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Cardiovascular Surgery and Interventions

Interesting Image Open Access

Cardiac magnetic resonance image of late pulmonary artery aneurysm after total correction of tetralogy of Fallot

Mehmet Taşar, Nur Dikmen Yaman, Zeynep Eyileten, Adnan Uysalel

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Tetralogy of Fallot is the most common cyanotic heart defect. Total correction procedures are performed in most centers and pulmonary insufficiency can be problematic in long-term period.[1] Definite diagnosis is essential for the development of appropriate treatment. Cardiac magnetic resonance can provide comprehensive information about the nature of congenital heart defect in a safe fashion.[2] Herein, we present a 28-year-old female patient who underwent re-do surgery due to pulmonary homograft implantation for late pulmonary insufficiency with main pulmonary artery aneurysm detected by echocardiography and cardiac magnetic resonance during pregnancy 28 years later from total correction surgery of tetralogy of Fallot (Figures 1).

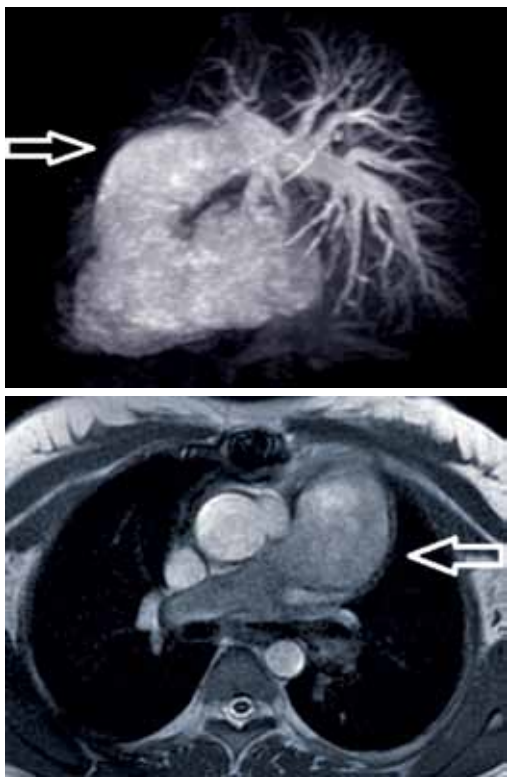

Figure 1. Arrows indicating an aneurysm of the main pulmonary artery.

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Cardiovascular Surgery and Interventions

Case Report Open Access

Prosthetic mitral valve dehiscence caused by infective endocarditis

Yüksel Dereli[1], Ömer Tanyeli[1], Halil İbrahim Erdoğan[2], Niyazi Görmüş[1]

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ABSTRACT

Despite therapeutic advances over the past few decades, prosthetic valve endocarditis remains a life-threatening condition and is associated with increased significant morbidity and mortality. Prosthetic valve dehiscence caused by endocarditis is one of the most serious complications of infective endocarditis. In this article, we present a case of acute subtotal dehiscence of prosthetic mitral valve caused by *staphylococcal* endocarditis. A 74-year-old female patient presented with fever and breathlessness. She underwent mechanical mitral valve replacement six years previously for mitral stenosis. Echocardiography confirmed subtotal dehiscence of prosthetic mitral valve. *Staphylococcus capitis* was detected in blood cultures. The patient was reoperated successfully.

Keywords: Dehiscence; infective endocarditis; prosthetic mitral valve.

Prosthetic valve endocarditis (PVE) is a life-threatening condition and has been reported to occur in 1 to 6% of patients.[1] Prosthetic valve dehiscence (PVD) caused by endocarditis is one of the most serious complications of infective endocarditis (IE). If early diagnosis and treatment are not performed, it may lead to acute decompensation, pulmonary edema, cardiogenic shock, and death, eventually. Herein, we present a case of acute subtotal dehiscence of prosthetic mitral valve after six years caused by staphylococcal endocarditis.

CASE REPORT

A 74-year-old female patient was admitted to our clinic with complaints of fever and shortness of breath. She underwent mitral valve replacement six years ago for mitral stenosis. The patient had atrial fibrillation. On physical examination, she had bilateral inspiratory rales, jugular venous distension, S3 gallop, and peripheral edema. Laboratory analysis revealed an increased white blood count (14,000 mm/L), erythrocyte sedimentation rate (71 mm/hr) and C-reactive protein (41 mg/L). The international normalized ratio (INR) was 3.42. The patient was then hospitalized and three-set blood cultures were drawn. the vegetation of the mitral prosthetic valve dehiscence, echocardiography was performed and identified patients with severe mitral regurgitation (Figure 1). Ampiric antibiotic therapy was initiated. *Staphylococcus capitis (S. capitis)* was detected in blood cultures and antibiotherapy was revised by the culture antibiogram. The patient underwent one week of antibiotherapy and infection parameters decreased. The patient was reoperated. During surgery, subtotal prosthetic mitral valve dehiscence was observed (Figure 2). Infected valve and annular vegetations were excised. Bioprosthetic mitral valve replacement was performed successfully.

DISCUSSION

Despite advances in medical treatment and surgical techniques, PVE carries a high mortality risk ranging from 20 to 80% of affected patients.[1] Early PVE occurs within the first year of surgery with a risk ratio of 1 to 3% and is frequently caused by *Staphylococcus epidermidis*. *Staphylococcal* endocarditis has a higher rate of morbidity and mortality than that caused by other microorganisms and surgery is usually required. Pathogens causing late PVE are similar to that of native valve endocarditis.[2] In our case, PVE developed six years after surgery and pathogenic microorganism was reported as *S. capitis*.

Staphylococcus capitis is a subtype of coagulase-negative staphylococci (CoNS) and it is

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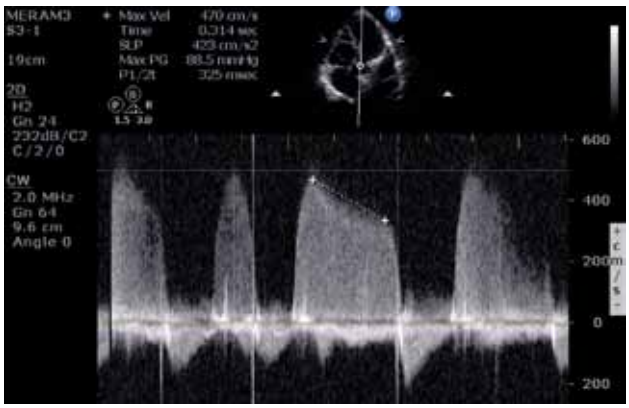

Figure 1. Preoperative echocardiography, showing prosthetic mitral valve dehiscence.

