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Brachial Plexus Compression

Complication of Delayed Recognition of Arterial Injuries of the Shoulder Girdle

Seshadri Raju, MD, David V. Carner, MD

• **In six cases of penetrating trauma to the subclavian or axillary arteries without primary coincident injury to the brachial plexus, the vascular injury was not initially recognized as there were no suggestive clinical signs. The first clinical sign of vascular injury in all cases was delayed onset of brachial plexus palsy due to compression by an expanding false aneurysm. Following vascular repair, neurological recovery occurred only in two of six cases. Since brachial plexus injuries are associated with a poor prognosis, and the functional impairment of the upper extremity is serious, an aggressive investigative approach to all penetrating shoulder girdle injuries is advocated. Arteriography should be considered, even when suggestive clinical signs of vascular injury are absent. Even relatively small false aneurysms should be repaired without delay before the onset of neurological symptoms.**

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The brachial plexus is closely related to the subclavian and axillary arteries at the thoracic outlet (Fig 1). Because of this close anatomical relationship, penetrating injuries to the shoulder girdle often result in direct injury to both neural and vascular structures.[1] However, isolated penetrating injury of the subclavian or axillary artery alone without concomitant brachial plexus injury does occur. Failure to recognize and surgically correct arterial injury in such instances may result in delayed injury of the brachial plexus by compression of an expanding false aneurysm. Even small false aneurysms can result in injury to the neuroplexus due to the close anatomical relationship, and since a single fascial envelope surrounds the structures,[2] the compression effect is heightened. The compression injury of the brachial plexus is often insidious, resulting in a delay in diagnosis and treatment. Furthermore, injuries to the brachial plexus are associated with a poor prognosis, are often irreversible, and result in substantial functional impairment of the upper extremity.[3] For this reason, arterial injuries of the shoulder girdle should be approached aggressively and treated without delay.

Unfortunately, isolated penetrating injury to the subclavian and axillary arteries may be present without overt clinical signs[4-6]; distal pulses are often present and the pulsatile hematoma may be small and difficult to palpate, especially in an individual with well-developed musculature of the shoulder girdle. Therefore, we recommend that arteriography be considered in all cases of penetrating injuries of the shoulder girdle, even when overt clinical signs of arterial injury are absent.

The possibility of late brachial plexus compression by delayed recognition of a false aneurysm of the subclavian or axillary artery and the desirability of arteriography in all cases of penetrating injuries of the shoulder girdle have not been sufficiently emphasized in the literature.

REPORT OF CASES
Delayed Onset of Neurological Symptoms

CASE 1.—On Dec 1, 1962, a 36-year-old man was shot in the right shoulder. The patient was seen at another hospital at the time of injury and was found to have good distal pulses and to be neurologically intact. Two weeks after the injury, he noticed pain and swelling followed by loss of sensory and motor function in the right arm. He was admitted to the University of Mississippi

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From the Department of Surgery, University of Mississippi Medical Center, Jackson.
Reprint requests to Department of Surgery, University of Mississippi Medical Center, 2500 N State St, Jackson, MS 39216 (Dr Raju).

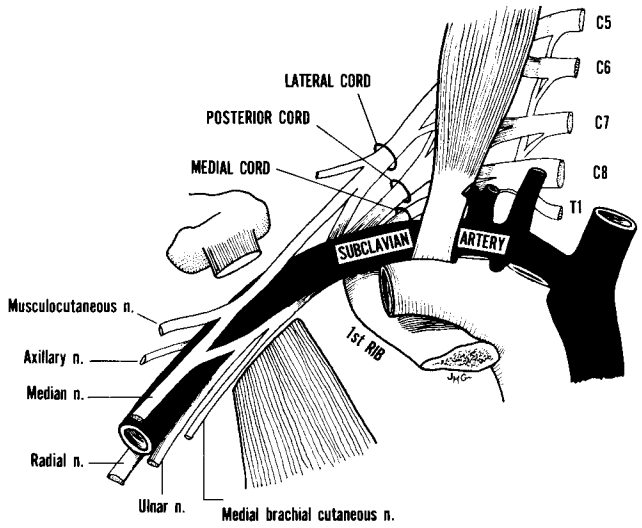

Fig 1.—Diagrammatic illustration of close association between subclavian and axillary arteries and brachial plexus. The last four (C5-C8) cervical roots and the first thoracic root (T1) combine to form the plexus.

