Extracranial Right Vertebral Artery Dissection in an Advanced Heart Failure Patient with Heartware Left Ventricular Assist Device Support: Case Report

Son Dönem Kalp Yetmezliği Sebebiyle Heartware Sol Ventrikül Destek Sistemi İmplantasyonu Yapılan Hastada Ekstrakranial Sağ Vertebral Arter Diseksiyonu

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Geliş Tarihi/*Received:* 24.02.2014
Kabul Tarihi/*Accepted:* 03.03.2014

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ABSTRACT We report a 57-year-old lady who had been suffering from advanced heart failure for more than 3 years under maximal medical therapeutic treatment, and had left ventricular assist device implantation (LVAD) due to exacerbation of her heart failure. She was New York Heart Association Class IV and Interagency Registry for Mechanically Assisted Circulatory Support level I. She had HeartWare LVAD implantation and recovered well after the postoperative period. She was admitted to emergency department with headache, vertigo, neck and occipital pain in the postoperative 4th month. She could not have magnetic resonance imaging since she had cardioverter defibrillator implantation(CRT-D). Her kranial computerized tomography (CT) reported a small right thalamic infarct which was treated with medical therapy. Twenty days later after the first event, she was admitted to emergency unit with dysarthria, dysphasia, right paresthesia and truncal ataxia. She had CT angiography, and was reported as right vertebral artery dissection which is an uncommon cause of mild and significant trauma. Our patient could not have benefit from endovascular management since posterior inferior cerebellar artery supply would be affected, and possible infarct might occur. The only accepted medical therapy was management of possible ischemic stroke by anti-coagulation or by anti-platelet therapy. Our current case shows that the neurologic symptoms patients with LVAD may not be only due to pump thrombosis or malfunction of the LVAD. We report the first case in the literature with advanced heart failure supported with LVAD and diagnosed with extracranial right vertebral artery dissection.

Key Words: Heart-assist devices; carotid artery diseases; endovascular procedures

ÖZET Son 3 yıldan beri son dönem kalp yetmezliği tedavisi sebebiyle yüksek doz medikal tedavi alan, sol ventrikül destek sistemi (LVAD) implante edilen 57 yaşında bayan hastayı sunuyoruz. Hastanın son dönem kalp yetmezliğinin dekompanse olması sebebiyle New York Heart Association Klas IV ve Interagency Registry for Mechanically Assisted Circulatory Support Seviye I idi. Hastaya HeartWare LVAD implantasyonu yapıldı ve operasyondan sonra hastada hızlı bir düzelme gözlendi. Hasta postoperatif 4. ayında acil servise baş ağrısı, baş dönmesi, oksipital bölgede ve boyunda ağrı şikayetiyle başvurdu. Hastanın daha önce implante edilen kalp pili sebebiyle hastaya manyetik rezonans görüntülemesi yapılamadı. Hastanın bilgisayarlı beyin tomografi görüntülemesinde, sağ hemisferde talamik infarkt tespit edildi, medikal tedavi uygulandı. İlk nörolojik olaydan 20 gün sonra, hasta acil servise disarti, disfazi, sağ parestezi ve trunkal ataksi ile başvurdu. Hastanın çekilen bilgisayarlı beyin angiografisinde, orta ve ciddi travmaya bağlı olduğu düşünülen sağ vertebral arter diseksiyonu tespit edildi. Hasta, fizik tedavi ve rehabilitasyon amaçlı agresif boyun masajı yaptırdığını belirtti. Sağ vertebral artere endovasküler stent işlemi sonucunda posterior inferior serebellar arterin kan akımının etkileneceği ve infarkt gelişebileceği düşüncesiyle, hastanın endovasküler müdahaleye uygun olmadığına karar verildi. Sağ vertebral arter diseksiyonuna bağlı gelişebilecek iskemik felç durumunu önlemek için anti-koagulasyon ve anti-platelet tedavisine devam edildi. Sunduğumuz olguda, özellikle nörolojik semtoplarla başvuran LVAD hastalarında sadece pompa trombozu veya LVAD disfonksiyonundan şüphenilmemesi gerektiğini, ve ayırıcı tanı için ileri tetkikler yapılmasının gerekliliğini gösterdik. Bu hastayı son dönem kalp yetmezliğine bağlı sol ventrikül destek sistemi ile takip edilen ve ekstrakranial sağ vertebral arter diseksiyonu tanısı alan literatürdeki ilk olgu olarak sunuyoruz.

