Iatrogenic occlusion of bilateral jugular veins, subclavian vein, and superior vena cava after repeated jugular cannulation associated with Arnold-Chiari malformation: Successful endovascular treatment

Przemyslaw Nowakowski, MD, PhD,[a,b] Piotr Buszman, MD, PhD,[c,d] Stefan Kiesz, MD, PhD,[e] *and* Pawel Buszman, MD, PhD,[c] *Chrzanow, Katowice, and Krakow, Poland; and San Antonio, Tex*

ABSTRACT

An Arnold-Chiari malformation is a congenital central nervous system defect. Raised intracranial pressure is commonly observed, and posterior decompression neurosurgery is the treatment of choice. We describe a patient with iatrogenic occlusion of bilateral jugular veins, subclavian vein, and superior vena cava resulting from repeated central venous cannulations. Because of venous hypertension, the patient suffered from neurologic symptoms: headaches, vision disturbances, and marked head edema. Two stents were used to recanalize the right internal jugular vein and superior vena cava. Symptoms subsided, and the patient returned to work. During 24-month follow-up, stents were patent. The patient remains symptom free and continues working. (J Vasc Surg Cases and Innovative Techniques 2020;6:18-20.)

Keywords: Vena cava obstruction; Endovascular treatment; Stenting

An Arnold-Chiari malformation is a congenital defect in which the cerebellum structures are displaced into the upper spinal canal. A diagnosis is based on the patient's history, careful observation, and neuroimaging (computed tomography [CT] or magnetic resonance imaging), which provides detailed insight into brain structures and especially the cerebellum. Typically, symptoms exhibited by patients suffering from Arnold-Chiari malformation are a consequence of structural congenital defects in the brain and spinal cord. Neurosurgery, the only treatment, is aimed at elimination of the cause; it consists of a posterior fossa decompression, most frequently Pudenz ventriculoperitoneal or ventriculoatrial shunt placement, resulting in a decrease of intracranial pressure and symptom alleviation.

Because of repeated central venous cannulation, patients with Arnold-Chiari malformations are at risk for complications that may lead to occlusion of the jugular veins, subclavian veins, or superior vena cava (SVC). This iatrogenic disease of the venous system caused our patient to develop symptoms suggestive of intracranial hypertension. We obtained the patient's consent for the publication of clinical data.

CASE REPORT

A 37-year-old man had been diagnosed with Arnold-Chiari I in childhood. The most common symptoms of type I Arnold-Chiari malformation are mild hydrocephalus, displacement of the cerebral tonsils toward the foramen magnum, vertigo, paresis, cranial nerve palsy, problems with balance, poor coordination, urinary incontinence, and sleep apnea. Coughing or sneezing may lead to syncope. In this case, intracranial hypertension resulted from extracranial venous system occlusion because in the course of neurologic and neurosurgical interventions, he had several central catheters placed with resultant occlusion of both internal jugular veins, subclavian veins, left external jugular vein, and SVC. Twenty years before presentation to our department, the patient had received a ventriculoatrial shunt, which after 7 years was replaced with a ventriculoperitoneal shunt. He had also undergone meningioma resection. Despite a well-functioning shunt, severe neurologic symptoms (ie, headaches, head edema, and visual impairment) recurred about a year preceding the treatment. This ultimately led him to visit the neurosurgery outpatient clinic. Following consultation and confirmation of proper shunt function, the patient was admitted to the vascular surgery department for further evaluation including CT angiography with venous phase imaging.

Reports from previous investigations, including 128-slice CT angiography, were thoroughly analyzed. Considering difficulties in interpreting the CT angiograms (slow outflow of contrast material from the head and upper body), venography was performed with administration of contrast material into the aortic arch using left radial artery access to avoid puncture of the jugular veins before the ultimate decision regarding management

From the Department of Vascular Surgery, American Heart of Poland, Chrzanow[a]; the Katowice University of Technology,[b] and the Center for Research and Development, American Heart of Poland,[c] Katowice; the Andrzej Frycz-Modrzewski Krakow University, Krakow[d]; and the San Antonio Heart and Endovascular Institute, San Antonio.[e]

Author conflict of interest: none.

Presented in the poster session of TCT 2018, San Diego, Calif, September 21-25, 2018.