Medical Center, Jackson, on Jan 26, 1963, six weeks after the onset of symptoms. Examination showed brawny edema of the right arm, with absent right brachial and radial pulses. A bruit was heard over the right shoulder. Sensation was absent except in the distribution of the musculocutaneous and radial nerves. There was no motor function except in the biceps and deltoid muscles. An intraoperative arteriogram showed a large false aneurysm of the axillary artery. The aneurysm was resected and the ends of the artery were oversewn. No injury other than marked stretching of the brachial plexus was found. Three months after surgery there was return of sensation except in the hand, but no increase in motor function was noted.

CASE 2.—A 56-year-old man was shot in the right supraclavicular area on Dec 14, 1967. He was admitted to another hospital for observation at the time of injury. One month after injury the patient noticed swelling in the area of the wound, and progressive loss of sensory and motor function in the right hand began to develop. He was admitted to the University Medical Center on Feb 2, 1968, approximately three weeks after the onset of neurological symptoms. Examination showed a bruit in the right supraclavicular area, pitting edema of the right arm, and loss of sensation over the ulnar aspect of the right hand and forearm with weakness of the extensors and flexors of the wrist and fingers and intrinsic muscles of the hand. An arteriogram showed a false aneurysm of the right subclavian artery. On Feb 9, the aneurysm was resected and the artery repaired primarily without a graft. At surgery, the brachial plexus was found to be stretched, but there was no evidence of direct injury to the plexus. One month after surgery there was some return of sensory function and only a slight increase in strength.

In both of these patients, neurological deficit developed several weeks after the original injury. This is characteristic of compression injury to the brachial plexus from an expanding false aneurysm. It should be noted that there was a further delay of several weeks even after the onset of neurological symptoms before either of these patients sought medical help. The insidious onset of these symptoms and the gradual progression of the neurological deficit before it became pronounced probably explains the delay. A timely arteriogram soon after the injury on both patients would probably have resulted in prompt vascular repair and avoided the loss of motor function. When an arteriogram is omitted, patients should be warned to watch for late onset of neurological symptoms.

Delayed Recognition of Vascular Injury

CASE 3.—On May 26, 1973, a 25-year-old man was shot in the right shoulder and was admitted to the Veteran's Administration Hospital, Jackson, Miss, for observation. He complained of pain in the right arm, but no evidence of vascular or neurological injury was found on careful physical examination, and he was discharged. On June 21, he was readmitted complaining of pain, numbness, and weakness of the right arm. Good peripheral pulses were present at physical examination, but a pulsatile mass in the right axilla with a bruit was noted for the first time. There was also weakness of the flexors of the wrist, biceps weakness, and sensory loss in the distribution of the musculocutaneous and median nerves. A false aneurysm of the right axillary artery was shown by arteriography (Fig 2) and resected on July 9, with interposition of a saphenous vein graft. There has been no long-term follow-up.

CASE 4.—A 15-year-old boy was shot in the right pectoral area just inferior to the deltopectoral groove. On admission to the University Medical Center approximately two hours after injury, there was no hematoma or bruit, and distal pulses were excellent and equal bilaterally. There was questionable slight weakness of the biceps muscle, but neurological function was otherwise intact. However, 14 hours after admission to the hospital, paresthesia of the right arm and hand and moderate weakness in the radial and ulnar distribution developed. There was now marked weakness of muscles in the distribution of the median nerve. An arteriogram done at this time showed a small false aneurysm in the region of the thoracoacromial artery, but the subclavian and axillary arteries had a normal contrast contour. A venogram showed absence of filling of the basilic and axillary veins but indicated filling of the subclavian vein. This defect was apparently a pressure effect due to compression of the axillary vein by an adjacent hematoma. Surgical exploration of the right subclavian and axillary arteries showed a through-and-through injury to the proximal axillary artery, with surrounding hematoma. This was repaired by resection and end-to-end anastomosis. The brachial plexus was found to be intact. The patient had near complete return of neurological function prior to discharge.

