic dissection in a 15-year-old child

Ivo Petrov[a], Anna Kaneva-Nencheva[b], Elisaveta Levunlieva[b], Kamelia Genova[b], Iskren Garvanski[a], Georgi Konstantinov[c], Gloria Adam[d]

[a] *Cardiology Department, "City Clinic" Sofia University Hospital, Bulgaria*
[b] *Pediatric Cardiology Department, National Heart Hospital, Sofia, Bulgaria*
[c] *Vascular Surgery, National Heart Hospital, Sofia, Bulgaria*
[d] *Diagnostic Imaging Department, "City Clinic" Sofia University Hospital, Bulgaria*

ARTICLE INFO

Article history:
Received: 22. 3. 2016
Accepted: 17. 4. 2016
Available online: 24. 5. 2016

SOUHRN

Disekce aorty je u dětské populace a u populace mladých dospělých vzácná [1]. V tomto článku popisujeme neobvyklý případ 15letého chlapce s náhlým nástupem bolesti na hrudi, bolesti zad spolu s necitlivostí levé dolní končetiny. Vyšetření výpočetní tomografií hrudníku a oblasti břicha odhalilo disekci aorty typu B Stanford (typu III klasifikace DeBakey) s výslednou subokluzivní stenózou horní mezenterické tepny (vyvolávající kritickou dysfunkci střev a ileus) a úplným uzávěrem levé iliacké a renální tepny. Pacient byl přijat v kritickém stavu 48 hodin po nástupu bolesti na hrudi. Po několika následných endovaskulárních výkonech bylo dosaženo revaskularizace a pacient byl propuštěn z nemocnice na základě příznivého klinického výsledku s úplnou obnovou průtoku krve tepnami cílových orgánů. Při první intervenci byly do horní mezenterické tepny a břišní tepny implantovány stenty, a byla provedena balonková dilatace levé iliacké tepny. Pro rezistentní renovaskulární hypertenzi byl o 20 dní později implantován stent do úplně uzavřené levé renální tepny; následně bylo dosaženo optimální úpravy krevního tlaku. O 33 dní později byl implantován stentgraft s cílem uzavřít primární trhlinu v hrudní tepně. Genetické vyšetření odebraných vzorků prokázalo mutaci v genu *ACTA2*. Tento případ je výjimečný tím, že se jednalo o život ohrožující postižení aorty současně s vaskulopatií, řešené poprvé endovaskulárně u dětského pacienta, a upozorňuje na možnost endovaskulární léčby u dětské populace. V článku popisujeme použití zobrazovacích metod, samotnou léčbu tohoto závažného onemocnění i její úspěšný výsledek.

ABSTRACT

Aortic dissection is a rare condition in the pediatric and young adult population [1]. Here, we present an unusual case of a 15-year-old male patient with sudden onset of chest and back pain and numbness in the left leg. Chest and abdominal CT revealed Stanford type B (DeBakey type III) aortic dissection, leading to subocclusive stenosis of the superior mesenteric artery (causing critical intestinal dysfunction with ileus) and total occlusion of left iliac and left renal arteries. The child was admitted 48 hours after chest pain onset in critical clinical condition. Revascularization was achieved by several consecutive endovascular procedures, and the patient was discharged after favorable clinical evolution with full restoration of flow in the target organ arteries. The first interventional treatment included stenting of the superior mesenteric artery, stenting of the abdominal aorta, and balloon dilatation of the left iliac artery. Due to resistant renovascular

Adresa: Assoc. Prof. Ivo Petrov, MD, PhD, "City Clinic" Cardiology Center University Hospital, 127 Okolovrasten pat Street, 1407 Sofia, Bulgaria, e-mail: petrovivo@hotmail.com
DOI: 10.1016/j.crvasa.2016.04.006

Please cite this article as: I. Petrov, et al., Successful endovascular treatment of type B aortic dissection in a 15-year-old child, Cor et Vasa 59 (2017) e163–e170 as published in the online version of *Cor et Vasa* available at http://www.sciencedirect.com/science/article/pii/S0010865016300248

hypertension, stenting of the left renal artery, which was occluded, was conducted 20 days later, leading to optimal blood pressure control. Thirty-three days following the initial procedure, an endovascular endograft prosthesis implantation was performed to close the primary tear in the thoracic aorta. Genetic samples revealed *ACTA2* mutation. This case is extraordinary because of its combined life-threatening aortic and vessel pathology, treated for the first time with endovascular means in a child, and highlights the feasibility of endovascular treatment in the pediatric population. We discuss the imaging, management, and successful outcome of this severe condition.