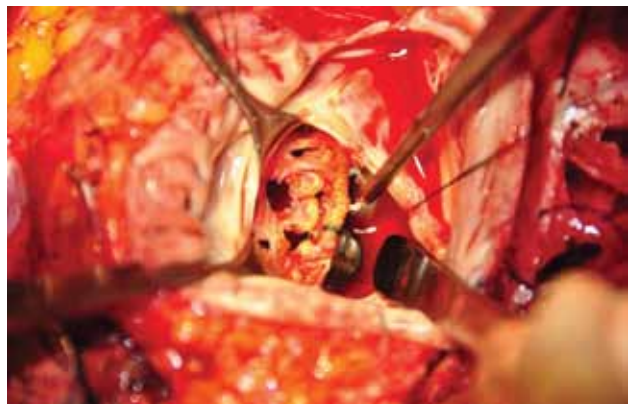

Figure 2. Intraoperative view showing subtotal prosthetic mitral valve dehiscence.

part of the normal flora of the skin of the scalp, face, ears and neck.[3] Rarely, it may present as a significant pathogen causing IE, PVE, and late-onset sepsis. In 2011, Takano et al.,[4] reported four cases of PVE caused by *S. capitis* which were identified at their hospital over the past two years. The pathogenesis of CoNS is mainly due to their ability to form biofilms on indwelling medical devices which confers tolerance to disinfectants during surgery.[5]

Furthermore, prosthetic valve dehiscence is a catastrophic complication of IE. Patients with PVD may have a stable hemodynamic profile or cardiogenic shock. Echocardiography is sufficient for diagnosis. As transthoracic echocardiography has low sensitivity, transesophageal echocardiography recommended for these patients. Recently, three-dimensional echocardiography is the favorite diagnostic method.[6] Treatment of PVD is usually surgical. Inadequate surgical debridement of infected material may result in recurrent PVE. Timing of surgery is of utmost importance for these patients. The success of surgery would increase by stabilizing the hemodynamic and laboratory parameters. In these patients, another important point is the selection of the type of prosthesis. However, Newton and Hunter reported that the choice of the prosthesis, either a bioprosthesis or mechanical valve, had no effect on the rates of recurrence in patients with PVE undergoing surgery.[7]

In conclusion, PVE is a rare cause of PVD in late stage. Early diagnosis and treatment is life-saving for this condition.

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Cardiovascular Surgery and Interventions

Case Report Open Access

Coexistence of pulmonary stenosis and pulmonary artery aneurysm in a young patient: a case report

Anıl Özen, Aytaç Çalışkan, Utku Ünal, Bahadır Aytekin, Erman Kiriş, Boğaçhan Akkaya, Levent Birincioğlu

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ABSTRACT

A 22-year-old female patient diagnosed with stenosis and regurgitation of the dome-shaped pulmonary valve presented with dyspnea. She also had significant dilation of the pulmonary artery which extended to the left pulmonary hilum. The patient was treated surgically with infundibular myectomy, enlargement of the transannular right ventricular outflow with a pericardial patch, pulmonary valve replacement with a 25 mm stentless bioprosthetic valve and plication of the main pulmonary artery and the left pulmonary artery. We believe that using her own tissue for the reconstruction of the pulmonary artery aneurysm and pulmonary valve replacement through a stentless bioprosthesis with a minimum gradient and full competence to prevent the dilation of the pulmonary artery may be an ideal treatment option.

Keywords: Pulmonary artery aneurysm; pulmonary artery plication; pulmonary valve replacement; stentless bioprosthesis.

Pulmonary artery aneurysm (PAA) is a rare disease which may be idiopathic or secondary to other pathologies, either, such as pulmonary valve stenosis, congenital cardiac anomalies associated with pulmonary hypertension, Behçet's disease, trauma and infections.[1,2] The natural history of the disease is not clearly understood and currently no definite guideline recommendations are available for the optimal treatment.

Post-stenotic dilatation frequently progresses to PAA. Herein, we discuss a female case of stenosis and regurgitation of the dome-shaped pulmonary valve with a significant aneurysm of the pulmonary artery.

CASE REPORT

We present a 22-year-old female patient diagnosed with PAA concomitant with pulmonary valvular stenosis. She suffered from dyspnea, palpitation, and fatigue. She had no past medical history of infection or trauma. Physical examination revealed a 2/6 systolic ejection murmur on the left sternal border. Posteroanterior chest X-ray revealed an enlarged pulmonary artery. Transthoracic echocardiography revealed pulmonary artery stenosis with a gradient of 85/55 mmHg. Pulmonary valve was thick and presented a dome-shape opening with a significant dilatation of the pulmonary artery which was 5.1 cm. Computed tomography of the pulmonary artery showed that the pulmonary artery diameter at the valvular level was 22 mm. The pulmonary artery had a fusiform aneurysm starting from the supravalvular level with a maximum diameter of 49 mm (Figure 1). In addition, other differential diagnoses such as Behçet's disease and other vasculitidies were excluded by the rheumatology team.

During surgery, median sternotomy was performed and pericardial patch was prepared and exposed to glutaraldehyde. The pulmonary aneurysm extended up to the left pulmonary hilum (Figure 2). A vertical incision from the right ventricular outflow tract (RVOT) through the pulmonary artery was made for the exposure of the pulmonary valve and the pulmonary trunk. The main pulmonary artery and the left pulmonary artery branches were placated circularly using 4/0 prolene sutures. The valve was excised and the pulmonary incision was extended to the ventricular infundibulum with septal myectomy. The RVOT was relieved and controlled with a 23-sized hegar buji. A 25 mm stentless

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Figure 1. Fusiform aneurysm of the pulmonary artery.

SOLO bioprosthetic valve (SORIN Biomedica Cardio S.r.l.; Saluggia, Italy) was replaced to the pulmonary annulus (Figure 3). The annular gap was completed using a transannular pericardial patch. Intraoperative echocardiography revealed a gradient at the RVOT. Therefore, we enlarged the RVOT by myectomy and closed the infundibular incision using the pericardial patch facilitating a dilatation of the RVOT (Figure 4). No inotropic support was required to wean off cardiopulmonary bypass. After a day of intensive care unit stay, she was discharged on the sixth postoperative day.

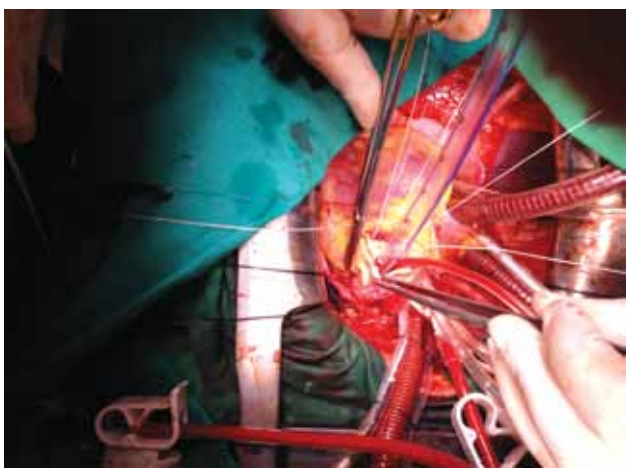

Figure 3. Implantation of the 25 mm stentless SOLO brand bioprosthetic valve to the pulmonary annulus.

