

CASE REPORT

OTONEUROLOGICAL MANIFESTATION IN SUBCLAVIAN STEAL SYNDROME

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ABSTRACT: -The subclavian steal syndrome is characterized by a subclavian artery stenosis located proximal to the origin of the vertebral artery. In this case, the subclavian artery steals reverse-flow blood from the vertebrobasilar artery circulation to supply the arm during exertion, resulting in vertebrobasilar insufficiency. As the vertebrobasilar arterial system feeds both the peripheral and central auditory and vestibular systems. In subclavian steal syndrome, neurotological symptoms are expected because of the vertebrobasilar insufficiency. In this report, we describe a patient suffering from subclavian steal syndrome, who presented with isolated dizziness, recurrent vertigo, on moving around, while sitting no complaints were there and no postural vertigo was seen. Upon magnetic resonance imaging no ischaemic lesions were observed, suggesting that the central auditory and vestibular system are more likely to be involved in the pathogenesis of neurotological symptoms in subclavian steal syndrome. Patients complaining of numbness of the upper arm and isolated neurotological symptoms should be thoroughly examined for subclavian steal syndrome. Furthermore, regular follow-up must be undertaken in order to prevent other neurological deficits in the vertebrobasilar arterial territory.

KEYWORDS: Vertigo, Sensorineural hearing loss, Subclavian steal syndrome, Vertebrobasilar insufficiency

INTRODUCTION: The subclavian steal syndrome (SSS) refers to a vascular disorder in which there is occlusion or stenosis of the subclavian artery proximal to the vertebral artery origin (which is the subclavian artery) leads to altered vascular haemodynamics resulting in retrograde blood flow in the ipsilateral vertebral artery toward the upper arm, distal to the subclavian artery narrowing, where decreased blood pressure had been established. Subclavian artery lesions are usually asymptomatic because of the abundant collateral blood supply in the head, neck, and shoulder. However, these lesions produce neurologic symptoms when compensatory flow to the subclavian artery from the vertebral artery diverts too much flow toward the arm and away from intra-cranial structures leading to vertebrobasilar insufficiency (VBI) ¹ for example during arm swinging. The most important collateral circulation to the posterior fossa is through the circle of Willis, principally through the posterior communicating artery. When this communication is absent or inadequate, possibly due to concurrent extracranial carotid stenosis, then vertebrobasilar symptoms manifest.

The rate of SSS is estimated at 1.3% (324 cases in 25,000 persons) in European patients referred for carotid and vertebral artery Doppler ultrasound, most frequently in Caucasians

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because of the increased incidence of atherosclerosis in this population. SSS generally occurs in patients > 55 years of age and has a 2:1 male-to-female ratio². Clinically, SSS may be suspected when the difference in blood pressure between the two arms exceeds 20 mmHg, but the diagnosis is essentially confirmed by Doppler ultrasound, although magnetic resonance imaging (MRI) with or without magnetic resonance angiography (MRA), computed tomography (CT) scan of the brain and digital subtraction angiography can also be used. Symptomatic patients require surgical intervention such as carotid-subclavian bypass, axillo-axillary bypass, or percutaneous transluminal angioplasty of the subclavian artery with stent placement. Thus, the goals of surgical treatment consist in restoration of the antegrade vertebral artery flow, alleviation of cerebral hypoperfusion, and improvement of arterial perfusion to the upper arm³. Moreover, aggressive management of risk factors, such as hypertension, diabetes and tobacco use, is also essential for successful treatment of this syndrome³.

The present report deals with a patient suffering from SSS, who was admitted to our unit on account of neurotological symptoms.

CASE REPORT: A 54-year-old male, admitted with complaint of , unsteadiness and instability especially while walking, which still persists to this day on examination left radial was feeble no nystagmus , no cerebellar signs , rhombergs sign was negative , symptoms increased on active movement of left hand. He had no auditory disturbance or tinnitus. As the pulsations were feeble on left radial artery, CT Angio and Digital subtraction angiography done which revealed SSS on the left side, stenosis at the origin of the left vertebral artery and stenosis of both the carotid bifurcations (fig. 1). MRI didn't show any ischaemic lesions in the area of the brainstem (Fig.2). Work up was done to rule out takayasu arteritis was normal, even angiography findings were not consistent with it. Patient underwent carotid-subclavian bypass , left radial pulsation improved , but still his giddiness didn't improved literature was searched which showed coexistence of neurotological symptoms associated with SSS, there are few case reports which have reported it .

At neurotological examination, when performing Unterberger testing, he deviated towards the left side., there was no spontaneous nystagmus, but in the position of Rose (supine position, head hanging down and rotated to the left, middle, right, with eyes closed), a positional vertical up-beating nystagmus was present . Caloric testing was normal on both sides.

