Unusual Clinical Signs in Left Subclavian Artery Occlusion: Clinical and Angiographic Correlation

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SUMMARY A case of left subclavian steal syndrome with transient ischemic attacks of left carotid artery distribution is presented. An attempt to explain this uncommon symptomatology is based on a rare patent cervical arterial network, stealing blood from the left common carotid artery and supplying the distal portion of the obstructed left subclavian artery.

THE OCCURRENCE OF CEREBRAL INSUFFICIENCY in the subclavian steal syndrome is well known, and was described by several authors as mainly regarding the vertebrobasilar system.1-4 Later, Lord et al5 proved statistically that discontinuity of the circle of Willis is the principal factor activating these symptoms in this syndrome. On the other hand, the numerous clinical signs of the subclavian steal syndrome described up till now are thought to be due to additional coexistent atherosclerotic extra- and intracranial lesions in the same patient.6

The importance and variety of collaterals and their task in the hemodynamics of the subclavian steal syndrome was stressed in some reports.5-7 The role of the carotid system, which is included among these collaterals, has always been overshadowed by the well known vertebro-vertebral shunt.7 Thus, little is known about probable clinical signs that might occur when the carotid system acts as an unusual collateral pathway in that syndrome.

We therefore intend to discuss a case of left subclavian steal syndrome with presenting signs of transient ischemic attacks of the region supplied by the left internal carotid artery. We found it interesting to relate these symptoms to the internal carotid system as an unusual collateral network in this case, as well as with the absence of coexistent demonstrable atherosclerotic lesions of that artery and its branches.

References


Case Report

A right handed 52 year old man was examined in the emergency room because of difficulties of speech and right hemiparesis. As reported by his family, the onset was abrupt, about two hours prior to hospitalization.

The neurological examination revealed a fully alert athletic man, with hypotonic mild weakness of the right arm and leg, a positive Chaddock sign in the right, and an obvious right central facial weakness. On examining his speech, a nominal aphasia was noted which caused him tension and anxiety. The examination of the optic discs was essentially normal, except for slight arteriosclerotic changes; spontaneous venous pulsations were observed bilaterally. The remainder of the cranial nerves were normal and meningeal signs were absent. A perceptible difference was felt on palpation between the carotid pulses (Rt > Lt) without bruits. The arterial blood pressure was 160/80 mm Hg in the right arm and 110/70 mm Hg on left. A distinct systolic murmur was heard in the left supraclavicular fossa. No murmurs were audible over the precordial area. Otherwise, the physical examination was normal. At this stage the patient was given intravenous Rheomacrodex (Dextran 40) and transferred from the emergency room to the ward. On neurological examination three hours later, these findings disappeared with a complete recovery of the right hemiparesis and aphasia.

The past history of our patient revealed a mild diabetes of four years duration and a previous transient episode of right unilateral weakness and speech distur-
bances, which occurred one month prior to hospitaliza-

tion and was of short duration. No complaints of ische-
mic heart disease, arterial hypertension, nor venereal
disease were elicited from the history. His father ap-
parently died from a stroke and the mother disappeared
in the holocaust. Our patient was a heavy smoker but

took no alcohol. It should be pointed out that he
changed his job six months prior to his two neurologi-

cal episodes, when he began to work as a heavy truck
driver.

On the first day of hospitalization his EEG tracing

was normal, except for bursts of slow wave activity in

the left hemisphere which appeared only on pressure

over the right carotid artery. This manoeuvr was per-
formed after ophthalmodynamometry revealed bilater-
al normal values. On brain scanning, both static and
dynamic studies were normal. These tests were repeat-
ed two weeks later with identical results. Routine labo-
ratory tests, ECG, plain skull and chest X Rays were

within normal limits. A slightly elevated fasting blood
sugar was noted. The antinuclear factor was negative,
complement fixation, protein electrophoresis and total
lipids were also normal. Cardiological investigation

including ECG holter monitoring and echocardiogra-

phy revealed no pathology. Angiography performed
two days after admission showed

1. a complete proximal obstruction of the left sub-
clavian artery, normal cervical segments of both
carotid arteries, and a dilatation of the right ver-
tebral artery (fig. 1),

2. an intact intracranial carotid system (fig. 2),

3. marked dilatation of the left external occipital
artery, with retrograde flow in the left deep cer-
vical artery (fig. 2), with a short anastomotic
branch between this artery and (probably) the
vertebral artery (fig. 3).