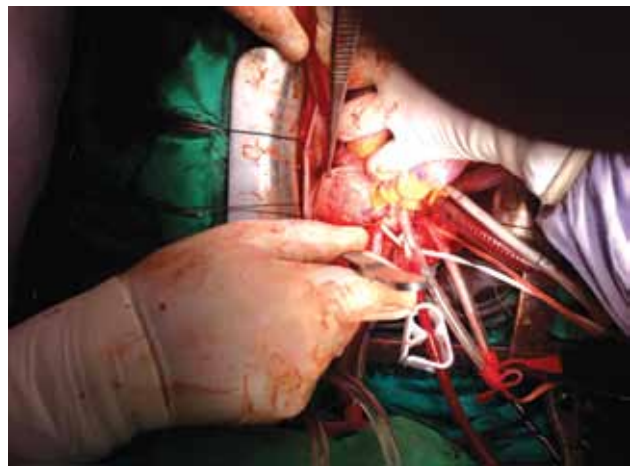

Figure 2. Intraoperative view of the pulmonary artery aneurysm.

DISCUSSION

Pulmonary artery aneurysm is a rare disease with common coexisting conditions. It can be secondary to other pathologies such as pulmonary valve stenosis, congenital cardiac anomalies associated with pulmonary hypertension, Behçet's disease, trauma and infections.[1,2] Pulmonary stenosis may cause

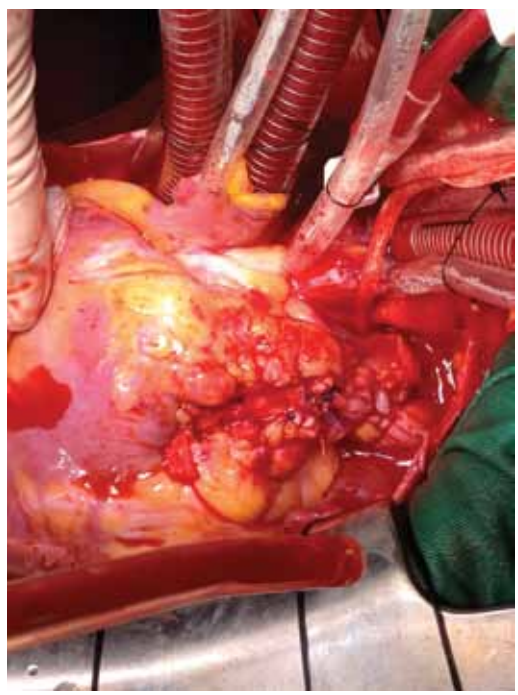

Figure 4. Closing the infundibular incision using the pericardial patch facilitating a dilatation of the right ventricular outflow tract.

excess volume or pressure load resulting in aneurysm formation.[3]

Although conservative management of large aneurysms has been reported,[4] documentation of rupture and dissection would make this strategy inadvisable for patients who would otherwise be considered suitable for surgery. Surgery should be considered in patients with dilatation of the pulmonary trunk and pulmonary arteries ≥5 cm, as in our case.

Valve reconstruction is the goal of all the surgical interventions, since restoration of anatomy and physiology employs the native tissue which allows for growth and potentially results in better long-term outcome. In case of repair failure or inconvenience, valve replacement would become inevitable.[5] In our case, ideal body weight of the patient, pulmonary artery aneurysm as a coexisting anomaly and no requirement for growth were particularly critical issues for the choice of valve replacement.

In patients with significant pulmonary valve stenosis and or insufficiency, reconstruction of the RVOT is performed in cases with congenital heart disease, when there is discontinuity between the right ventricle and the pulmonary branch arteries. The patients with significant pulmonary valve annulus hypoplasia have been previously treated using transannular patch or valve conduit insertion. The transannular patch immediately relieves the right ventricular hypertension and enhances right ventricular growth proportionally with patient growth which is particularly important for the young patients. We enlarged the RVOT by myectomy and closed the infundibular incision using a pericardial patch facilitating the dilatation of the RVOT.

The mid-term results of porcine bioprosthetic valves in the RVOT reconstruction were reported as excellent in the literature.[6] The main benefit of using bioprosthetic valve is avoidance of extensive dissection, easiness of implantation and good hemodynamic characteristics.[6] Mechanical valves have been used for pulmonary valve replacement in a limited number of centers.[7] Most centers recommend relatively large doses of warfarin and several reports of thromboses have been documented.[8,9] In this case, we preferred a stentless bioprosthetic valve to obtain a larger effective orifice area and prevent complications of warfarin and re-intervention of RVOT.

Conclusion

For the patients who reach an ideal body weight and older than 18 years old with a pulmonary trunk aneurysm which extends to the left hilum, there is no ideal conduit which can provide a reliable anatomical size in the present condition. If the wall thickness of the aneurysm permits, plicating the aneurysm with semicircular continuous sutures and reconstruction of the RVOT by a stentless porcine valve to provide the widest choice of effective orifice may be the closest way for an ideal treatment option.

Declaration of conflicting interests

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Case Report Open Access

Ogilvie's syndrome: an uncommon gastrointestinal complication following coronary artery bypass graft surgery

Hamit Serdar Başbuğ, Macit Bitargil, Kanat Özışık

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ABSTRACT

Ogilvie's syndrome or acute colonic pseudo-obstruction is characterized by an acute distention of the large bowel in the absence of any mechanical obstruction usually occurring in critical illness or following an extensive surgery. It is a rare gastrointestinal complication of open heart surgery. Massive distention of the colon may cause perforation in the cecum which causes subsequent fecal peritonitis and associated with a high mortality rate. In this article, we report a case of acute colonic pseudo-obstruction occurring following coronary artery bypass graft surgery and our treatment approach is discussed.

Keywords: Coronary artery bypass graft surgery; neostigmine; Ogilvie's syndrome.

Ogilvie's syndrome also called as colonic pseudo-obstruction is an acute colonic dilatation without any mechanical obstruction which may develop after surgery or systemic illness.[1] It is seen in less than 3.5% of patients after cardiovascular or thoracic surgery.[2,3] This syndrome is a type of megacolon syndrome and it remains as a poorly understood condition which is characterized by massive dilatation of the colon and the presence of fluid levels on abdominal X-ray.[4] It is also associated with a broad range of medical and surgical conditions including open heart surgery, trauma, other surgical interventions, malignancy, and pregnancy.[5] It is a rare, but lethal postoperative gastrointestinal complication following coronary artery bypass graft (CABG) surgery with a mortality rate as high as 50%.[4] In this report, a patient who developed an acute colonic pseudo-obstruction (ACPO) following CABG and the treatment approach was discussed.

CASE REPORT

A 67-year-old male patient with no significant history except hypertension for the past five years admitted to the cardiology department with unstable angina pectoris. Coronary angiography revealed multivessel coronary artery disease and he was scheduled for CABG surgery. He had a smoking history of two-packs per day for the past 50 years.