Correspondence: Przemyslaw Nowakowski, MD, PhD, Topolowa 16, 32-500 Chrzanow, Poland (e-mail: nowakowski.mcsn@gmail.com).

The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2468-4287

https://doi.org/10.1016/j.jvscit.2019.09.006

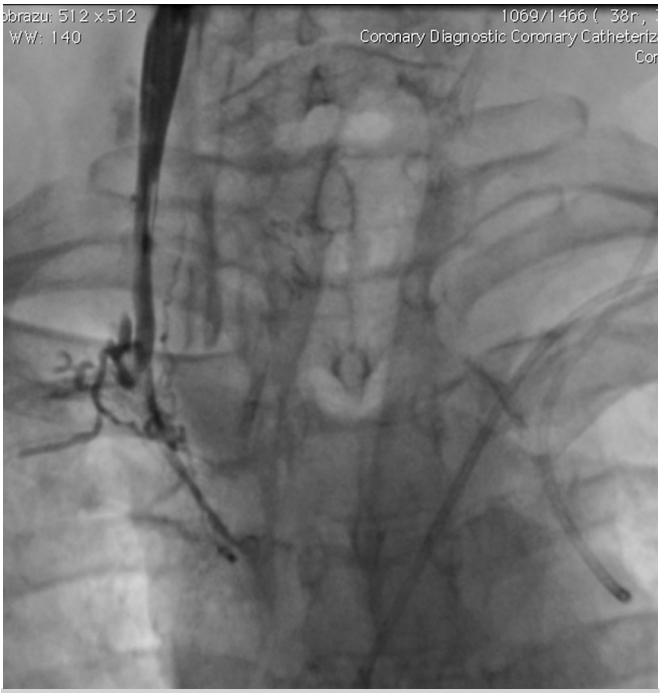

Fig 1. Patent part of right jugular vein.

strategy. Contrast material was also injected into the markedly dilated azygos vein through a right femoral vein access. The findings helped resolve doubts concerning previously performed CT angiography. The venograms showed occlusion of the SVC, both subclavian veins, left external and internal jugular veins, and right internal jugular vein down to the level of the thyroid veins (Fig 1). Venous drainage from the brain was by a collateral pathway through the still patent right external jugular and the patent segment of the right internal jugular vein.

The patient was considered eligible for attempted endovascular treatment. Three vessels were considered as access vessels—the left radial artery, the right femoral vein, and the patent segment of the right internal jugular vein. A 7°F introducer was inserted into the femoral vein, a 6F introducer into the right internal jugular vein, a 6F introducer into the left radial artery, and a pigtail catheter into the aortic arch. An intra-arterial bolus of 5000 units of heparin was administered during the interventional procedure; the patient had been receiving long-term therapy with acetylsalicylic acid.

Hydrophilic PT^2 (Boston Scientific, Marlborough, Mass), Pilot (Abbott Vascular, Abbott Park, Ill), and AqWire (Ev3) guidewires were used. After unsuccessful attempts at recanalization through the azygos vein, a hydrophilic guidewire was navigated across the right internal jugular vein occlusion and SVC to reach the area of the right atrium. After it was confirmed that the guidewire tip was in the right atrium, predilation was performed with a 4-mm balloon catheter. Contrast-enhanced scans showed venous drainage from the right jugular vein to the SVC and right atrium. A dedicated venous stent (Zilver Vena, 14 × 100 mm; Cook Medical, Bloomington, Ind) was then inserted into the SVC and right jugular vein. In addition, Wallstent (8 × 40 mm; Boston Scientific) was inserted into the distal segment of the jugular vein. Consecutive

