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Commentary

Evaluation of Pain in Vertebral Artery Dissection

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Commentary

Vertebral Artery Dissection (VAD) is an important cause of stroke in the young adult frequently under diagnosed, as symptoms are various and subtle in their early manifestation [1]. VAD might be asymptomatic at onset in almost 50% of cases, but when symptoms are present, they mainly consist in local posterior neck pain and occipital cephalalgia, associated or not to other neurological symptoms ranging from cranial nerves deficit, to dizziness, postural instability, syncope, and peripheral neurological deficit [1-3]. Following this misleading clinical presentation, a high risk of early cerebrovascular complications (e.g., transient ischemic attacks, and stroke) is reported [4-6], probably due to an artery-to-artery mechanism [7]. The risk of stroke after VAD is higher during the first 2 weeks following the dissection, and then becoming significantly decreased [8]. As a consequence, the early diagnosis of VAD is essential to correctly address the therapeutic strategies, minimizing the complications [9]. This early diagnostic phase based on rapid recognition of symptoms (when present) is crucial, particularly if we consider VAD as a condition asymptomatic at presentation, becoming symptomatic later [1].

The term "stroke chameleons" is currently employed to describe a heterogeneous group of conditions that do not initially appear to be due to cerebrovascular accidents [10]; however, they are found to be strokes after additional investigation [11-16]. Almost 1% of strokes might clinically manifest as a peripheral neurological deficit [12]. In this context, VAD represents not only a well-known cause of an acute and persistent headache or cervical pain, but it has been also described as a cause of isolated radiculopathy [2,17,18]. According to the available literature, on average, neurological symptoms develop 4 days to 5 days after cervical pain onset, involving most frequently the C5 tract. Sensory impairment has been also occasionally reported. Predisposing conditions might be genetic connective tissue pathologies, history of minor trauma, chiropractic manipulations, and forceful neck positions [4,6].

In a recent article [2], we have described a case of VAD, underlying the diagnostic workout that led us to diagnose a rare cause of a common symptom: an extracranial VAD compressing C5 root and causing acute cervicobrachial pain with arm weakness in a young lady. At first, we focused on the topographic diagnosis (radiculopathy *vs.* plexopathy or peripheral neuropathy), then to the etiological diagnosis. Cervical disc prolapse and stenosis is the primary concern when considering a patient with cervicobrachial pain. In the case we described the clinical history, the neurological examination, and the EMG exam led us to assume a C5 radiculopathy not caused by other traumatic conditions, excluding further neuropathies as tunnel carpal syndrome [2]. When examining pain-related features, all of them were slight atypical for a classic disc hernia, namely the young age at onset, the negative history of discopathies, the absent response to both NSAIDs and steroid therapy, the scarce modification of pain between the rest condition and neck movements, and finally the association with chiropractic manipulations. The cervical MRI with MR angiography of neck vessels confirmed the diagnosis, revealing a focal stenosis of the right vertebral artery. Fat saturation T1 weighted sequences showed a subacute C4- C5 hematoma causing compression on the emergent ipsilateral C5 nerve root. Antithrombotic therapy was promptly started, and one month later the patient became completely asymptomatic. The three-month follow-up MRI exam showed the whole disappearance of the hematoma.

There were two clinical pitfalls when dealing with the present case. First, when considering a continuous and persistent pain, with acute onset and no improvement after pharmacological therapy, neurologists should take into account also possible alternative diagnosis different from a common discopathy. Second, if neurologists would have been asked only for standard cervical MRI, no hematoma would have been detected, and the acute C5 motor deficit would have been erroneously interpreted as a manifestation of a cervical disc prolapse, also easily detectable in young patients. The hematoma can be displayed only with a fat saturation MRI scan that in our case was expressively asked by the neurologist.

We know that VAD is a well-known cause of an acute and persistent headache or cervical pain (IHS, 2013), but rarely it has been described also as a cause of isolated radiculopathy [17,18]. We suggest the importance to keep in mind VAD as a possible causal factor of "radiculopathy" in young individuals without a personal history of discopathy or with predisposing factors. The diagnosis of VAD is achieved with difficulty in a patient with isolated headache

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or cervical pain in the Department of Emergency, and at this purpose the International Classification of Headache Disorders-beta version (HIS, 2013), might be a valid tool for clinicians to improve diagnostic sensitivity, as previously suggested by Schytz et al. [18]. We finally recommend that Fat saturation MRI sequences should be performed in selected cases, to exclude cervical artery dissection as part of the clinical-diagnostic workup of a patient with suspected cervical radiculopathy, when standard MRI do not show any disc hernia or prolapse.

Future prospective studies on large samples of patients with VAD are warranted to better assess the clinical features and all the possible future therapeutic strategies specific for this disorder.

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