Preoperative blood tests were normal. Echocardiographic evaluation revealed a reduced ventricular function with a left ventricular ejection fraction of 35% with apical dyskinesia and no significant valvular pathology. His routine anti-hypertensive prescription included an angiotensin receptor blocker monotherapy.

After performing four vessel CABG surgery, the patient was transferred to the intensive care unit with an infusion of moderate doses of positive inotropic agents. The intra-aortic counter pulsation was established in the postoperative seventh hour due to the low cardiac output. Hemodynamically-stabilized patient was extubated on the postoperative first day. The inotropic agents were initiated to attenuate gradually. The otherwise stable patient developed an abdominal distention during the end of postoperative first day. Gastrointestinal symptoms worsened on the second day. All blood tests and blood gas analysis produced normal results without any electrolyte imbalance.

On physical examination, diminished bowel sounds with a markedly distended abdomen were inspected. The abdominal palpation was painful. However, there was no rebound, an indicator of the peritoneal irritation. The erected position plain abdominal roentgenogram revealed a generalized significant

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colonic flatus (Figure 1). Abdominal ultrasound investigation showed no intra-abdominal pathology regarding neither an organomegaly nor a peritoneal free fluid. There was no evidence of a mechanical bowel obstruction or an intestinal perforation, either. A nasogastric (NG) and a rectal tube were introduced following a useless rectal enema. After the constitution of nil per os (NPO), parenteral nutrition (Kabiven Peripheral™, Fresenius Kabi AG, Germany) was initiated to maintain a daily calorie intake. The pharmacologic agents which promote the bowel motility such as metoclopramide and simethicone/alverin citrate were initiated with a spasmolytic agent hyoscine-N-butylbromide. Intravenous fluid and electrolyte replacement continued for hydration as the oral intake had been totally stopped. The intra-aortic balloon pump was retrieved as the hemodynamic parameters were normal. He was mobilized to reconstitute the intestinal motility. Despite all these supportive measures, no fecal passage or discharge of flatus through the rectal tube were achieved. Purgation, enemas, decompression of the flatus via the gastric tube failed.

In the postoperative fourth day, abdominal distention progressed to a risky level and roentgenogram showed a further distention of the colon (Figure 2). Continued medical treatment failed and the patient was unable to be mobilized due to pain. As the diagnosis was considered to be Ogilvie's syndrome, we decided to administrate neostigmine methylsulfate, a parasympathomimetic agent. At two hours following the initial intravenous bolus dose of 2.5 mg neostigmine methylsulfate, the bowel gas output was observed. The patient was able to defecate at six hours following the initial dose and the abdominal distention gradually resolved. No side effects except a slight bradycardia (heart rate of 65/bpm) were observed. Despite the reconstitution of the intestinal motility, the medication was continued for two more days with a daily dose of 2.0 mg intravenously.

The patient was discharged in the postoperative eight day with a well overall hemodynamic condition with no residual abdominal symptoms.

DISCUSSION

Progressive colonic dilatation following surgery is clinically indicative of the diagnosis of Ogilvie's syndrome in the absence of a mechanical obstruction.[4] In 1948, Sir William Heneage Ogilvie, the Chilean-born British gastrointestinal surgeon and orthopedist, initially described a novel clinical syndrome with a report of two cases.[6] This new syndrome which is characterized by acute abdominal pain, constipation and large bowel distention without a mechanical obstruction was then named as Ogilvie's syndrome.

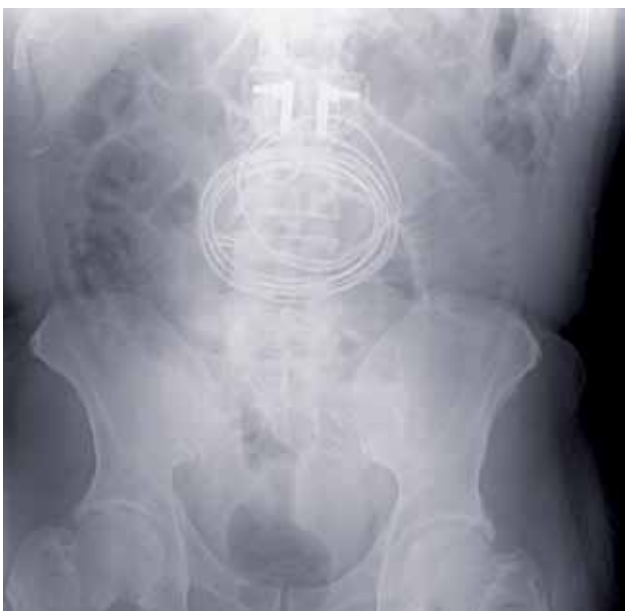

Figure 1. The plain abdominal roentgenogram showing moderate colonic distention (postoperative day 2).

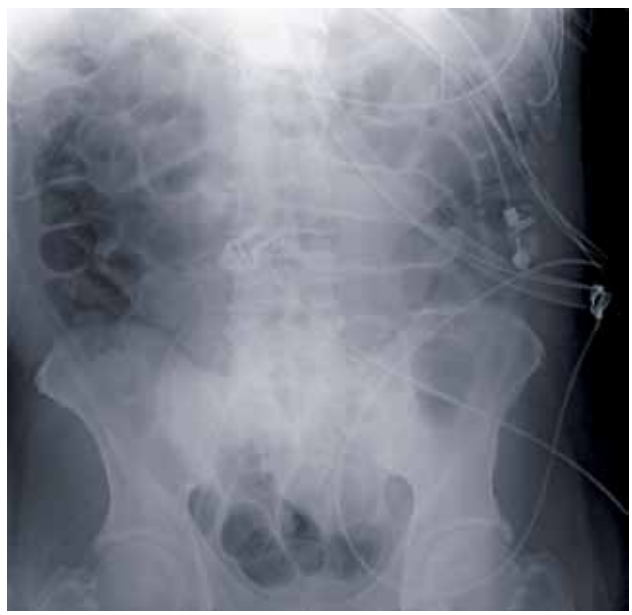

Figure 2. The plain abdominal roentgenogram showing severe colonic distention (postoperative day 4).

Although the exact pathophysiology of the ACPO is still obscure, a parasympathetic deprivation is thought to be responsible.[7]

Ogilvie's syndrome is mostly seen in sexagenarians and septuagenarians. Also, it is two times higher in males than females.[4] The initial studies investigated the etiology were concentrated on cesarean sections. However, later studies revealed that cardiothoracic surgery as well as lumbar spine and hip procedures were dominantly responsible.[4,8]

Although the pathophysiological mechanism of Ogilvie's syndrome following cardiovascular surgery still remains to be elucidated, it is often thought to be neurogenic. The possible scenario explaining the mechanism of the colonic pseudo-obstruction seems to be the lack of parasympathetic innervation to the colon.[4] The success of a parasympathomimetic drug neostigmine in the treatment of the ACPO also proves this theory of neuropraxia.[1,5] If it is a type of a neurological dysfunction, what could be the cause of this parasympathetic neuropraxia? Is it related to a trauma of the parasympathetic ganglia? Alternatively, is it solely because of excessive sympathetic discharge due to the pain or stress during the operation?