Introduction

Acute aortic dissection (AD) is an emergency and life-threatening condition that is extremely rare among the population under 21 years of age. A past analysis shows that an average of 0.67% (range, 0.37–3.5%) of all aortic dissections occur in young patients, predominantly in males. Because of its rare occurrence, data regarding patient characteristics and management of such lesions are sparse and limited to small populations and few case reports [1–3]. The most common predisposing factors for the development of AD are congenital cardiovascular diseases, connective tissue disorders, trauma, chronic hypertension, drug abuse, strenuous exercise, and a family history of aortic aneurysms and dissections [4–8]. Marfan syndrome is considered a major risk factor among young patients [9,10]. Prior research has also been focused on certain forms of structural congenital abnormalities, such as bicuspid aortic valve, coarctation of the aorta, and patent ductus arteriosus (PDA) [11,12]. The most specific and frequent presentation of AD is sudden onset of severe chest pain, irradiating to the back and migrating along the aorta. Other signs and symptoms include uni- or bilateral peripheral pulse deficit, ischemic limb, ischemic bowel, focal neurological deficits, hypotension, shock, and heart failure [13,14]. CT scan and MRI modalities have a high accuracy, specificity, and sensitivity for the detection of all forms of dissection. They can assess the tear localization, side branch involvement, and complications [15–18]. No case of endovascular treatment of AD in a child has been published to date.

Case report

A 15-year-old child presented at the Emergency Department (ED) of a municipal hospital with sudden onset of back and chest pain and numbness in the left leg. On first examination, the patient was well built, with normal skin texture and no marfanoid features. He was hemodynamically stable, with blood pressure of 130/60 and no difference between the two arms. His heart rate pulse was 70 bpm and bilaterally symmetric. The peripheral pulsations of the lower limbs were weak and almost missing on the left femoral and popliteal artery. ECG was unremarkable. There were no heart murmurs on auscultation. The respiratory system was normal, with clear breath sounds. The patient's abdomen was soft but with reduced peristaltic sounds. The patient denied any allergies or prior drug or alcohol use. His past medical history was remarkable, with a surgical correction of PDA at the age of 6 months. He had no family history of connective tissue disorders. During the past month before the hospitalization, he had begun to train more intensely, including weight lifting. With the suspicion of acute aortic syndrome, a CT scan was performed and revealed AD of DeBakey Type III (Stanford B) with severe compression of the true lumen and subsequent occlusion of the superior mesenteric artery, left renal artery, and left iliac artery. The origin of the intimal tear was detected just distal to the left subclavian artery. The dissection reached the aortic bifurcation and pervaded the left common iliac artery. The celiac trunk originated from the false lumen as well as the superior mesenteric artery and left renal artery. The right renal artery originated from the true lumen (Figs. 1 and 2).

The patient was transferred to a specialized cardiovascular hospital. At the time of hospitalization he had symptoms of critical end-organ ischemia, leading to ileus (due to an occlusion of the superior mesenteric artery), severe limb ischemia and necrosis of the left lower limb (with peak creatine phosphokinase [CPK] value of 20,000 IU/L), and occlusion of the left renal artery. A multidisciplinary heart and vascular team discussion was held. Due to the underlying life-threatening emergency condition, conservative medical treatment was not considered as an option. Radical surgical treatment was considered too risky. A multi-stage endovascular treatment strategy was adopted.

The first stage included recanalization and restoration of flow into the branches of the abdominal aorta and centralization of the blood flow in the true lumen of the abdominal aorta. After 6Fr radial vascular access recanalization and stenting of the superior mesenteric artery with a 6 × 20mm Valeo vascular stent (Bard), recanalization (fenestration with a TrailBlazer support catheter [Co-

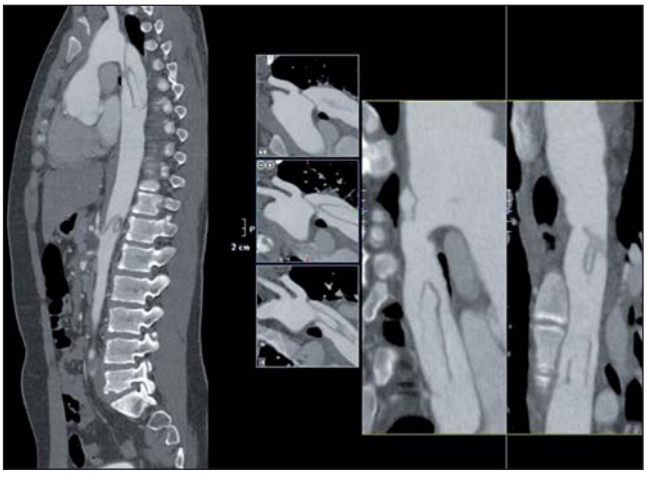

Fig. 1 – Initial CT. The origin of the intimal tear just after the left subclavian artery. The tear reached the aortic bifurcation and pervaded the left common iliac artery.