These two cases demonstrate the difficulties of diagnosis of vascular injury around the shoulder girdle by physical examination. Both patients had excellent arm pulses despite vascular injury. The presence of distal pulses with proximal arterial injury has been well described.[4-6] A brachial pulse may be present even after complete disruption of the axillary artery due to the rich collateralization around the shoulder. More frequently the injury is tangential, with good flow persisting despite a false aneurysm involving one wall (case 3). A notable hematoma may not always accompany arterial injury. Furthermore, the surrounding bony framework of the thorax and shoulder girdle tends to obscure a hematoma in this area to clinical examination. A pulsating false aneurysm may be difficult to palpate deep in the axilla, either because it is small or because there is local tenderness and well-developed musculature. The course of the projectile in the tissues may not

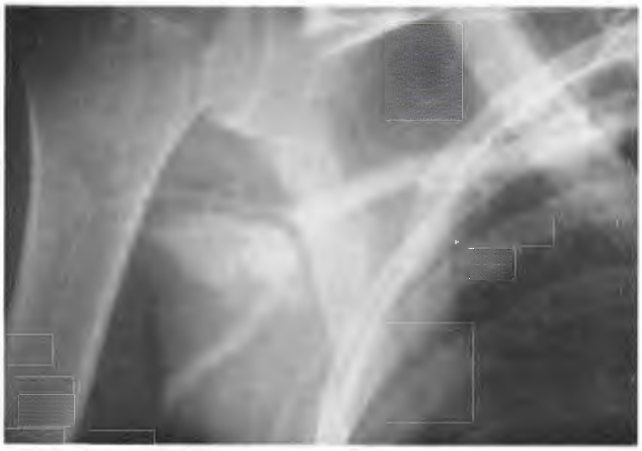

Fig 2.—Arteriogram of patient 3 showing false aneurysm of right axillary artery.

be a straight line for a variety of reasons, including deflection by bony structures. For this reason, suspicion of vascular injury should not depend on estimation of the missile path. In both of these patients, arteriograms were delayed due to a false sense of security created by negative physical findings for vascular injury. For reasons already stated, normal clinical findings do not rule out substantial vascular injury. In both patients, the developing neurological deficit due to compression was the first clinical manifestation of vascular injury. Due to the devastating nature of this complication in functional impairment of the upper extremity, and the often incomplete (if any) restoration once the neurological deficit sets in, a program of prompt angiography in all cases of penetrating injuries of the shoulder girdle appears reasonable. Even a considerable percentage of normal arteriograms under the circumstances is justified in our view, if brachial plexus compression can be avoided in the few cases by prompt recognition and treatment of the vascular injury.

Surgical Urgency Once Neurological Deficit Develops

Case 5.—A 39-year-old man was shot in the right shoulder on April 24, 1969, and was treated in the emergency room at another hospital. He was admitted to the Veteran's Administration Hospital on April 29, complaining of swelling in the shoulder and paresthesia in the right hand. Examination on admission showed good distal pulses but decreased sensation in the distribution of the median nerve. Subsequently, this progressed to complete right median nerve paresis, and at this time a pulsatile mass was noted in the right upper arm. Arteriography confirmed a false aneurysm at the junction of the right axillary and brachial arteries. This was resected and a cephalic vein graft interposed. The median nerve was found to be intact, and postoperatively there was complete return of median nerve function.

Case 6.—A 26-year-old man was wounded in the upper left area of the chest on June 29, 1969. There was transient neurological deficit when he was first seen at another hospital, but two weeks after injury he was reported to have recovered good strength in the biceps, triceps, and deltoid muscles. However, there was recurrence of neurological weakness and progressive loss of function in these muscles; the patient was transferred to the Veteran's Administration Hospital on Aug 12, 1969. On examination, a pulsatile mass below the left clavicle, patchy sensory loss in the left arm, and no motor function of the left upper extremity except for slight wrist flexion were found. A false aneurysm of the left axillary artery was shown by arteriography and resected on Aug 22, 1969. Two years after surgery, the patient had only partial return of motor function in the proximal left upper extremity.