An intraoperative trauma or direct manipulation of the ganglia or its branches seems to be impossible in cardiothoracic, orthopedic, and the lumbar spinal cord operations. However, the vagus nerve, which is the main parasympathetic trunk of the gastrointestinal system, may be injured during CABG surgery due to local hypothermic slush application or during extensive lateral pericardiotomy.[9] Besides, sympathetic discharge may occur, when the patient accidentally awakes due to inadequate anesthesia and this excessive sympathetic surge may easily cause relative loss of parasympathetics.[4] Although it is still unproven, the extracorporeal circulation during CABG surgery may contribute to neurogenic disarrangement.[4] An elongated extracorporeal circulation may also lead to increased ischemia of the parasympathetic ganglia due to hypotension, thereby, eventually cause synaptic retardation.[10] In our case, a reasonable cardiopulmonary bypass time was achieved comparing to a four-vessel CABG surgery. The circulation pressure of the heart-lung machine was around 70 mmHg during the extracorporeal circulation period which corresponds to the normal mean value. In the light of all these discussed hemodynamic parameters, the possibility of a mechanical or ischemic trauma can be excluded.

Furthermore, the diagnosis depends on the physical examination, follow-up and imaging with an erected abdominal roentgenogram, irrespective of the pathophysiology. During the postoperative period, a massive distention of the abdomen with impaired flatus or stool outflow should always remind the Ogilvie's syndrome.[4]

In the treatment of Ogilvie's syndrome, the NPO, NG, and rectal tubes should be the first to constitute by means of immediate colonic decompression. The supportive intravenous medication including fluid and electrolyte replacement should be applied afterward. Follow-up may lead to complete resolution of the ACPO or may show further progression. In this presented case, as the supportive measures did not improve the patient's overall status, neostigmine was initiated as an advanced treatment.[11] Neostigmine is a parasympathomimetic which inhibits the acetylcholinesterase enzyme, which is responsible for the breakdown of the acetylcholine (Ach) molecule. As it blocks the binding site of the acetylcholinesterase, the enzyme can no longer interfere the Ach before it interacts with the receptors of the postsynaptic membrane.[12] It eventually helps the threshold to be reached and the impulse can be triggered in the next neuron. Increase in the parasympathetic activity further initiates the intestinal motility resulting the resolution of the abdominal distention.[13]

When any other medical treatments are useless to resolve the state of ACPO, colonic decompression via colonoscopy, percutaneous decompression or laparotomy should be considered.[8,14] If left untreated, colon perforation due to necrosis of the cecum may cause fecal peritonitis and death, eventually.[15]

In conclusion, in case of an acute megacolon following the CABG surgery, the diagnosis should be considered as Ogilvie's syndrome. Immediate diagnosis and effective treatment are necessary to prevent colonic necrosis and the eventual perforation. In addition to the supportive treatment, neostigmine, a parasympathomimetic, can be used to reconstitute the intestinal motility. Surgical option should be reserved as the last chance due to its high potential for mortality.

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Cardiovascular Surgery and Interventions

Case Report Open Access

A vascular phenomenon which should be kept in mind: subclavian steal syndrome

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ABSTRACT

The subclavian steal syndrome is a rare, yet well-known phenomenon which presents when a steno-occlusive lesion of the proximal subclavian artery results in the flow reversal of the vertebral artery, giving rise to vertebrobasilar sufficiency. A 50-year-old male patient was admitted to our clinic with complaints of the left arm coldness and pain on exertion. Physical examination revealed a cold, pale and pulseless left arm. We performed carotico-subclavian bypass with a 6 mm ringed polytetrafluoroethylene graft. Postoperative radial and ulnar pulses were palpable and his complaints resolved.

Keywords: Carotid artery; steal phenomenon; subclavian artery.

The subclavian steal syndrome (SSS) is a rare yet well-known phenomenon which presents when a steno-occlusive lesion of the proximal subclavian artery results in the flow reversal of the vertebral artery, giving rise to vertebrobasilar sufficiency. It was first described in 1961 by Reivich in the New England Journal of Medicine.[1] The author reported a patient with a neurological symptom which was directly attributed to this reversal phenomenon. However, the term "subclavian steal" was introduced by Miller Fisher, on an editorial paper few months later.[2]

Subclavian steal phenomenon is a functional result of the proximal subclavian artery steno-occlusive disease with subsequent retrograde blood flow in the ipsilateral vertebral artery. The symptoms related to the compromised vertebrobasilar and brachial blood flows constitute the SSS and include paroxysmal vertigo, drop attacks or claudication on arm. Once thought to be rare, the emergence of novel imaging techniques has improved its diagnosis and prevalence. The syndrome, however, remains characteristically asymptomatic and solely poses no serious danger to the brain. Recent studies have shown a linear correlation between increasing arm blood pressure difference and the development of the symptoms. Atherosclerosis is the most common cause of subclavian steal syndrome and it is more common on the left side, possibly due to a more acute origin of the left subclavian artery, resulting in accelerated atherosclerosis caused by increased turbulence.[3] Doppler ultrasound is a useful screening tool, however, the diagnosis must be confirmed by computed tomography or magnetic resonance angiography. Conservative treatment is the initial therapy for this syndrome and surgery is needed for refractory symptomatic cases. Other treatment modalities include percutaneous angioplasty and stenting, rather than bypass grafts of the subclavian artery.

CASE REPORT

A 50-year-old male patient was admitted to our clinic with complaints of left arm coldness and pain on exertion. Physical examination revealed cold, pale and pulseless left arm. Doppler ultrasonography of the left upper limb showed monophasic flow in both radial and ulnar arteries. We applied digital subtraction angiography which revealed proximal subclavian artery occlusion and retrograde filling through vertebral artery (Figure 1). A carotico-subclavian artery bypass was scheduled. Before surgery, a carotid artery Doppler ultrasonography was applied for screening of carotid plaque. Under general anesthesia, patient was positioned in supine position, the left arm was adducted

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and the left shoulder was slightly elevated. The incision started from sternoclavicular joint to acromioclavicular joint parallel and 1.5 cm above to clavicle. Dissection made through platysma and anterior scalene muscles. Below to the anterior scalene muscle, the proximal subclavian artery was exposed. Particular attention was given to preserve phrenic nerve and thoracic duct. In the anterior to the sternocleidomastoid muscle, the carotid artery was exposed. After systemic heparinization, both arteries were clamped and a 6 mm ringed polytetrafluoroethylene graft was anastomosed end-to-side. Postoperative radial and ulnar pulses were palpable and his complaints resolved (Figure 2). In the postoperative third day, the patient was discharged uneventfully.