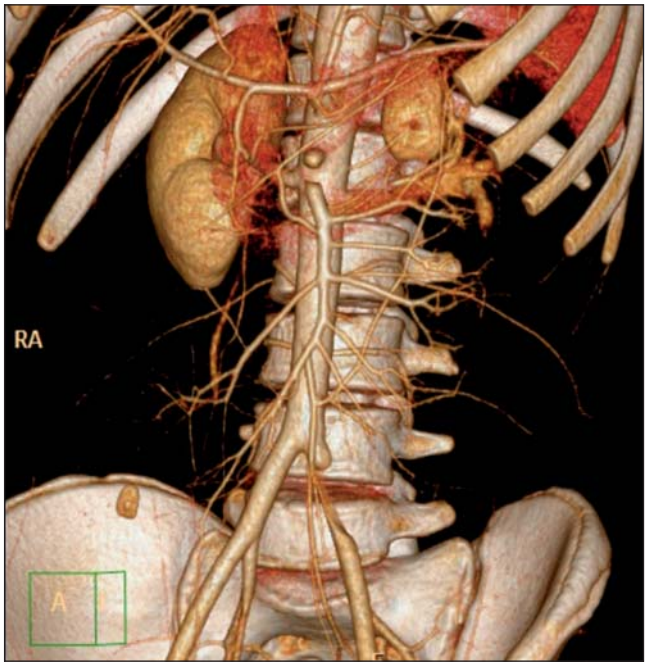

Fig. 2 – Occlusion of the superior mesenteric artery, left iliac artery, and left renal artery.

vidien] and 0.035" hydrophilic straight Terumo Radiofocus wire) and optimal balloon dilatation (Admiral 10.0/60 mm) of the left iliac artery were performed. During this first emergent procedure with limited right femoral surgical approach, stenting of the extremely compressed part of the abdominal aorta with a 36 × 186mm Zenith dissection stent (Cook Medical) was undertaken. This first procedure restored normal blood flow in the vessels compressed by the false lumen, leading to the restoration of full flow and end-organ perfusion. These maneuvers led to an excellent clinical result: immediate improvement in the patient's condition with restored bowel movement and blood flow to the lower limbs (Figs. 3A and 3B).

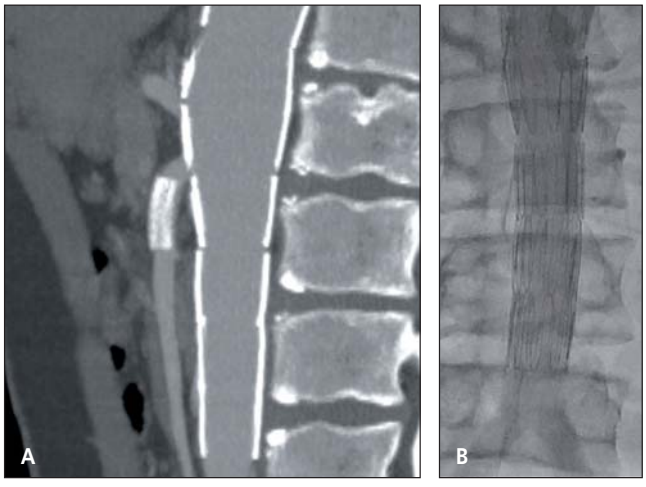

Fig. 3 – (A) Final result after the first stage of endovascular treatment. Stented abdominal aorta and superior mesenteric artery with restored normal flow. (B) Final result after the first stage of endovascular treatment. Zenith dissection stent well apposed, full resolution of the false lumen compression.