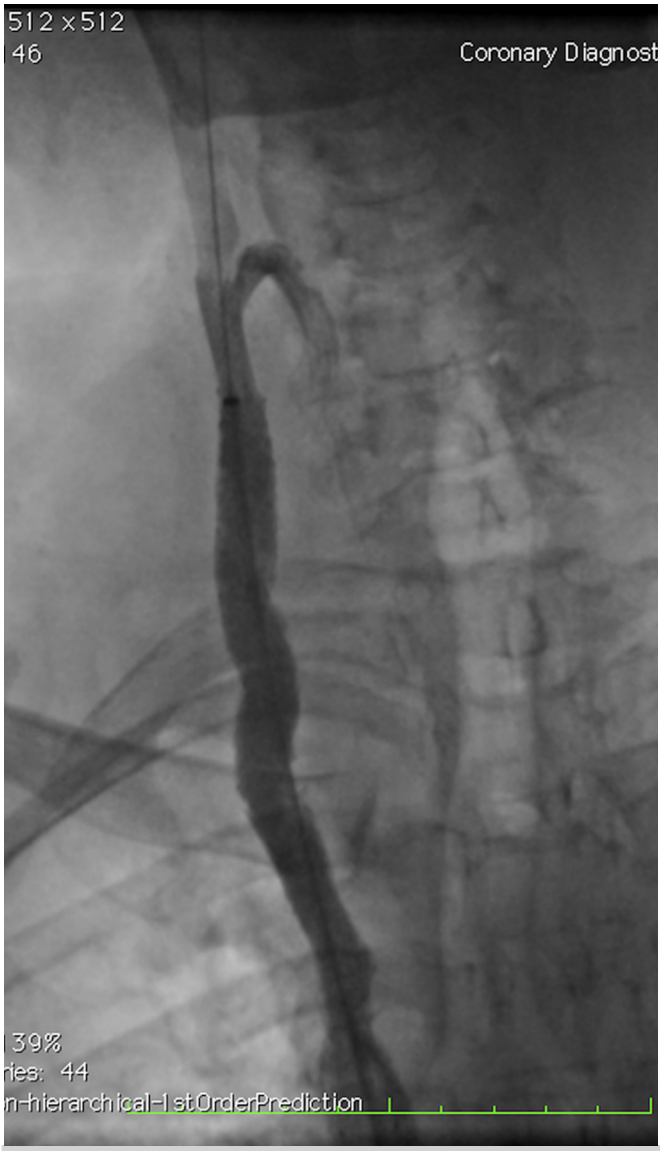

Fig 2. Final result after recanalization and stenting.

postdilations were carried out with 8-, 10-, and 12-mm catheter balloons. A checkup examination at the end of the procedure confirmed that both stents were in the correct position; adequate venous outflow from the head was also seen (Fig 2). The postinterventional course was uncomplicated; the patient's condition improved, head edema decreased, and he was discharged on postoperative day 3.

During the 30-day observation period, the patient was prescribed therapeutic doses of low-molecular-weight heparin. He also received dual antiplatelet therapy of aspirin and clopidogrel.

A follow-up appointment on day 30 revealed good general condition and subsidence of neurologic symptoms and head edema. Color duplex Doppler ultrasound confirmed venous flow pattern through both stents. The antithrombotic regimen was replaced with dabigatran 110 mg plus aspirin 75 mg daily.

The patient continued follow-up and support from a neurologist and vascular surgeon and returned to work. At 1 year after

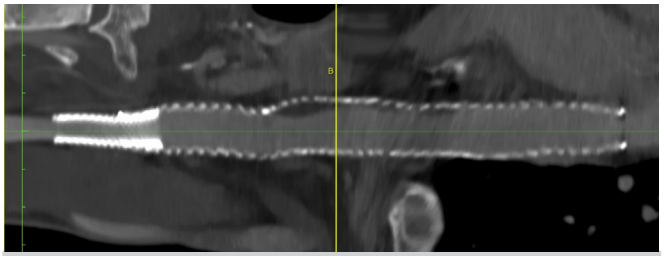

Fig 3. Control computed tomography (CT) angiography scan at 2-year follow-up.

the treatment, he underwent CT angiography, which revealed patency of both stents and satisfactory venous outflow from the head; no stent displacement was found. Color duplex Doppler ultrasound and CT angiography (Fig 3) performed at 24 months after the procedure confirmed stent patency and increased venous flow velocity; no stent thrombosis was visualized. During 24-month follow-up, the patient continued working, did not report any neurologic symptoms, and continued pharmacotherapy as specified before. No complications of anticoagulation treatment were observed.