DISCUSSION

Due to the rarity of the SSS, suspicion is crucial for the diagnosis of the disease. The clinician must receive a detailed history. A careful physical examination is essential looking for common SSS findings to distinguish from the differential diagnosis which includes intracranial vascular disease, carotid artery disease, vertebral artery disease, brain tumors and subdural hematomas.[4-8] The SSS may present with absence or diminished ipsilateral radial pulse, cervical or supraclavicular bruits or thrills, and the presence of a systolic blood pressure difference >20 mmHg between two upper limbs.[9]

There are two surgical treatment options in the management of SSS. Subclavian-subclavian bypass is the first choice in patients with carotid artery stenosis or plaques, in particular. The major disadvantage of subclavian-subclavian bypass is difficulty of exposure, while another disadvantage is the requirement of long graft length. The second management option is carotico-subclavian bypass featured in patients without a carotid lesion, in particular. Short graft length and high long-term patency rates are the main advantages of carotico-subclavian bypass. In our case, the carotid artery was patent and there was no plaque compromising flow, therefore, we performed carotico-subclavian bypass. In the literature, there are some reports which described effectiveness of percutaneous treatment options.[10,11]

In conclusion, subclavian steal syndrome is a rare vascular phenomenon which occurs due to a steno occlusive disease of subclavian artery and it can be treated with surgery or percutaneous angioplasty and stenting. Suspicion is crucial for the diagnosis of the disease. For this reason physicians should raise their awareness for this rare vascular disease. We hope this case report will help clinicians to improve life quality of their patients.

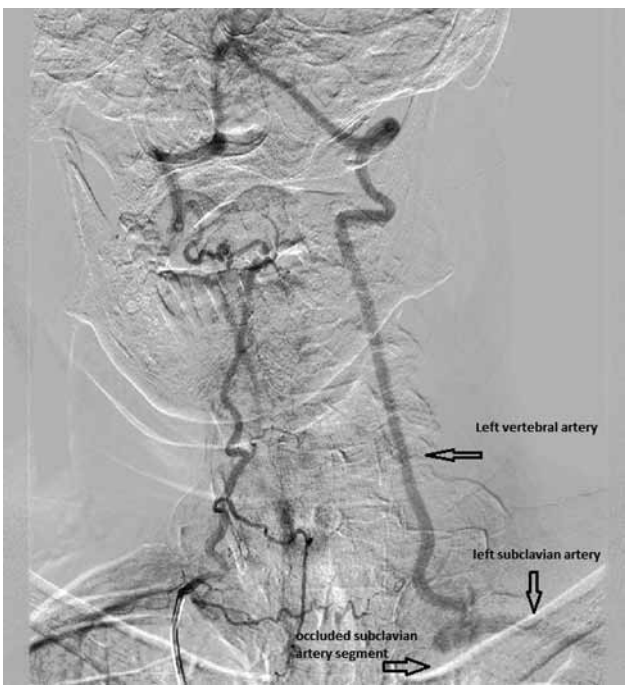

Figure 1. Digital subtraction angiography image showing proximal subclavian artery occlusion and retrograde filling through vertebral artery.

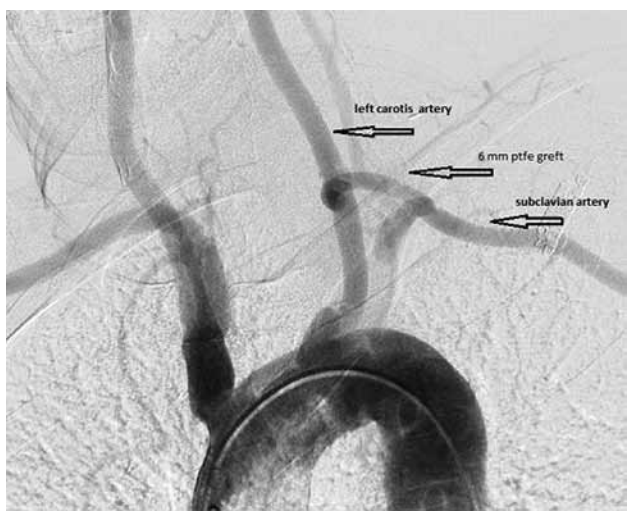

Figure 2. Digital subtraction angiography image showing carotico-subclavian bypass with a 6 mm ringed polytetrafluoroethylene graft.

Declaration of conflicting interests

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Cardiovascular Surgery and Interventions

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Anesthesia management in left cardiac sympathetic denervation with catecholaminergic polymorphic ventricular tachycardia

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ABSTRACT

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a rare familial cardiac arrhythmia characterized by RYR2 or CASQ2 gene mutation. This arrhythmia occurs in patients with a structurally normal heart. The syndrome may cause sudden cardiac arrest due to exercise or emotional stress related ventricular fibrillation. Most frequent clinical symptom is syncope. Currently, no definite anesthetic approach is available for the management of these patients. In this article, we report a case of CPVT and discuss anesthetic management.

Keywords: Anesthesia; catecholaminergic polymorphic ventricular tachycardia; left cardiac sympathetic denervation.

The catecholaminergic polymorphic ventricular tachycardia (CPVT) is potentially lethal, cardiac channelopathie. Treatment strategies include pharmacotherapy with β-blockers, implantable cardioverter defibrillators, and left cardiac sympathetic denervation (LCSD).

CASE REPORT

A 19-year-old female patient with a prior history of exercise-related syncope for two times was admitted to our clinic. She was five- and eight-year-old during her first and second syncopal episode, respectively. Further investigation demonstrated premature heartbeat, bidirectional ventricular tachycardia on 24-h Holter monitoring. The patient was diagnosed with CPVT accompanied by typical electrocardiogram (ECG) findings (Figure 1). Because of recurrent episodes of exercise-related syncope, she was treated with bisoprolol and verapamil. Although she was on medical treatment, she had cardiac arrest. Her mother applied basic life support and she was brought to the hospital. An implantable cardiac defibrillator (ICD) implantation was recommended with beta-blocker therapy. The first ICD was implanted; however, it was discharged in five months. Then, the second ICD was implanted. Nevertheless, it was discharged one month later. As a result, she was treated with a daily dose of bisoprolol 10 mg three times a day and flecainide 100 mg bid. The patient remained asymptomatic for five years. At five years, she had a repeated syncopal episode as assessed by regular examinations. Following this episode, LCSD was recommended.

After written informed consent was obtained, she was pre-medicated with midazolam 3 mg (intramuscular) before the entrance to the operation room. Upon arrival to the operating room, an external defibrillator pad was placed and monitored through electrocardiogram (recording leads D2 and V5), non-invasive blood pressure and pulse oximetry.

Induction was performed with propofol 2 mg/kg, fentanyl 2 µg/kg and rocuronium 0.5 mg/kg. Single-lung ventilation was achieved with a 35 F Portex endobronchial double lumen tube (Smiths Medical International Ltd., Keene, NH, USA). The patient was positioned on the operating table with her right side down. An epidural catheter was inserted into the thoracal epidural space 5-6th and then 3 mL 0.5% bupivacaine, 50 µg fentanyl and 3 mL normal saline were injected through the catheter. Total volume was 7 mL. For the maintenance of anesthesia, propofolol 2 mg/kg/h was applied.