4. Late reversed flow in the left vertebral artery
(fig. 4).

Due to the presence of this obvious steal from the left
common carotid artery additional to the classic right-
left vertebral steal, we performed the Javid test; pres-
sure on the left carotid artery abolished the left radial
pulse. A brain CT done three days after angiography
failed to show any evidence of cerebral infarction or a

space occupying lesion. There was no dilatation of the
ventricular system or the subarachnoidal space.

The patient was transferred to the department of
vascular surgery, where he underwent an Axillo-Axil-
lary by-pass. Follow up for a year and a half reveals a
complete improvement. His EEG tracing remains normal, even on pressure over the carotid artery.

**Discussion**

There is no doubt about the presence of a classical vertebro-vertebral shunt in this patient, in view of the dilatation of the right vertebral artery and the late retrograde filling of the left vertebral artery. This shunt did not produce any sign of vertebrobasilar insufficiency probably because the circle of Willis was intact. On the other hand, we observed an arterial collateral pattern with a steal of blood from the left common carotid artery via the left external occipital to the deep cervical artery. It is of roentgenological interest to stress the direct feeding of the distal left subclavian artery by the deep cervical artery. This pattern is less frequent than the direct feeding of the subclavian by the vertebral, which in turn benefits from branches of the deep cervical artery. In our patient there is only one visible anastomosis between these two arteries. This relatively uncommon arterial collateral pattern i.e. "stealing" blood from the left common carotid artery may have a primary function in the etiology of the clinical syndrome.

Our opinion is based on:
1. The positive Javid test as mentioned above, proving the relationship between the left carotid and the left radial pulse.
2. The occurrence of left hemispheric slow wave pattern in the EEG during pressure on the right carotid artery proves the relative incapacity of the left carotid artery to feed alone its ipsilateral hemisphere. The absence of any arteriographic demonstrable narrowing or arteriosclerotic lesions in that artery reinforces the fact that part of its flow takes part in the steal dynamics.

Independently of the angiographic findings, three other pathological processes, which may present with transient ischemic attacks, were considered as possible causes of the patient's symptomatology:
1. Angiographically not identified small ulcerations or thrombi could have been present in the left carotid system. This possibility was mentioned by De Weese and Lipschik, who found normal angigrams in such cases, especially when performed weeks after the transient ischemic attack. Such processes should have been identified in the angiography performed two days after the last attack in our patient, as they diappear quickly.
2. Angiography may be completely normal in cerebral embolism of cardiac origin. This possibility was ruled out on cardiologic investigation and lack of signs of embolism in other parts of the body.
3. Hemispheric deep lacunar infarcts with normal angiogram may cause transient ischemic attacks. Normal isotope brain scan and CT cannot rule out this diagnosis. Therefore the possibility cannot be rejected. Even if proved however, such infarcts could occur as a result of carotid insufficiency due to the shunting to the subclavian artery, in the absence of any other potential risk factors.

Although the acceptance of neurological symptoms as a direct result of exercising the involved arm is an important diagnostic aid in subclavian steal syndrome, this fact should not be regarded as a "sine qua non." It is well known that Fields et al reported the oral communication of E. J. Wylie in 1971, who was able to produce this phenomenon in only two patients out of approximately fifty. Soliti et al, found this phenomenon in four of their eight patients with cerebral insufficiency and subclavian steal syndrome. In our patient, we did not elicit a clear history of exercising with the arm related to his symptoms. It should be noted however, that transient episodes of focal cerebral insufficiency occurred after the patient changed his work to a field involving significant use of the arms.

Considering the complete relief of symptoms following the Axillo-Axillary by-pass and maintenance of the normal pattern in the EEG even following pressure on the right carotid artery, we believe that among the other possibilities mentioned here above, the most logical explanation is the occurrence of the transient left hemispheric syndrome due to the steal from the left carotid artery.

**References**

8. Hughes RR: An introduction to clinical electroencephalography. Bristol, John Wright and Sons Ltd. page 68, 1961
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Stroke. 1983;14:396-398
doi: 10.1161/01.STR.14.3.396
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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