

Case report: Unilateral conduction hearing loss due to central venous occlusion

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ABSTRACT

Central venous stenosis is a well-known complication in patients with vascular access for hemodialysis. We report two cases involving patients on hemodialysis with arteriovenous fistulas who developed reversible unilateral conductive hearing loss secondary to critical stenosis of central veins draining the arteriovenous dialysis access. A proposed mechanism for the patients' reversible unilateral hearing loss is pterygoid venous plexus congestion leading to decreased Eustachian tube patency. Endovascular therapy was conducted to treat the stenosis and the hearing loss of both patients was returned to near normal after successful central venous angioplasty.

Keywords: Deafness, ESRD, Eustachian tube, Pterygoid plexus, Vascular access stenosis

Introduction

In current literature, central vein disease (CVD) is defined as greater than 50% narrowing of the thoracic central veins. These veins include the superior vena cava (SVC), brachiocephalic (BCV) and subclavian (SCV) (1). The incidence of CVD has been reported to be as high as 23% in the total dialysis population and 41% in those with access-related complaints (2). Many fistulas do not sustain dialysis due to poor maturation, thrombosis, or critical stenosis (3, 4). Central venous stenosis and occlusion are a result of high vascular wall shear stress due to the presence of arteriovenous fistula (AVF) or arteriovenous graft (AVG) upstream and vascular compression or distortion frequently seen in aging and due to previously placed central venous catheters (5). Once critical stenosis has developed patients are at risk for central venous occlusion, which can result in face, neck, or arm swelling, along with other severe disabling complications (6, 7).

After careful literature review, there is a paucity of data regarding the development of reversible unilateral conductive hearing loss secondary to critical stenosis of venous

access. This disabling symptom of central venous occlusion has gone under recognized. We present what we believe are the first two case reports of reversible unilateral conduction hearing loss due to central venous occlusion. Both of these patients were on hemodialysis and hearing returned to near normal after central venous angioplasty.

Case report

Case #1

Our first case is of a 66-year-old male with end-stage renal disease (ESRD) on hemodialysis for the past eight years. His vascular access was a left upper-arm brachiocephalic AVF. He presented to our vascular access center with left arm swelling. He also complained of suffering spasmodic hearing loss from his left ear for the past five months. On further inspection, we noticed a wick of cotton in the patient's left ear that he places before sleeping because he hears a continuous, loud streaming noise. Physical examination showed a left upper arm brachiocephalic fistula that was hyper-pulsatile on palpation and did not collapse on raising the arm. The patient was taken for an angiogram of his AVF, which showed a 90% stenosis of the left innominate vein. He underwent successful venous angioplasty with a 14 mm x 40 mm atlas venous angioplasty balloon to 10% residual. Patient had significant improvement in left arm swelling, and most noticeably had significant improvement in hearing immediately following angioplasty in the recovery area.

Case #2

Our second case is of a 71-year-old female with past medical history of hypertension, type II diabetes mellitus, and

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ESRD, who had been on hemodialysis for the past two years. Her vascular access was a left upper-arm brachiocephalic AVF. She presented to our vascular access center with left-arm and left-sided facial swelling. She also complained of gradual left-sided hearing loss for the past two months. Physical examination revealed a left upper-arm brachiocephalic fistula that was hyper-pulsatile on palpation and did not collapse upon raising the arm. The patient was taken for an angiogram of her AVF, which showed an 80% stenosis of the left innominate vein. She underwent angioplasty with a 14 mm × 40 mm atlas venous angioplasty balloon to 20% residual. Immediately post-procedure she had significant improvement in her hearing. She has since returned to our access center three times with similar complaints of arm swelling and hearing loss both of which resolve after central venous angioplasty.

Discussion

Unilateral reversible conductive hearing loss is an uncommon but under-recognized consequence of central venous occlusion. The exact mechanism of deafness, although not known, is likely due to swelling and congestion of the Eustachian tubes (ETs).

The ET is a narrow tube that connects the middle ear to the back of the nose. Blockage of the ET isolates the middle ear space from the outside environment. The lining of the middle ear absorbs the trapped air and creates a negative pressure that pulls the eardrum inward. When it becomes stretched inward, patients often experience pain, pressure, and hearing loss. Long-term blockage of the ET leads to the accumulation of fluid in the middle ear space that further increases the pressure and hearing loss (8).

There are several muscles that allow for opening of the ET. The principal and perhaps only dilator of the tube is the tensor veli palatini (TVP) (9). The TVP functions to tighten the anterior part of the soft palate and assist in opening the pharyngotympanic tube. Studies have shown that abnormal insertion of the TVP into the cartilage of the ET was found to cause a functional obstruction that led to an increase in incidence of otitis media. This anatomical relationship between the TVP and the ET is well described in literature.

Concurrently, the pterygoid plexus is the main venous drainage for the ET (9). The proposed mechanism for our patients' reversible unilateral hearing loss is congestion of the pterygoid venous plexus (PVP) leading to decreased ET patency. The study of Oshima et al (10) used MRI to evaluate the anatomical relationship of the muscles of the neck and the PVP before and after neck compression. The results showed that the lateral pterygoid muscle became enlarged after neck compression. Simultaneously, the volume of the PVP observed between the medial pterygoid muscle and TVP muscle was increased. The increased volume of the PVP led

to protrusion of the ET anterior wall to the luminal side, and thus decreased ET patency (10). Given this anatomical relationship, we postulate that in our patients, central vein occlusion led to PVP congestion, which in turn led to compression of the ET.

The loss of ET patency is what likely contributed to our patients' unilateral hearing loss. After adequate venous outflow was restored with angioplasty, our patients' hearing was restored. These symptoms can be quite disabling to patients and significantly affect their quality of life. Timely recognition of the cause and treatment can reverse deafness, improve patient's quality of life, and prevent disability from becoming permanent.

Disclosures

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