Arch Psychiatr Nervenkr (1982) 232:179–181

Archiv für Psychiatrie und Nervenkrankheiten
Archives of Psychiatry and Neurological Sciences

Brachial Neuropathy—A Vascular Cause?

Two Case Reports

Bengt Hindfelt

Department of Neurology, University Hospital, S-221 85 Lund, Sweden

Summary. The pathogenesis of brachial neuropathy remains unknown though an infectious or allergic disorder has been postulated. The causes are probably diverse. Two patients are presented suffering from bracial neuropathy of presumed vascular origin.

Key words: Plexus brachialis lesion - Neuropathy - Vascular origin

Introduction

The pathogenesis of brachial neuropathy is unknown, though an infectious or allergic disorder has been suggested (Allen 1971; Doyle 1933; Bárdos and Somodská 1961; Tsairis et al. 1972). In single cases it may probably be due to vascular disease of the brachial plexus as indicated by the following case reports of unilateral painless brachial neuropathy.

Case Reports

Case 1

A 56-year-old constructor had previously enjoyed excellent health. During a brief morning coffee break he suddenly developed a painless weakness of his left shoulder and upper arm, making him immediately unable to continue his work. He was unaware of any recent trauma and had had no vaccinations in the years prior to this event. His family history did not reveal any neurological disorders.

Within weeks the weakness was followed by marked atrophy of the left deltoid, the supraspinam and infra-spinam, the biceps and the brachioradial muscles. The hand retained normal motor and sensory functions. At examination the biceps, brachioradial, and triceps reflexes of his left arm could not be elicited and there was a loss of cutaneous sensation covering the lateral portion of the supraclavicular fossa, the shoulder and the outer margin of the upper arm. The "flare-reaction" was markedly reduced over the left shoulder. In all other respects neurological examination was normal. Somatic examination was also normal except for a difference in blood pressure between the right and left arm (175/100 and 145/110 mm Hg, respectively). A slight systolic murmur was detected over the left supraclavian fossa, and the Adson's maneuvers did not affect the arterial pulses in the arms.

0003-9373/82/0232/0179/$ 01.00

Routine blood tests including electrophoresis for serum proteins and a test for antinuclear factor were normal. The CSF was normal with respect to cells and protein content (0.52 g/l). An electromyogram confirmed the clinical findings and showed denervation of the following muscles in the left arm: the deltoid, supra-spinam, biceps, brachioradial, radial extensor carpi and the pronator teres muscle. The left triceps muscle also exhibited some denervation on electromyographic examination. The motor nerve conduction velocities of the left ulnar and median nerves were moderately lowered (49.5 and 44.0 m/s, respectively).

An X-ray of the neck showed lowered discs and spondylotic changes with bilateral narrowing of the intervertebral foramina, particularly at the levels of C VII - C VIII and C VIII-Th I. A myelogram indicated minor central protrusions into the spinal canal within the lower cervical region, but no evidence of spinal cord compression.

Because of the difference in blood pressure between the right and left arm four-vessel angiography was performed. The left subclavian artery was proximally slightly stenotic and distally it was narrower than usual and somewhat irregular. There was no filling of the left vertebral artery. The right subclavian artery was slightly irregular distally to the vertebral bifurcation. The extracranial parts of the carotid arteries were normal.

To date 4 years have elapsed since the onset and no restitution has occurred. No other neurological symptoms or signs have emerged during this follow-up.

Case 2

A 72-year-old man suffered a spontaneous compression fracture of the spine in his early fifties. Almost a decade later hyperparathyroidism was diagnosed and he was investigated twice for a parathyroid adenoma, none being found. The serum level of parathyroid hormone was consistently high and serum calcium above 3 mEq/l.

