Dural Arteriovenous Fistula Caused by Jugular Vein Stenosis
—Case Report—

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Abstract

A 32-year-old female presented with a dural arteriovenous fistula in the transverse-sigmoid sinus caused by stenosis of the left internal jugular vein. The feeding arteries were embolized, resulting in nearly complete disappearance of the fistula. This case supports the idea that dural arteriovenous fistula is an acquired lesion caused by intravenous hypertension.

Key words: dural arteriovenous fistula, jugular vein stenosis, venous hypertension

Introduction

Dural arteriovenous fistula (DAVF) in the transverse-sigmoid sinus may be an acquired lesion developing from preexisting dural sinus or venous thrombosis. Venous thrombosis may cause a fistula by opening physiological shunts in the dura mater, which divert blood flow into the cortical veins, reduce the increased intracranial pressure, and stop the spread of thrombosis. DAVF development after trauma or surgery may be secondary to sinus thrombosis or thrombophlebitis. However, some patients with DAVF have only had stenosis or irregularity of the dural sinus without preexisting venous thrombosis. These findings suggest that venous hypertension caused by sinus abnormality may trigger the development of acquired DAVF. We describe a patient with a DAVF in the transverse-sigmoid sinus associated with stenosis of the ipsilateral internal jugular vein.

Case Report

A 32-year-old female was admitted to our department with headache and pulsatile tinnitus in January 1996. She had suffered from amenorrhea since her birth and had been regularly receiving hormonal therapy (human menopausal gonadotrophin; Humegon©; Sankyo, Tokyo) prescribed by her gynecologist for 6 years. She complained of pulsatile tinnitus and headache in the left retroauricular region. These symptoms had become most prominent during the week prior to admission. There was no history of trauma, infection, or surgery.

Physical examination found she was obese, but without hypogonadism. Neurological examination revealed no abnormality. Endocrinological investigation demonstrated normal levels of the pituitary hormones. No left retroauricular bruit was heard, although the patient was aware of a pulsatile murmur. Magnetic resonance (MR) imaging of the head revealed an isointense mass containing a hypointense portion, which corresponded to calcification in the suprasellar region. These findings suggested a hypothalamic tumor indenting the third ventricle. MR imaging demonstrated no other abnormal lesions. The most likely diagnosis was a suprasellar hamartoma because no growth of the mass had occurred in the last 6 years based on comparison of the MR images with those taken 6 years previously. Left carotid angiography demonstrated a DAVF fed by the temporosquamous branches of the left middle meningeal artery, the left occipital artery, and left ascending pharyngeal artery and drained mainly into the junction of the left transverse and sigmoid
The left transverse and sigmoid sinuses were well visualized without the finding of dural venous sinus thrombosis. The left internal jugular vein from the jugular bulb was poorly visualized, and was tapered. These findings suggested internal jugular vein stenosis. Most venous outflow reflux to the right internal jugular vein was presumably retrograde (Fig. 2). Stenosis of the left jugular vein was confirmed by phlebography.

The temporosquamous branches of the left middle meningeal artery, left occipital artery, and left ascending pharyngeal artery were selectively catheterized and embolized with polyethylene vinyl alcohol, resulting in nearly complete disappearance of the DAVF. She was free from pulsatile tinnitus after the embolization.

**Discussion**

The hypothesis that venous hypertension due to sinus abnormality triggers the development of acquired DAVF can explain the pathogenesis of DAVF, whether associated with dural sinus thrombosis or not. There have been few clinical cases supporting this idea. Chronic venous hypertension was created in a rat model by surgical anastomosis of the carotid artery to a proximal jugular vein occlusion,
suggesting that only venous hypertension without thrombosis could cause development of new acquired DAVF. The capillaries in the sinus or venous wall are similar to the vasa vasorum, and venules connect the capillaries to the sinus and are very short. Therefore, increased venous hypertension in the dural sinus or vein would be transmitted directly to the capillaries and arterioles. Exposure of these vessels to venous hypertension via a retrograde route would lead to development of new DAVF. Hemodynamic and angiogenetic factors during venous hypertension may also promote formation of DAVF. However, the presence of venous hypertension was not confirmed in previous cases. In our case, angiograms demonstrated that stenosis of the left jugular vein in the midcervical region was not associated with dural sinus thrombosis in the transverse-sigmoid sinus, and most venous outflow reflux to the right internal jugular vein was retrograde. Based on the angiographical findings, we judged the DAVF in the present case was caused by venous hypertension due to stenosis of the left internal jugular vein.

The causes of dural sinus thrombosis include trauma, surgery, oral contraceptives, coagulation disorders, and dehydration. In our case, the patient took hormonal drug (Humegon®) for 6 years because of infertility, which might have been affected by the suprasellar neoplasm. We speculate that the hormonal drugs induced stenosis of the left internal jugular vein, resulting in formation of the DAVF.

Embolization of the feeding arteries and the affected sinus, and surgical procedures including excision and reconstruction of the obstructed venous sinus, are recommended for the treatment of DAVF, but we believe that the intravenous hypertension should be corrected. Hypertension in patients with DAVFs is thought to be caused by both arterial inflow from the feeding arteries and obstruction of the venous outflow. Nowadays it is known that it is difficult to cure DAVFs completely only using transarterial embolization. We suggest that the combined treatments of embolization of the feeding arteries and the affected sinus, reconstruction of the affected sinus or vein, and surgical excision to change the pathological condition of DAVFs should be performed.

References


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