Duplication of the common carotid artery and the ipsilateral vertebral artery with a fenestration of the contralateral common carotid artery

Sir,

Fenestration and duplication of the cervical arteries are considered to be extremely rare anatomic variants. Difficulties can arise when it comes to diagnosing the pathological significance of anomalies of the cervical arteries using imaging procedures. It is important to distinguish between congenital anatomic variants such as duplication and fenestration and acquired anomalies such as dissections, aneurysms and pseudofenestration. While the latter implicate a specific and partially extensive therapy, duplications are coincidental findings with no pathological significance.

In a 61-year-old female patient suffering from unexplained dizziness we incidentally observed extremely rare anatomic variants of the cervical arteries. The cervical magnetic resonance (MR) angiography revealed an asymptomatic duplication of the right common carotid artery (CCA) as well as an extracranial duplication of the ipsilateral vertebral artery [Figure 1]. Furthermore, a fenestration of the contralateral CCA was found [Figures 2A, B].
In describing anomalies of cervical arteries, the terms duplication and fenestration are to be distinguished clearly, even though they are incorrectly used as synonyms.[1] Arterial duplication is defined as the persistence of a doubly constructed arterial vessel, originally formed in the embryonic phase. This vessel has two separate origins and converges to form one main trunk.

In the case of a duplicate vertebral artery, there are in general two outlets from the subclavian artery which then reunitie to a single vessel. A faulty degeneration of the primitive dorsal aorta and two intersegmental arteries is considered to be responsible for the development of duplicate vertebral arteries.[3] The common carotid artery is formed in the fourth and fifth week of embryonic development from parts of the ventral aorta. Disturbances in this period are therefore considered to constitute the trigger for duplication.[4]

Duplications are generally rare and, as in the case of our female patient, for the most part incidentally discovered in the course of a cervical cranial MR angiography, a digital subtraction angiography (DSA), computed tomography-angiography (CTA) or an autopsy. On account of their normal wall structure, artery duplications are not linked to an increased predisposition to pathological processes in their supply function.[5] Arterial fenestration on the other hand is to be understood as a short arterial segment with two patent lumens, a single vessel origin and one main trunk into which the two lumen converge. Embryonic misdevelopments are also considered to be responsible for the formation of fenestrations.

Fenestrations are of significance in so far as histopathological examinations frequently reveal irregularities in the wall structure of the vessels, in particular in connection with the tunica media.[5] These irregularities could increase the risk of subsequent problems in those areas supplied by the respective arteries (thromboses, embolisms) or be accompanied by other vessel pathologies (aneurysms).[4]

In the context of neuroradiological diagnosis, knowledge concerning potential congenital anatomic anomalies in vessels supplying the brain is essential, in order that these may be distinguished from acquired pathological arterial changes such as aneurysms, dissections and pseudofenestrations.[2]

In the case described above, incorrectly diagnosing a dissection would have led to therapeutic treatment in the form of, for example, anticoagulation.

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REFERENCES
Polyradiculopathy is a rare but potentially devastating syndrome more common in Acquired Immunodeficiency Syndrome (AIDS). Although cytomegalovirus (CMV) is most commonly implicated, the differential diagnosis includes tuberculosis, syphilis, and lymphoma. We admitted a 36-year-old army truck driver from Haryana, India, HIV-positive since May 2004, with ascending, asymmetric flaccid paraplegia, lower extremity paraesthesia, and bladder and bowel retention since three months. His CD4 count was 4 cells/µl. He received intravenous (IV) immunoglobulin, oral Gancyclovir and highly active anti-retroviral therapy (HAART) comprising Zidovudine, Lamivudine, and Nevirapine, without improvement.

On examination, all muscles of the lower extremity were hypotonic, wasted, with 0/5 strength, and absent deep tendon reflexes. Bulbocavernous and anal reflexes were absent. Light and deep touch sensations, nociception, proprioception, and vibration sense was all diminished bilaterally extending superiorly to approximately the L1 dermatome.

Cerebrospinal fluid (CSF) showed 40 polymorphonuclear leukocytes per µl, 336 mg/dL protein, 90 mg/dL glucose (CSF : serum 62%), and negative on gram, India ink, and acid-fast bacilli staining. Suspecting polyradiculopathy due to cytomegalovirus or tuberculosis (TB), both intravenous Gancyclovir and anti-tuberculosis treatment (ATT) were initiated along with HAART. Magnetic resonance imaging (MRI) of the spinal cord showed confluent arachnoiditis of the cauda equine [Figure 1A]. CSF was sterile and CSF polymerase chain reaction (PCR) for mycobacterium and CMV were negative. Anti-CMV IgG was strongly positive in serum; blood testing for anti-CMV IgM, anti-HSV IgG/IgM, VDRL for syphilis, anti-toxoplasma IgG/IgM, HBsAg, and anti-HCV were all negative; CSF for anti-CMV IgM/IgG, anti-HSV IgG/IgM, VDRL, and PCR for TB and CMV were negative.

The patient gradually improved, and a repeat MRI after one month showed marked improvement in the confluent nerve roots [Figure 1B]. At three months follow-up, the patient was taken off Gancyclovir, and had recovered >50% of motor, sensory, and bladder and bowel function. He became ambulatory and tendon reflexes normalized. The final clinical impression was that of AIDS-related CMV polyradiculopathy that failed to respond to oral Gancyclovir but responded well to IV Gancyclovir and HAART.

Polyradiculopathy in AIDS is a rare but devastating syndrome. This report, to the best of our knowledge, is the first such report from the Indian subcontinent. The differential diagnosis includes CMV, tuberculosis, Varicella zoster virus, Ebstein Barr virus, Herpes simplex virus, syphilis, and lymphoma. CMV causes a subacute, progressive, asymmetric ascending paraplegia with urinary retention and sacral dysesthesias. Initial CSF examination in CMV may reveal the “classic” pattern of polymorphonuclear pleocytosis, elevated protein, and decreased glucose in only 50%. Nucleic acid amplification techniques are often rapid, sensitive, and specific, though expensive. In our case PCR for CMV was negative in CSF; although this test generally has sensitivity greater than 80%, prior treatment decrease yield. Detection of CMV antigens (pp65) in CSF has a sensitivity of 91% and specificity approaching 100%.

Histopathological examination of the cauda equina roots shows polymorphonuclear or mononuclear...