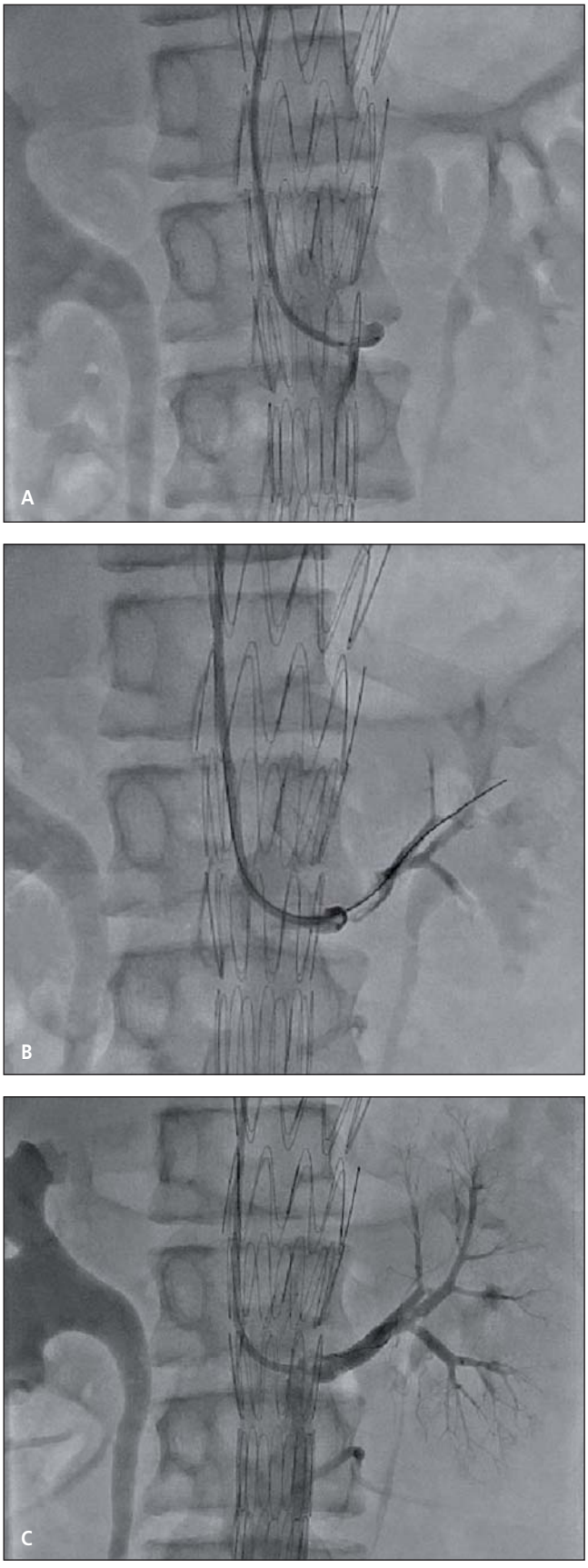

Fig. 4 – (A) Guiding catheter cannulating the occluded left renal artery through the stent struts. (B) Recanalization of the left renal artery. (C) Final result after recanalization and stenting of the left renal artery.

During the postprocedural period, acute renal failure with tendency for oliguria, elevated creatinine, and resistant hypertension were noted. Serum renin levels up to 398 ng/l were recorded. Because of these findings, on the 20th postprocedural day, recanalization and stenting of the left renal artery were carried out. The procedure was performed by right radial approach using a 6Fr JR guiding catheter (Launcher, Medtronic) placed across the struts of the previously implanted Zenith Dissection stent. After recanalization with a hydrophilic Whisper 0.014" guide wire (Abbott Vascular) supported with a low-profile Sprinter 1.5/10mm balloon (Medtronic), predilatation was performed with a Sprinter 3.5 × 20mm balloon, and a Dynamic 5.0 × 19mm renal stent (Biotronik) was implanted. This procedure restored the blood flow to the left kidney and in the following days led to the normalization of blood pressure and serum renin levels (Figs. 4A–4C).

During this procedure, diagnostic aortography revealed further expansion of the proximal false lumen. This finding prompted us to decide on a primary entry point for endovascular closure. This third stage of the endovascular treatment was performed 33 days after the first procedure. The procedure was carried out under general anesthesia with a limited left femoral surgical approach and bilateral radial approaches (the right one used for achieving diagnostic angiography during the implantation and the left one to place a pigtail catheter in the ostium of the left subclavian artery [LSA] as a target to be covered with the stent-graft during the implantation). Pigtail catheters were positioned, the first in the aortic arch for diagnostic angiography purposes and the second in the left subclavian ostium to mark the target zone to be covered (the left subclavian artery was covered intentionally after evaluation of the size of the two vertebral arteries and the potentially good collateral flow for the left arm) with surgical back-up for potential immediate carotid-to-subclavian bypass. After lowering the blood pressure to 80/50 mmHg, implantation of a Zenith Alpha stent-graft (Cook Medical) over a stiff Lunderquist wire (Cook Medical) was performed, followed by balloon postdilatation to ensure that complete isolation of the entry of the dissection would be achieved (Figs. 5A–5C).

After stent-graft implantation, normal blood flow in the carotid arteries and optimal collateral flow in the LSA were observed, with 40-mmHg difference between the right and left arms and no need for further LSA revascularization procedures. The left subclavian artery was intentionally covered by the endograft and had collateral filling through a "vertebral steal", and the BP within it was 53/42 mmHg compared to a BP in the right radial artery of 84/56 mmHg.