Anahtar Kelimeler: Kalp destek cihazları; karotis arter hastalıkları; endovasküler prosedürler

doi: 10.9739/uvcd.2014-39430

Damar Cer Derg 2016;25(1):34-8

Advanced heart failure therapy is challenging. New therapies would increase the survival and quality of life of these end-stage heart failure patients. Mechanical circulatory support is required for bridging end-stage heart failure patients to heart transplantation or destination therapy. Left ventricular assist device (LVAD) is a new physiology and paradigm. Mortality and morbidity of LVAD therapy increase with the severity of the heart disease and Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) Level; the profiles range from critical cardiogenic shock to patients with advanced New York Heart Association (NYHA) Class III symptoms.

The LVAD patients may experience ischemic or hemorrhagic stroke as adverse events while being supported with the pump. Thrombus development within these pumps greatly increases the risk of cerebral embolism. Any thrombus that forms adheres to pump's internal surface, and eventually metabolizes or may be ingested from left atrium or left ventricle.[1]The incidence of cerebrovascular accidents (CVA) after LVAD placement was reported to range between 8% to 30%, observed after 3 to 6 months.[1-3]

Traumatic vertebral artery dissection (VAD) is frequently unrecognized, the common symptoms of VAD at presentation include headache, vertigo, severe neck and occipital pain. Ophthalmic symptoms occur as primary presenting complaint in 15-86% of cases. Endovascular management of extracranial arterial dissection might be safe and technically feasible in selected cases.

Herein, we describe a 57-year-old lady with end-stage heart failure, had HeartWare LVAD implantation (HeartWare Inc, Framingham, MA, USA), re-admitted to emergency department with motor aphasia, severe neck and occipital pain and dysphasia, and diagnosed with extracranial right vertebral artery dissection in the postoperative 4th month.

CASE REPORT

A 57-year-old lady had been suffering from end-stage heart failure more than 3 years. She had several admissions to hospital due to exacerbation of her heart failure. She had cardioverter defibrillator implantation (CRT-D) (St.Jude Medical Inc, St. Paul, MN, USA) in June 2012. She had admitted to our heart failure center in June 2013, was INTERMACS Level I, New York Heart Association (NYHA) Class IV. She had life threatening hypotension despite escalating inotropic support, critical organ hypoperfusion and pulmonary edema.

Her cardiac catheterization report revealed the cardiac index (CI) as 1.93 L/min/m^2, cardiac output (CO) as 3.4 L/min, and pulmonary capillary wedge pressure (PCWP) as 22 mmHg. Her echocardiographic studies showed the ejection fraction (EF) as 19%, left ventricular end-diastolic diameter (LVEDD) as 7.6 cm, right ventricular fractional area (RVFAC) as 20%, as well as mild mitral regurgitation, moderate tricuspid regurgitation with a systolic pulmonary artery pressure (sPAP) of 57 mmHg.

It was decided to implant left ventricular assist device as a bridge to transplantation. She had HeartWare LVAD implantation and tricuspid De-Vega annuloplasty procedure at mid-June 2013. Her postoperative period went uneventful, and had the cardiac rehabilitation program. She was discharged from hospital at postoperative 2nd week with NYHA Class I. Her CO was 4.5 L/min. She was able to mobilize by herself, her motor functions were improved when compared to the preoperative period when she was almost bed-ridden due to advanced heart failure.

Her routine follow-up was done in the out-patient clinic, her echocardiographic studies were done each month, and her biochemical and laboratory data were in the normal limits.

In October 2013, she was admitted to emergency unit with motor aphasia and occipital pain. There was no spinal tenderness, and her neurovascular examination was initially normal. Dysarthria and 3rd nerve paralysis were found on further examination. She was hospitalized, a magnetic resonance imaging (MRI) study could not be done due to implantation of CRT-D. Her first cranial computerized tomography (CT) reported an ill-defined,

slight edematous right thalamic infarct. Her cranial CT was repeated 24 hours later, and it clearly defined a right thalamic infarct (Figure 1).

Since the patient had LVAD implantation 4 months ago, she was been receiving anti-platelet and anti-coagulation therapy. Her INR level was between 2,5-3. She had an echocardiographic study to detect possible pump thrombus in LVAD. Four chambers of the heart, inflow cannula, out-flow graft anastomosis, and cusps of aortic valve were all reported as normal. She was discharged with anti-platelet and anti-coagulation therapy.

She re-admitted to emergency care unit 25 days later after the first event with severe occipital pain, headache, right eye ptosis, right hemiparesis and truncal ataxia.

Her cranial CT reported a subacute-chronic infract in right cerebellar hemisphere (Figure 2). Her CT cervical-cranial angiography demonstrated right vertebral artery irregularity between V_1-V_2 segments, and flow defect in V_3-V_4 segments.