DISCUSSION: In SSS, occlusion or marked stenosis are present, usually due to atherosclerosis, of either the subclavian or innominate artery proximal to the origin of the VA. The established difference of pressure between the subclavian and basilar artery may lead to a siphoning effect with blood flowing from the opposite VA across the basilar artery and down the VA on the occluded side¹. This results in a steal blood phenomenon at the expense of the basilar artery, as blood flow is reversed to the ipsilateral occlusion VA, which leads to a hypoperfusion state in the vertebrobasilar arterial (VBA) system and thus vertebrobasilar insufficiency (VBI).

SSS is classified as asymptomatic, oligosymptomatic, if only neurological symptoms or upper limb ischaemia are present and complete when both symptoms are found⁶. When symptomatic, the SSS can be manifest with a variety of VBI symptoms such as headache, blurred vision, diplopia, impairment of consciousness, dysarthria, and facial paraesthesiae. Neurotological symptoms, such as vertigo, are also mentioned in VBI and are considered to be of central origin⁷⁻⁸. Our patient suffering from SSS and thus from VBI presented with unsteadiness , recurrent vertigo. Although VBI is related to disturbed haemodynamics in the posterior

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cerebral circulation, neurotological symptoms may be isolated, without any other neurological signs of brainstem ischaemia ⁹.

In our case, a positional nystagmus was revealed. The positional nystagmus could be an isolated finding, in VBI, even if there were no other neurological signs ¹¹. Moreover, positional vertical down-beating nystagmus has already been described in VIB ¹²⁻¹³; positional vertical up-beating nystagmus is attributed to brainstem lesions located at the ponto-mesencephalic and ponto-medullary junctions ¹⁴; accordingly, in our case no ischaemic lesions were located in the low brainstem. Some studies have reported that the most common finding in VBI is the unilateral vestibular paresis to caloric testing ¹⁹. In our study, the caloric response reduced; it is currently accepted that caloric testing remains normal in central lesions ¹⁰. Abnormal oculomotor testing, such as smooth pursuit, is frequently seen in VBI patients ¹⁴, in our case, abnormal saccades were detected, indicating a central nervous system involvement.

In our patient, sensorineural hearing loss was demonstrated, unilateral in one and bilateral in the other. Unilateral hearing loss, attributable to VBI is not so frequent, estimated at approximately 20%, according to Yamasoba et al. ¹⁵ in a series of 70 patients suffering from VBI; bilateral, sudden sensorineural hearing loss, although rare, is also described in VBI, in isolation ¹⁶ or accompanied by neurological symptoms ¹⁷.

Since the VBA system feeds both the peripheral and central region of the auditory and vestibular systems, it is difficult, in SSS, to precisely localize the site of insult. The presence of positional vertical nystagmus and prolonged wave V and increased wave I-V latency interval in ABRs, the finding of abnormal saccades and the radiological evidence of cerebral ischaemic lesions support the hypothesis that, in SSS, the central auditory and vestibular system is implicated in the pathogenesis of neurotological symptoms.

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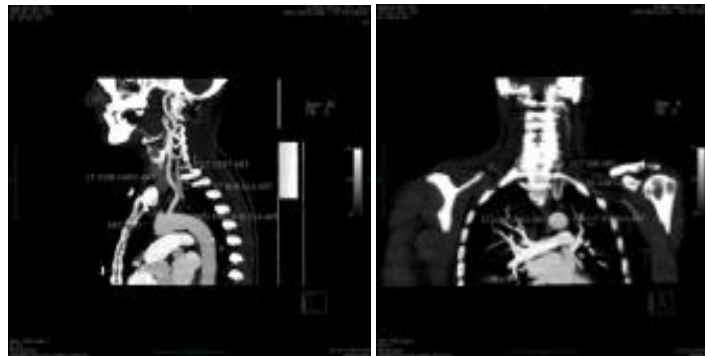


Fig 1a

fig 1b

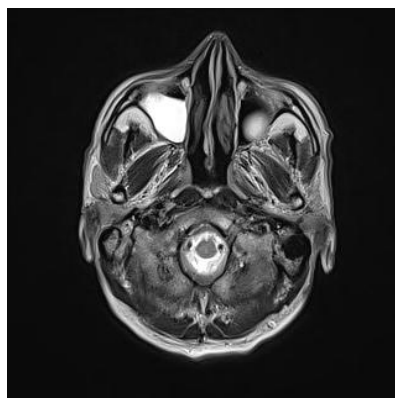


Fig. 2 Axial Gd-MRI scan of showing normal (no ischaemic lesions) brainstem.