In the days after this procedure, due to ongoing hypoxemia, a chest X-ray and CT scan were performed and revealed compression of the left main bronchus by the thoracic stent-graft. A series of bronchoscopies showed that the left main bronchus was patent only when lifting the left shoulder and putting the patient in the supine position. In the following days, antibiotic treatment and mechanical ventilation led to stabilization of the patient's status. After mobilization and rehabilitation, the left bronchus was shown to be patent without dependence on body position. On day 59 after hospitalization, the patient was discharged in stable condition: normal end-organ perfusion, optimal blood pressure control with only one antihypertensive drug, beta-blocker nebivolol (5 mg), normal kidney function, and almost completely restored movement of the left leg with mild left ankle paresis. CT scan at discharge showed preserved results from the endovascular treatment (centralized aortic blood flow, normal visceral arteries and iliac flow, and isolated false lumen with no filling at the thoracic level) and revealed that the left bronchus was patent despite the minor external compression. CT at 1- and 9-month (Fig. 5D) follow-up revealed homogeneous contrast of the thoracic and abdominal aorta, with preserved blood flow in the previously affected arteries. The left kidney was relatively smaller, but renin levels were normal, and the patient had no hypertension and/or creatinine level elevation. The left bronchus was compressed by the aortic arch but was still completely patent from a functional point of view. Our young patient had no major complaints, had effectively returned to school, and achieved full functional recovery in a short time.

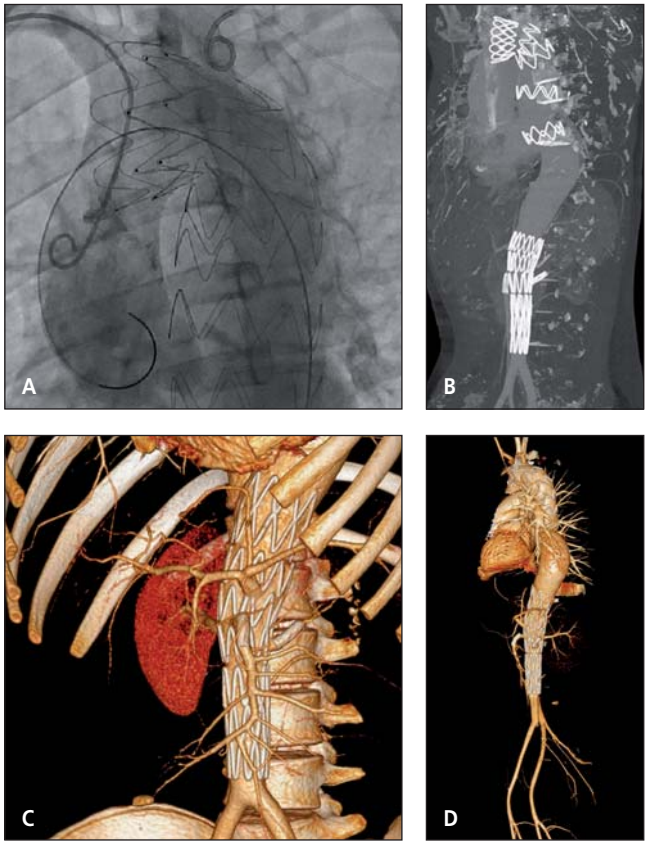

Fig. 5 – (A) Final result after stent-graft implantation. Proximal "free-flo" part positioning into the arch with wide coverage of the left subclavian artery ostium (pigtail catheter positioned by purpose in the LSA to mark its ostium), thus providing sufficient proximal landing zone for better isolation of the proximal entry tear. (B) Final result after stent-graft implantation. Restoration of normal aortic flow and immediate isolation of the proximal false lumen. (C) MSCTA control of the final result after stent-graft implantation. Restored flow into the aorta, normalized visceral and iliac arteries flow. (D) Multi-slice CT at 9-month follow-up showing full restoration of flow into the end organs and full false lumen isolation. All stents implanted are patent and with optimal apposition.

At the 6-month follow-up, genetic analysis revealed *ACTA2* gene mutation related to connective tissue disorders.

Discussion

AD is very rare in children. It has been reported to occur at a rate of less than 3.5% among young people. As a result of its low occurrence, the literature includes insufficient data and cases of AD in this population. It is a potentially dangerous medical condition that must be diagnosed and treated without delay [1–3,19]. The primary event in AD is a tear in the aortic intima, which is preceded by degeneration or cystic necrosis of the aortic media. Blood passes through the tear, separating the intima from the surrounding media and/or adventitia and creating a false lumen. The site of a previous PDA surgical correction can be predilective for this tear to occur [20,21]. Propagation of the dissection can proceed both distal and proximal to the initial tear, involving side branches and in this way causing specific clinical manifestations and complications. AD can be acute or chronic. Acute dissection is defined by a symptom duration of up to 2 weeks, whereas chronic dissections have symptom duration longer than 2 weeks [22]. The mortality rate of untreated AD is 50% in the first 48 h, 80% at the end of the first month, and less than 10% up to the end of the year. AD is among the most common causes of cardiovascular death in the pediatric population, with a mortality of 0.035% in patients under 19 years of age [1,2,9,19]. Fatal complications of AD include rupture of the thoracic aorta, malperfusion syndromes, tamponade, aortic valve insufficiency, and stroke [14,23].