These two cases pinpoint the need for urgency in operative intervention when a neurological deficit develops, if permanent damage is to be avoided. In case 5, there was complete return of neurological function following surgical intervention. In case 6, the opportunity for prompt vascular repair was missed; the neurological deficit that was initially transitory recurred and became permanent. Initially, compression injuries of the brachial plexus are neuropraxic in nature. At this stage, if compression can be relieved, complete recovery may be expected. When demyelination occurs due to persisting compression, recovery is delayed and may be incomplete. Return of motor function of a particular muscle is dependent to a large extent on the distance between the muscle and the site of nerve injury. The regenerating axons grow along the Schwann tubes at a fixed rate (approximately 1 mm per day). If intrinsic muscles of the hand are affected due to injury at the brachial plexus level, regenerating axons may take several months or even years to reach their destination. Unfortunately, when recovery time is prolonged, there are irreversible secondary changes in the denervated muscle that result in a poor functional outcome even when axon recovery is complete.[3] Because the small muscles of the hand play such a vital role in the functional hand and, indeed, in a useful upper limb, neurological deficit of these muscles due to compression at the brachial plexus level should be approached as a surgical emergency.

COMMENT

With increasing availability of angiography, there has been an increasing trend toward aggressive investigation of the vascular tree in the traumatized patient. Arteriography is resorted to if there are suggestive clinical signs or a high likelihood of vascular injury,[1] such as occurs with certain types of trauma (viz, subclavian artery injury with first rib fracture). The need for aggressive investigation and management of penetrating shoulder girdle injuries has been discussed by several authors.[4,5,7,8] When direct injury to the brachial plexus occurs with penetrating trauma, the indication for arteriography is clear,[1] as there is a high incidence of associated arterial injury in such instances. A less common but potentially serious injury occurs when the artery is directly traumatized without primary involvement of the brachial plexus initially. As demonstrated in these cases, such an injury often escapes prompt diagnosis and results in delayed compression injury to the brachial plexus from an expanding false aneurysm and produces considerable functional impairment of the upper extremity. Because of the seriousness of this particular complication, routine arteriography is indicated, in our opinion, in all cases of penetrating injuries to the

shoulder girdle. The first suggestive clinical sign when the arterial injury is initially missed is the onset of neurological symptoms (as in cases 3 and 4). The predilection of even a small false aneurysm to produce neurocompression is well recognized in the roentgenographic literature in relation to arterial puncture of the axillary artery for contrast study.[2] The neurovascular bundle, as it exits from the root of the neck into the upper arm, is surrounded by a fascial envelope that tends to restrict expansion of a hematoma, thus serving to heighten the compression effect on the neuroplexus.

Surgical exposure of the subclavian and axillary arteries has been described in detail by several authors.[4,5,7-9] However, certain aspects of surgical approach and technique merit special attention. It has been our experience that proximal control of the subclavian or axillary arteries through a separate proximal incision (as advocated by some) is often unsatisfactory. Due to profuse collateral flow through numerous branches in this area, considerable blood loss occurs even if the proximal clamp is placed only a short distance proximally.[1] A direct approach to the injured segment has worked better in our experience, provided care is taken not to disturb the thrombus at the injured site until vascular clamps are in place. When active bleeding is encountered, pressure to the point of injury can be maintained while clamps are placed. Blind application of clamps in a hasty manner through a blood-filled field is avoided, as it carries a high risk of inadvertent injury to the adjoining neuroplexus.

Inadvertent injury to the plexus is best avoided by limiting manipulation and dissection to the area within the perivascular sheath. It is important to avoid placement of heavy silk loops or umbilical tapes around the nerves, as traction will result in neural injury. The subclavian artery and, to a lesser extent, the axillary artery are friable vessels[1] and are easily torn. A tension-free anastomosis is mandatory. An interposed segment of saphenous vein is often required to effect a satisfactory tension-free anastomosis. It has been unnecessary to administer heparin to the patient even while the artery is clamped for repair. Again, the numerous collateral vessels maintain sufficient flow to prevent intravascular thrombosis.

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