It was considered that she might have right vertebral artery dissection, and further studies were performed. She had diagnostic cerebral angiography, and the dissection of the right vertebral artery was seen at V_2-V_3 level, and the dissected segment was occluded proximally until the origin of right posterior inferior cerebellar artery (PICA). Blood supply of the right PICA was maintained retrograde from the left side, through the vertebrobasiller junction (Figure 3).

Endovascular stenting of right vertebral artery was not considered as possible due to high risk of PICA infarct. The patient continued to have her anti-platelet and anti-coagulation therapy and her follow-up by neurology department. She was discharged from the hospital after her ophthalmic symptoms disappeared. She is now in post-LVAD 6th month, NYHA Class IV and has a good quality of life (Figure 4).

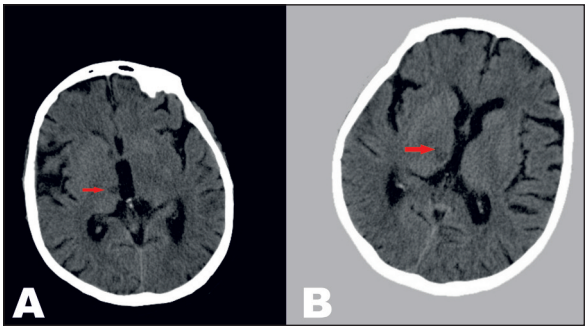

FIGURE 1: (A) The right arrow shows ill-defined right thalamic infract. **(B)** clearly defined right thalamic infarct after 24 hours in the cranial computerized tomography.

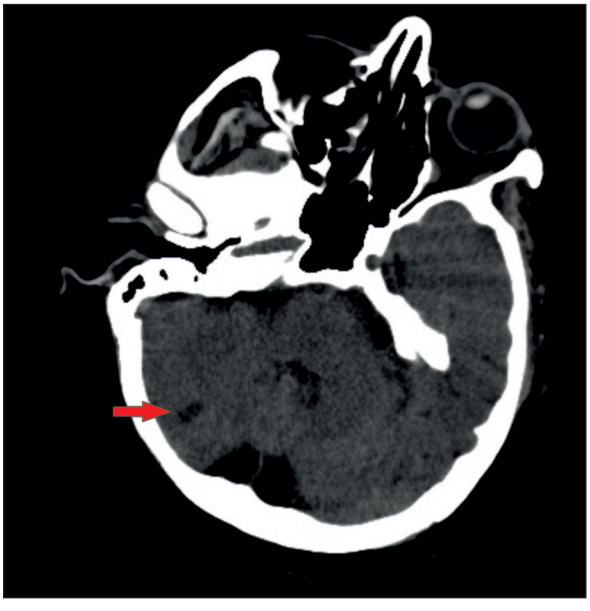

FIGURE 2: The arrow shows subacute-chronic infract in the right cerebellar hemisphere in the cranial computerized tomography.

DISCUSSION

Vertebral artery dissection can occur spontaneously, the annual incidence of spontaneous vertebral artery dissections has been estimated at 1 to 1.5 per 100 000 and reported that it occurs with mild or significant trauma in 80% of cases.[4] It is usually misdiagnosed as musculoskeletal pain at presentation which can lead to delay the diagnosis and treatment.

Our patient had idiopathic cardiomyopathy with enlarged heart chambers and left ventricle assist device implantation. In LVAD-related cerebrovascular events, there is a chance of possible ischemic and hemorrhagic stroke. The source of thrombus may be smaller deposits within the