The most important risk factors among young patients for developing AD are connective tissue disorders, congenital cardiovascular disease, trauma, drug abuse, weight lifting, and hypertension [4–8]. AD has been primarily reported in patients with Marfan and type IV Ehlers-Danlos syndromes or developmental anomalies of the aorta such as aortic coarctation, aortic valvular stenosis, unicuspid/bicuspid aortic valve, and PDA [11,12]. Intrinsic anomalies and secondary hemodynamic changes may lead to degeneration of the aortic wall. History of cardiovascular surgery and iatrogenic injury from intra-aortic catheterization also might cause AD [13]. However, AD can also occur in children and adolescents without any predisposing factor.

Due to the rarity of AD in young adults, a high suspicion is required to reach the diagnosis in a timely manner. It should be considered in patients who complain of sudden and severe chest pain in association with risk factors.

AD may present with diverse clinical manifestations in all age groups. In this case, the 15-year-old boy presented with abrupt and migrating chest and back pain and leg numbness. His distinctive symptoms and history of congenital heart disease suggested AD as a potential diagnosis. In the preceding days he had increased his training, and therefore strenuous physical activity should be considered as a precipitating factor for the acute onset of AD. Evaluation of the affected aorta with CT imaging confirmed the pathological finding of AD with an intimal tear directly after the left subclavian artery and a second distal dissection at the level of T11 that reached the aortic bifurcation and pervaded the left common iliac artery. CT is the first-choice diagnostic modality for AD. Furthermore, CT angiography combined with three-dimensional rendering is the preferred tool to plan endovascular interventional treatment. This modality depicts the tear of the aortic wall, the presence or absence of mural thrombus or calcifications, the exact intraluminal measurements of the diameters of the aortic lesion and the aortic segments proximal and distal to it, and also the anatomy of the abdominal aorta and the size and tortuosity of femoral and iliac vessels [15–18].

Given the rapid decline in survival with each passing hour, the prompt diagnosis and necessary management of AD are crucial. The first goal in medical treatment of an AD is to maintain the blood pressure and heart rate [22]. Dissections involving the beginning portion of the aorta (type A) are treated with emergency open surgery; those involving the descending aorta (type B) are usually treated with medications or endovascular stenting (EVS) when impending rupture, rapid progression of symptoms, or risk of abnormal perfusion of vital organs is present. In adults, endovascular stent placement for type B AD seems to be the most appropriate alternative in stable patients, whereas hemodynamically unstable patients should be managed with conventional surgery [22,24–27]. However, there is no consensus in the literature regarding the optimal management of AD in children. Treatment and follow-up of the condition should be individualized and discussed by a multidisciplinary team including various specialists to assess the risk/benefit ratio of every method available.

In the present case, multistage endovascular management was conducted with stenting of the superior mesenteric artery, dilatation and stenting of the left iliac artery, stenting of the abdominal aorta, fenestration and balloon angioplasty of the left iliac artery, and implantation of a stent-graft in the thoracic aorta. The interventional approach at every step of the treatment was based on clinical signs, symptoms, and the specific anatomical condition and hemodynamics of the dissection. The procedures had no significant complications. Effective isolation of the false lumen of the aorta and restoration of flow in the true lumen and branches were achieved. The revascularization of the affected branch vessels prevented potentially fatal ischemia and necrosis of the underlying anatomical structures. Relief of lower extremity ischemia was apparent, with return of pulses and sensation and resolution of pain. The patient's gradual improvement and full restoration reveal the success of the performed manipulations. EVS and balloon dilatation resulted in a safe and effective percutaneous method for managing peripheral ischemic complications of this life-threatening AD.

The value of endovascular treatment for AD is well established [23] but not in a pediatric population up to now. The durability of such an approach is of course questionable because of the potential growth of the child's body and vessels. For this reason, we calculated a significant oversizing (more than 20%) of the aortic open stent and stent-graft during the implantation. The

durability of the stent-graft treatment in young patients is also a matter of discussion because of possible material fatigue and late complications. Despite these concerns, our experience has shown more than 15 years of favorable outcome in our first patients treated with an endovascular aortic repair procedure in the early 2000s. Additionally, we published the case of a young woman affected by Turner syndrome resulting in aortic aneurysm treated with stent-graft implantation, who has had an absolutely uneventful follow-up for more than 10 years thus far [28].