Because of the persistent suspicion of a parathyroid adenoma the patient had been subjected to repeated angiographic studies over the years and another one was performed. A catheter was introduced into the right brachial artery and during the angiographic examination the patient suddenly noticed an anesthesic feeling in his right arm, from the axilla and distally. When examined neurologically approximately three weeks later he stated that from that moment he had suffered from an inability to abduct and adduct the fingers and a decreased sensibility of his right lower arm and hand. At the time of the examination the patient had improved, a somatic examination was normal and a detailed neurological examination revealed nothing abnormal except for motor and sensory deficits in his right arm and hand. The neurological dysfunction consisted of a decreased ability to perform finger abducting and adducting movements. Sensibility was decreased along the ulnar border of the lower arm, including the ulnar regions of the vola and dorsum of the hand. Vibration sense was decreased in the third, fourth and fifth finger. The right triceps reflex was decreased as compared to the left, while the other brachial reflexes were within normal limits. There was no Horner's syndrome on the right side. At follow-up approximately 3 years later the symptoms still persist.

Discussion

In these two elderly patients the evidence for a vascular cause of brachial neuropathy is circumstantial i.e. the very acute and fulminant onset, the occurrence while undergoing an angiographic procedure (case 2), and the angiographic findings (case 1), the lack of any other discernable reason i.e. recent immunization, infection, systemic illness etc. In both patients the onset of symptoms was painless and the course remarkable as no restitution occurred over a 3 to 4 year period. In the second patient some improvement took place within weeks but he is persistently disabled from sensory and motor deficits.

These two patients may have suffered from infarctions of the brachial plexus, which receives its proximal vascular supply from minor branches (aa. cervicalis ascendens, cervicalis superficialis, cervicalis profunda and cervicalis transversus; Mumenthaler and Schiack 1976) of the subclavian and vertebral arteries (Abdullath 1958). In the first patient the clinical findings were those of a predominantly upper lesion of the brachial plexus (supposedly distal to the dorsal root ganglia) which receives its arterial supply via the vertebral and ascending cervical arteries (Abdullah 1958). The vertebral artery was occluded ipsilateral to the brachial lesion and the subclavian artery on that side was narrowed from atherosclerotic changes. In the second patient the symptoms and signs were consistent with a lesion of the lower brachial plexus, a region which gets its main arterial supply via the costo-cervical trunk and the superior inter-costal artery (Abdullah 1958).

Brachial neuropathy due to infarction of the brachial plexus is not a new entity, though reported cases are few and not presented in great detail. Mumenthaler and Schliack (1976) briefly comment upon two patients with presumed infarction of the brachial plexus. In one of these patients the lesion was secondary to an occlusion of the axillary artery, in the other a peripheral arm paralysis developed progressively after radiation for a mammary neoplasm.

A partial infarction of the brachial plexus may easily be overlooked as a pathogenetic possibility since brachial neuropathy is a rather common and benign entity (Tsairis et al. 1972). Supposedly very few of these patients are subjected to angiographic procedures whereby an unsuspected gross vascular lesion may be revealed. However, among elderly patients with brachial neuropathy a vascular cause should be considered, particularly in case of a sudden and fulminant onset. In these cases the outcome may not be as favourable as with brachial neuropathy from other causes (Tsairis et al. 1972).

References

Abdullah S (1958) Studies of the anatomy of the human brachial plexus with special reference to its blood supply. Thesis for M Sc, University of London

Allen IM (1971) The hemological complications of serum treatment. Lancet 2 : 1128–1131

Bárdos V, Somodská V (1961) Epidemiologic study of a brachial plexus neuritis outbreak in northeast Czechoslavakia. World Neurol 2 : 973–979

Doyle JB (1933) Neurological complications of serum sickness. Am J Med Sci 185 : 484–492

Mumenthaler M, Schliack H (1976) Läsionen peripherer Nerven. Diagnostik und Therapie, 3. Auflage. Springer, Berlin Heidelberg New York

Tsairis P, Dyck PI, Mulder DW (1972) Natural history of brachial plexus neuropathy. Neurology 27 : 109–117

Received June 15, 1982