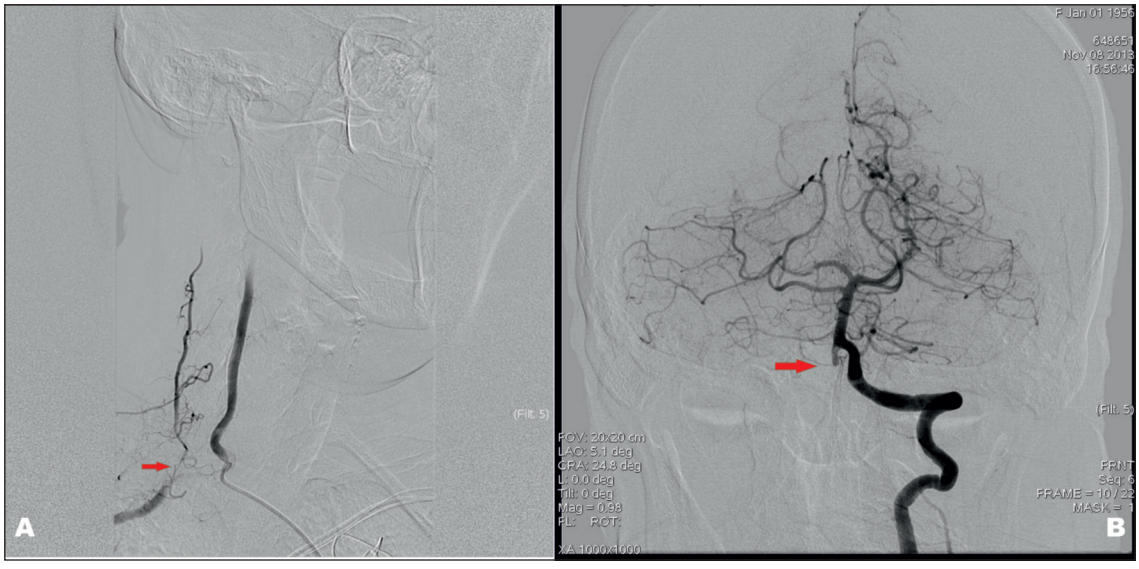

FIGURE 3: (A) The arrow shows the dissected segment between V2-V4. **(B)** The arrow shows the retrograde flow to dissected right vertebral artery from basilary artery.

pump or can be sucked from the left atrium, left ventricle or organized thrombus laying over the aortic valve.

We tried to understand the risk factors of this new technology and possible neurological adverse events when our patient admitted to hospital with severe occipital pain and motor aphasia. Further imaging studies demonstrated that the diagnosis was extracranial right vertebral artery dissection.

Inamasu and Guiot reviewed the literature between 1990 and 2004 and reported that main mechanism of injury with vertebral artery dissection was distraction/extension, distraction/flexion and lateral flexion injuries.[5] We questioned the patient about any blunt injury that occurred before these symptoms. She mentioned that she had physical therapy, and an aggressive massage over her neck due to musculoskeletal pain. Both neurologic event episodes occurred after this massage done in physical rehabilitation center.

Extracranial artery dissection is usually diagnosed using MRI T_1-suppressed axial images. Since our patient had CRT-D implantation, she could not have an MRI. This delayed the diagnosis, and we had to do several CT angiographic studies to see the cranial arterial circle.

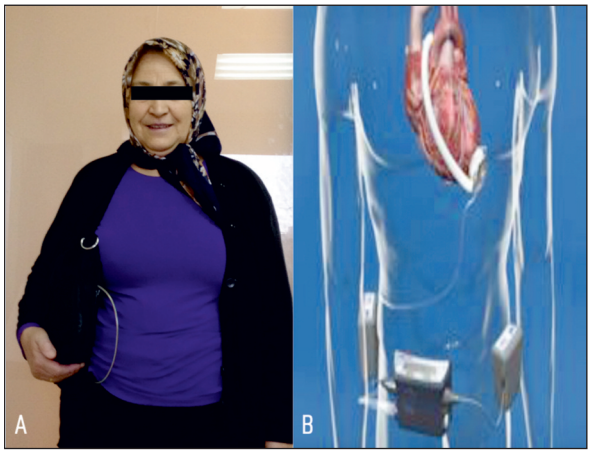

FIGURE 4: (A) The patient with the left ventricular assist device in our outpatient clinic, 6th months after the operation. **(B)** Mock model of HeartWare left ventricular assist device.

In the literature, there are no current emergency guidelines available for the management of patients with vertebral artery dissection. This may delay the diagnosis and treatment of these patients before a possible ischemic stroke occurs.[6] Generally accepted treatment modality is anti-coagulation or anti-platelet therapy. Our patient was receiving anti-platelet and anti-coagulation therapy due to LVAD implantation, her INR level was 2.5-3 when she admitted to emergency unit.

Another treatment modality is endovascular methods, associated with lower complication rates allowing reconstitution of the vessel lumen with immediate re-establishment of blood flow and treatment of the pseudoaneursyms in selected cases.[4]

In our case, endovascular stenting of the extracranial right vertebral artery was not an option, since we documented a flow defect in V_4 segment of right vertebral artery, and blood supply of the right PICA was maintained retrogradely from the left side through the vertebrobasiller junction. Any invasive intervention could effect PICA flow and cause infarct.

We report the first advanced heart failure case supported with LVAD and diagnosed with right vertebral artery dissection.

CONCLUSION

When a LVAD patient admits to emergency department complaining of head and neck pain, motor aphasia and dysarthria or focal neurological signs and symptoms, a possible pump thrombus or ischemic and hemorrhagic stroke may be considered due to LVAD physiology. An extracranial artery dissection should be suspected because early diagnosis and initiation of therapy will prevent the progression of dissection and progression of infarct.

Conflict of Interest

Authors declared no conflict of interest or financial support.

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