The *ACTA2* gene mutation that was present in the treated child is associated with a variety of vascular diseases, including thoracic aortic aneurysms and dissections [29]. As far as we know, there are no such cases of spontaneously occurring AD of Stanford type B in a child that were successfully treated by endovascular means. The literature includes a few cases of young patients who have recovered after endovascular treatment, but they involved traumatically caused iatrogenic AD [30–35]. In another reported case, of a 10-year-old child with AD after balloon angioplasty of a recoarctation, distal percutaneous membrane fenestration was performed [36]. There are a few cases describing the effective treatment of children with AD, but they were all treated surgically and not by endovascular approach [37–38]. Moreover, there are cases of AD described in children caused by various factors that were followed by a tragic outcome, independent of the measures taken (conservative treatment or surgical approach) [39,40].

Conclusion

AD is a life-threatening condition, but when diagnosed early it can be treated with success. It is rare among children, but because of its emergency status, it should not be underestimated. To our knowledge, this is the first reported case of spontaneously occurring type B AD in a child treated with this type of approach and ending with success. The results of these endovascular techniques are very promising. Long-term follow-up and research into their effectiveness are necessary. Continued studies of this rare disease are required to improve understanding of AD in the young with the aim of improving the effectiveness of treatment and decreasing mortality. Endovascular treatment likely provides an alternative for the management of AD and its complications in children.

Conflict of interest
None declared.

Funding body
None.

Ethical statement
I declare, on behalf of all authors that the research was conducted according to Declaration of Helsinki.

Informed consent
I declare that informed consent requirements do not apply to this manuscript.

References

[1] P. Shamszad, J.N. Barnes, S.A. Morris, Aortic dissection in hospitalized children and young adults: a multiinstitutional study, Congenital Heart Disease 9 (2014) 54–62.
[2] C.R. Fikar, R. Fikar, Aortic dissection in childhood and adolescence: an analysis of occurrence over a 10-year interval in New York State, Clinical Cardiology 32 (2009) E23–E26.
[3] A.B. Landman, S.S. Torbati, Chronic aortic dissection in a young adult, Journal of Emergency Medicine 42 (2012) e105–e108.
[4] C.R. Fikar, S. Koch, Etiologic factors of acute aortic dissection in children and young adults, Clinical Pediatrics 39 (2000) 71–80.
[5] B.A. Vogt, P.E. Birk, V. Panzarino, et al., Aortic dissection in young patients with chronic hypertension, American Journal of Kidney Diseases 33 (1999) 374–378.
[6] I.S. Hatzaras, J.E. Bible, G.J. Koullias, et al., Role of exertion or emotion as inciting events for acute aortic dissection, American Journal of Cardiology 100 (2007) 1470–1472.
[7] K. Uchida, K. Imoto, H. Yanagi, K. Date, Acute aortic dissection occurring during the butterfly stroke in a 12-year-old boy, Interactive Cardiovascular and Thoracic Surgery 9 (2009) 366–367.
[8] C.J. Hogan, An aortic dissection in a young weightlifter with non-Marfan fibrillinopathy, Journal of Emergency Medicine 22 (2005) 304–305.
[9] J.L. Januzzi, E.M. Isselbacher, R. Fattori, et al., Characterizing the young patient with aortic dissection: results from the International Registry of Aortic Dissection (IRAD), Journal of the American College of Cardiology 43 (2004) 665–669.
[10] D.P. Judge, H.C. Dietz, Marfan's syndrome, Lancet 366 (2005) 1965–1976.
[11] C.R. Fikar, J.A. Amrhein, J.P. Harris, E.R. Lewis, Dissecting aortic aneurysm in childhood and adolescence: case report and literature review, Clinical Pediatrics 20 (1981) 578–583.
[12] D.J. Schneider, J.W. Moore, Patent ductus arteriosus, Circulation 114 (2006) 1873–1882.
[13] M. Emmel, N. Sreeram, K. Brockmeier, Stenting of the aortic arch as an emergency palliation of aortic dissection after cardiac surgery in an infant, Images in Paediatric Cardiology 7 (2005) 8–11.
[14] P.C. Spittell, J.A. Spittell Jr., J.W. Joyce, et al., Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980 through 1990), Mayo Clinic Proceedings 68 (1993) 642–651.
[15] S. Willoteaux, C. Lions, V. Gaxotte, et al., Imaging of aortic dissection by helical computed tomography (CT), European Radiology 14 (2004) 1999–2008.
[16] T. Shiga, Z. Wajima, C.C. Apfel, et al., Diagnostic accuracy of transesophageal echocardiography, helical computed tomography, and magnetic resonance imaging for suspected thoracic aortic dissection: systematic review and meta-analysis, Archives of Internal Medicine 166 (2006) 1350–1356.
[17] M.J. Siegel, Multiplanar and three-dimensional multi-detector row CT of thoracic vessels and airways in the pediatric population, Radiology 229 (2003) 641–650.
[18] G. Garzón, M. Fernández-Velilla, M. Martí, et al., Endovascular stent-graft treatment of thoracic aortic disease, Radiographics 25 (2005) S229–S244.
[19] M.C. Fishbein, Cardiac disease and risk of sudden death in the young: the burden of the phenomenon, Cardiovascular Pathology 19 (2010) 326–328.
[20] I. Mészáros, J. Mórocz, J. Szlávi, et al., Epidemiology and clinicopathology of aortic dissection, Chest 117 (2000) 1271–1278.
[21] C.S. Roberts, W.C. Roberts, Aortic dissection with the entrance tear in the descending thoracic aorta. Analysis of 40 necropsy patients, Annals of Surgery 213 (1991) 356–368.
[22] I.A. Khan, C.K. Nair, Clinical, diagnostic, and management perspectives of aortic dissection, Chest 122 (2002) 311–328.
[23] M. Genoni, M. Paul, R. Tavakoli, et al., Predictors of complications in acute type B aortic dissection, European Journal of Cardio-Thoracic Surgery 22 (2002) 59–63.
[24] C.A. Nienaber, R. Fattori, G. Lund, et al., Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement, New England Journal of Medicine 340 (1999) 1539–1545.