The sympathetic ganglia were identified through the pleura, which were dissected to expose

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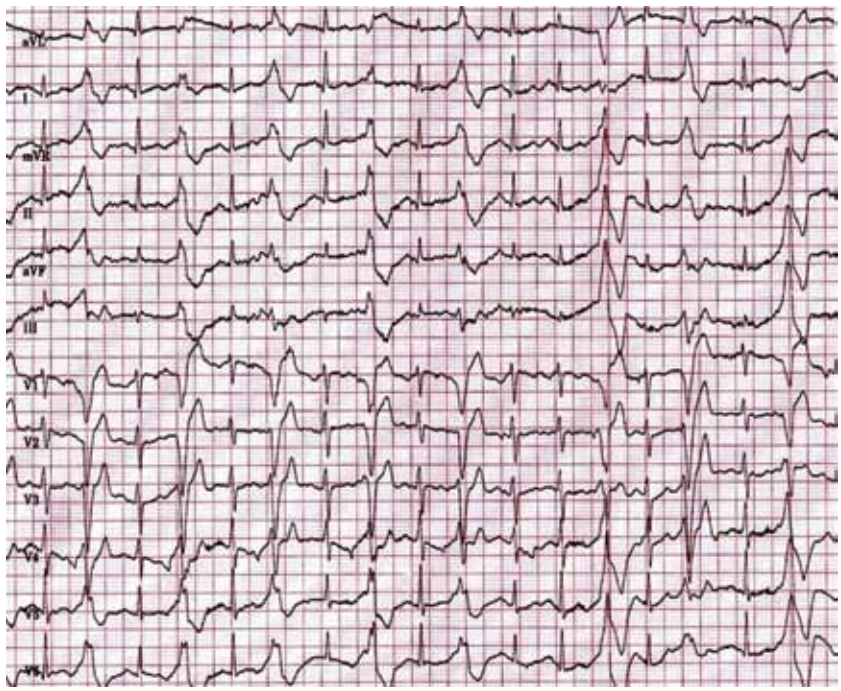

Figure 1. Electrocardiogram finding; catecholaminergic polymorphic ventricular tachycardia.

the left-sided sympathetic chain from T_4 to T_1 via videoscopic transthoracic approach. The patient received bupivacaine 15 mg and fentanyl 50 µg for intraoperative pain management through a thoracal epidural catheter. For nausea prophylaxis, the patient was given ondansetron 4 mg perioperatively and the patient received glycopyrrolate and neostigmine for the reversal of paralysis. The patient was, then, extubated in the operation room and monitored during 24 hours postoperatively.

DISCUSSION

Catecholaminergic polymorphic ventricular tachycardia is a rare malignant inherited arrhythmia syndrome.[1] It usually presents in childhood or young adulthood with a history of physical or emotional stress-induced syncope or cardiac arrest.[1] Arrhythmia is characterized by bidirectional or polymorphic ventricular tachicardia in patients with a structurally normal heart.[2-4]

Catecholaminergic polymorphic ventricular tachycardia is associated with RYR2 and CASQ2 gene mutation. The inheritance pattern is autosomal-dominant and autosomal-recessive, respectively.[4,5] These genes are related to calcium channels.[2] A family history of syncope or cardiac death is positive in approximately 35% of patients with CPVT.[3]

Catecholaminergic polymorphic ventricular tachycardia was first described in 1975 by Reid et al.[3,4,6] Diagnosis can be challenging. As ECG usually produces normal results in asymptomatic patients, ECG is not specific.[2,4] The diagnosis is based on ECG findings during exercise. During exercise, typical ECG finding is bidirectional tachycardia.[1,4,7,8]

The medical treatment of CPVT is based on beta-blockers.[1] Syncopal attacks can be controlled by beta-adrenergic blockers. Nadolol is the preferred agent thanks to its prolonged half-life. Therefore, beta-blockers are effective for the acute phase and they are the first-line of treatment.[1,3,7,9,10] Other therapeutic options for patient with CPVT include calcium channel blockers and/or flecainide. However, an ICD should be planted in patients with recurrent arrhythmias or cardiac arrest episodes.[1,3,4]

In this case, our patient presented due to exercise-related syncope with a prior history of cardiac arrest. She was diagnosed as CPVT based on her medical history and typical ECG findings. In the beginning, she was treated with a single beta-blocker agent, however, the patient presented again with cardiac arrest

while she was on medical treatment. An automatic ICD was implanted. The first ICD depleted in five months. As a result, the second ICD was implanted; however, it depleted one month later. The depletion was caused by inappropriate multiple ICD shocks. During a five-year follow-up, the patient remained asymptomatic. At five-years, she had a repeated syncopal episode as assessed by regular examinations. Therefore, LCSD was recommended. An ICD was recommended for CPVT patients who have cardiac arrest or syncopal attack despite receiving maximum dosage of beta-blocker therapy.[11,12] However, when the symptoms persist despite beta blockade, calcium channel blockers, ICD, and LCSD are effective alternative treatments.[1,11,13]

Left cardiac sympathetic denervation is described in 1971.[14,15] Surgically LCSD involves resection of the lower half of the left stellate ganglion and the left sided sympathetic chain. Left cardiac sympathetic denervation has been used as an effective option for patient with CPVT.[14,16] However, clinical experience of LCSD in CPVT patients is limited. In addition, there are few reports on the anesthetic procedure of patients with CPVT. The goal of the treatment is to manage the adrenergic stimulation, since adrenergic stimulation may provoke arrhythmias.[13]

Operative management of these patients is often challenging. There is a high risk of life-threatening arrhythmia and sudden cardiac arrest. Anesthetic management should be planned carefully.[13,17,18] Careful planning and monitorization are critical to ensure a safe operation. The operation room should be appropriate for the induction. Hyper-hypotension, bradycardia, tachycardia, hypothermia, hyper-hypocapnia, hypoxemia should be also controlled, as these conditions can effect cardiac activity.[11,19]

Serum electrolytes should be measured. An external defibrillator and blood pressure monitoring should also be present in the operating room. Premedication is necessary to avoid increased sympathetic activity.[19-21]

For premedication, midazolom is used in our patient. Propofol was used in our patient for the induction. Propofol has been used as induction and maintenance agent in patients with CPVT to avoid complications.[1] Rocuronium was administered to our patient for muscle relaxation. Rocuronium can be safely administered to this group of patients.[1] In this present case, video-assisted thoracoscopic cardiac denervation, a minimally invasive procedure, was made.

Bupivacaine and fentanyl were used for the pain management and to maintain medical sympathetic denervation. Then, a thoracal epidural catheter was placed. For the management of postoperative nausea and vomiting, ondansetron was initiated. No significant complication and adverse effects were noted during the operation.

In conclusion, anesthetic management of the patient with CPVT requires careful monitoring of cardiac parameter, understanding of risks, and good management of postoperative pain control.

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Cardiovascular Surgery and Interventions

Case Report Open Access

When should we re-cycle internal mammary artery in redo coronary artery bypass graft surgery?