DISCUSSION

Our patient with an Arnold-Chiari malformation is not among those typically treated with endovascular interventions. The need for endovascular treatment was associated with symptoms of iatrogenic occlusion of the jugular and subclavian veins as well as SVC occlusion revealed on history taking and resulting from multiple intravenous cannulations. The symptoms placed an enormous burden on the patient's everyday life, excluding him from work and causing severe depression. An attempt at endovascular treatment of intracranial hypertension resulting from stenosis or occlusion of the venous system was based on widely available publications regarding the efficacy of such treatment strategies.[1-4]

Because no clear recommendations are available regarding covered and uncovered stent placement, we decided to insert an uncovered Zilver Vena, a dedicated venous stent, at the SVC recanalization site. Although Haddad et al[5] suggested higher effectiveness of covered stents, they also emphasized the need for further studies in this area. Polytetrafluoroethylene-covered self-expanding or balloon-expandable stents definitely prevent passage of thrombi through the mesh. However, uncovered stents are less rigid and less thrombogenic; they allow better adaptation to vessel anatomy. The increasing use of dedicated venous stents might improve treatment outcomes. The risk of the procedure was considered low as major post-stenting complications (SVC rupture, mediastinal hemorrhage, cardiac tamponade, or pulmonary embolism) are rare. Other complications include chest pain, hemoptysis, and approach-related complications. SVC stent migration into the right atrium is another serious issue; its prevalence may be limited by proper device size and positioning. The most frequent long-term complications are stent thrombosis and in-stent restenosis resulting from thrombus formation or external compression. Fortunately, repeated endovascular treatment is possible.[6,7]

There is no consensus with respect to effective anticoagulant therapy for prevention of stent thrombosis after SVC recanalization. Oral anticoagulants or antiplatelet agents are typically used for several months. No comparative studies are yet available to analyze both strategies in this group of patients.[6-9]

The antithrombotic regimen used in our patient during a 30-day follow-up (ie, low-molecular-weight heparin and dual antiplatelet therapy) resulted from our experience in endovascular treatment of arterial disease and thrombosis prevention. Aggressive management was associated with the extent of recanalization and stent length exceeding 120 mm. Long-term antithrombotic therapy (dabigatran 110 mg and aspirin 75 mg daily) seems to have been an effective strategy in our patient.

CONCLUSIONS

Considering literature reports indicating relatively low complication rates after endovascular treatment of iatrogenic and post-thrombotic venous occlusion, we decided to use an endovascular intervention. Early and long-term effects were consistent with literature data; hence, it was concluded that endovascular procedures could be considered the treatment of choice in patients with structural defects and associated conditions.

REFERENCES

1. Zhou D, Meng R, Zhang X, Guo L, Li S, Wu W, et al. Intracranial hypertension induced by internal jugular vein stenosis can be resolved by stenting. Eur J Neurol 2018;25:365-e13.
2. Yim CD, Sane SS, Bjarnason H. Superior vena cava stenting. Radiol Clin North Am 2000;38:409-24.
3. Rachapalli V, Boucher LM. Superior vena cava syndrome: role of the interventionalist. Can Assoc Radiol J 2014;65:168-76.
4. Breault S, Doenz F, Jouannic AM, Qanadli SD. Percutaneous endovascular management of chronic superior vena cava syndrome of benign causes: long-term follow-up. Eur Radiol 2017;27:97-104.
5. Haddad MM, Simmons B, McPhail IR, Kalra M, Neisen MJ, Johnson MP, et al. Comparison of covered versus uncovered stents for benign superior vena cava (SVC) obstruction. Cardiovasc Intervent Radiol 2018;41:712-7.
6. Kuo TT, Chen PL, Shih CC, Chen IM. Endovascular stenting for end-stage lung cancer patients with superior vena cava syndrome post first-line treatments—a single-center experience and literature review. J Chin Med Assoc 2017;80:482-6.
7. Warner P, Uberoi R. Superior vena cava stenting in the 21st century. Postgrad Med J 2013;89:224-30.
8. Ji D, Gill AE, Ermentrout RM, Hawkins CM. Thrombogenic superior vena cava syndrome from long-standing central venous access in a 5-year-old patient treated with balloon-expandable stents. J Radiol Case Rep 2018;12:15-22.
9. Scalese MJ, Hayes SL, Lott S. Antithrombotic therapy post endovascular stenting for superior vena cava syndrome. Hosp Pharm 2017;52:666.

Submitted Feb 15, 2019; accepted Sep 30, 2019.