[25] H. Rousseau, C. Dambrin, B. Marcheix, et al., Acute traumatic aortic rupture: a comparison of surgical and stent-graft repair, Journal of Thoracic and Cardiovascular Surgery 129 (2005) 1050–1055.
[26] S.M. Slonim, D.C. Miller, R.S. Mitchell, et al., Percutaneous balloon fenestration and stenting for life-threatening ischemic complications in patients with acute aortic dissection, Journal of Thoracic and Cardiovascular Surgery 117 (1999) 1118–1126.
[27] T.A. Resch, M. Delle, M. Falkenberg, et al., Remodeling of the thoracic aorta after stent grafting of type B dissection: a Swedish multicenter study, Journal of Cardiovascular Surgery 47 (5) (2006) 503–508.
[28] I. Petrov, M. Nedevska, N. Chilingirova, et al., Endovascular repair of dissecting thoracic aortic aneurysm in a patient with Turner syndrome, Journal of Endovascular Therapy 13 (2006) 693–696.
[29] D.M. Milewicz, J.R. Østergaard, L.M. Ala-Kokko, et al., De novo ACTA2 mutation causes a novel syndrome of multisystemic smooth muscle dysfunction, American Journal of Medical Genetics Part A 152A (2010) 2437–2443.
[30] K.O. Papazoglou, C.D. Karkos, T.E. Kalogirou, I.T. Giagtzidis, Endovascular management of lap belt-related abdominal aortic injury in a 9-year-old child, Annals of Vascular Surgery 29 (2015) 365.e11–365.e15.
[31] V. Andrey, V. Bettschart, N. Ducrey, et al., Traumatic abdominal aortic rupture treated by endovascular stent placement in an 11-year-old boy, Journal of Pediatric Surgery Case Reports 1 (2013) 56–59.
[32] R. Karmy-Jones, D. Teso, N. Jackson, et al., Endovascular approach to acute aortic trauma, World Journal of Radiology 1 (2009) 50–62.
[33] Z.L. Milas, R. Milner, E. Chaikoff, et al., Endograft stenting in the adolescent population for traumatic aortic injuries, Journal of Pediatric Surgery 41 (2006) e27–e30.
[34] B.H. Goldstein, R. Hirsch, M.E. Zussman, et al., Percutaneous balloon-expandable covered stent implantation for treatment of traumatic aortic injury in children and adolescents, American Journal of Cardiology 110 (2012) 11541–11545.
[35] K. Keyhani, A.L. Estrera, H.J. Safi, A. Azizzadeh, Endovascular repair of traumatic aortic injury in a pediatric patient, Journal of Vascular Surgery 50 (2009) 652–654.
[36] T. Abu-Tair, C. Martin, C. Kampmann, Acute aortic dissection after balloon angioplasty of a recoarctation and treatment by stenting and distal membrane fenestration in a child, Heart 97 (2011) 1699–1700.
[37] T. Walther, H. Kiefer, I. Dähnert, et al., Successful treatment for intraoperatively evolving acute aortic dissection in a neonate, Annals of Thoracic Surgery 76 (2003) 1286–1287.
[38] K.F. Deml, U.J. Schoepf, T. Henzler, Acute aortic dissection in a 9-year-old boy with chest pain, Journal of the American College of Cardiology 56 (2010) e49.
[39] E. Bossone, P. Masiello, A. Panza, et al., Acute aortic dissection in the young: clinical series, Minerva Chirurgica 62 (2007) 305–307.
[40] K.W. Ngan, C. Hsueh, H.C. Hsieh, S.H. Ueng, Aortic dissection in a young patient without any predisposing factors, Chang Gung Medical Journal 29 (2006) 419–423.