Anıl Özen, Ertekin Utku Ünal, Emre Kubat, Sercan Tak, Başak Soran Türkcan, Ufuk Tütün, Cemal Levent Birincioğlu

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ABSTRACT

A 56-year-old male patient was admitted to our hospital with unstable angina pectoris lasting for six hours. He underwent left internal mammary artery to left anterior descending artery bypass grafting two years ago. Coronary angiography revealed significant stenosis of the LIMA-LAD bypass. The LIMA was re-cycled and anastomosed to the first diagonal branch and the right internal mammary artery was harvested and anastomosed to the distal part of the stenotic region. The advantage of this operation is re-using the previous arterial graft which is essential for this young patient with a longer life expectancy.

Keywords: Coronary artery bypass graft; internal thoracic artery; surgery.

Stenosis of the graft bypassed to the left anterior descending artery (LAD) is associated with an increased risk of morbidity and mortality. The major problem in re-do coronary artery bypass graft (CABG) surgery is the lack of an appropriate graft with a good long-term patency. Herein, we describe a patient reoperated for the significant stenosis distal to left internal mammary artery to left anterior descending artery (LIMA-LAD) anastomosis site. The LIMA was re-used having anastomosed to the distal part of the stenotic diagonal branch of the LAD and the right internal mammary artery (RIMA) was anastomosed to the distal part of the LAD stenosis.

CASE REPORT

A 56-year-old hypertensive male patient with hyperlipidemia who underwent LIMA-LAD CABG two years ago was admitted to the cardiology intensive care unit for unstable chest pain lasting for six hours. Coronary angiography revealed a significant stenosis of the anastomosis of the LIMA-LAD bypass. Physical examination and blood test results were normal. The decision for re-do CABG was made. The patient underwent re-do surgery the next day.

The right femoral artery and vein were encircled by tapes for possibility of an urgent cardiopulmonary bypass (CPB). Sternotomy was made from the previous incision. Adhesions surrounding the heart were dissected carefully. Following heparinization, aorto-atrial cannulation was performed. Cardiopulmonary bypass was initiated and the X-clamp was placed. A bulldog clamp (Stoelting Co, Illinois, USA) was placed onto the patent LIMA. Cardiac arrest was established after introducing cardioplegia via the antegrade route. The LIMA was freed from the proximal part so to extend enough to bypass the distal part of the previous anastomosis. Even so, the LIMA did not reach the desired area. Hence, it was anastomosed to the stenotic first diagonal branch. The RIMA was harvested and anastomosed to the desired area (1.5 cm distal to the previous anastomosis). Following this, warm cardioplegia was introduced. The X-clamp was removed and the bulldog clamp on the LIMA was removed. The heart, then, started beating spontaneously. Weaning from CPB was challenging. Following decannulation, the thorax was closed in layers.

The patient was extubated at the eighth postoperative hour and transferred to the ward. He was discharged at the fourth postoperative day. He was seen at the outpatient clinic at two months and two years following surgery. He had no complaint of chest

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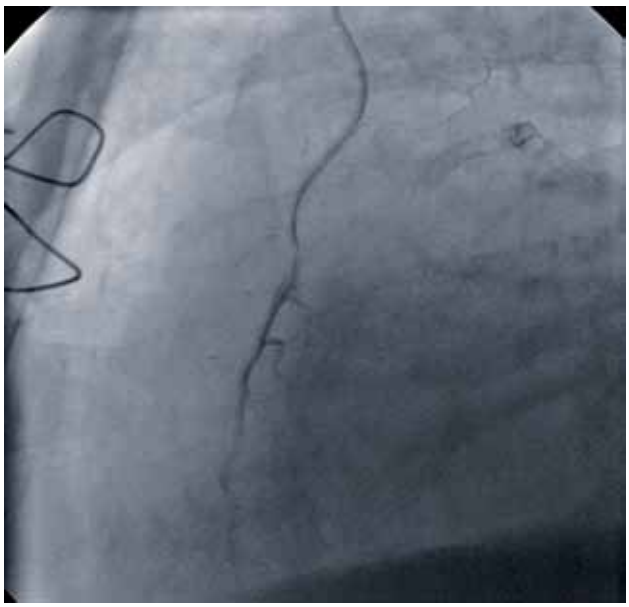

Figure 1. Significant stenosis of the left internal mammary artery to left anterior descending artery bypass seen in the postoperative coronary angiography.

pain. Doppler ultrasound revealed patent LIMA and RIMA grafts with a good flow.

DISCUSSION

In the recent years, the incidence of re-do CABG surgery has declined due to the increased use of multiple arterial grafts. Patients initially undergoing CABG with arterial grafts frequently have shortage of arterial grafts for their second revascularization procedure.[1] Hence, the re-cycling techniques may help optimizing arterial revascularization in young patients, in particular, with a longer life expectancy.

One possibility of re-cycling is to use the patent internal thoracic arteries (ITAs) as an inflow for the new Y composite configuration. This represents the most commonly applied technique in some practices like Barra et al.[2] and Tector et al.[3] The second possibility is the re-implantation of the distal ITA anastomosis on the same coronary vessel (15%). The main objective is to bypass a stenosis in the coronary artery distal to the anastomosis or due to a peri-anastomotic lesion. This technique obliges an adequate length of the preexisting left ITA. Pasic et al.[4] demonstrated excellent mid-term patency (2.6 years) for ITA re-implantation of 12 patients.

During re-do CABG, Dohi et al.[5] dissected the LIMA, re-used it *in situ* for the circumflex artery, and used the RIMA to the LAD. This possibility was the appropriate choice for our patient with a slight modification made by anastomosing the LIMA to the fist diagonal branch and the RIMA to the LAD.

Although Antona et al.[6] were able to obtain an IMA pedicle long enough to reach the heart. In certain cases, the anastomosis of the salvaged graft on the same coronary artery may cause excessive tension on the anastomosis itself. For such cases, the "short" IMA can be re-routed to a more accessible coronary artery which needs revascularization or it can be elongated with an interposition of great saphenous vein or with other arterial conduits.

Another method may be skeletonizing the LIMA. At a re-do operation, Uwabe et al.[7] skeletonized the LIMA and re-used it *in situ* to the LAD. Using the skeletonized method for re-harvesting LIMA made the graft reach to a more distal portion without tension.

Re-cycling is generally performed on a highly selected group of re-do CABG patients. Certain factors are mandatory for the re-cycling process such as a well-developed ITA with a minimal diameter >2.5 mm as confirmed by coronary angiography, patent IMA grafts without significant stenosis and sufficient LIMA length. Re-sternotomy has to be done very carefully to avoid any damage to the ITA graft. Young patients (<60 years) with a longer life expectancy, presenting for re-do CABG surgery with patent ITA may benefit from salvage and re-use of the ITA grafts.[8] As our patient met the necessary criteria mentioned above, LIMA was successfully re-cycled and used. Another point to attract attention is the possibility of follow-up using Doppler ultrasound.

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