Male Patient Post Polio With Ruptured Left Common Carotid Artery

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Keywords: radiology ■ magnetic resonance imaging ■ muscle atrophy ■ anatomy ■ stent ■ common carotid artery ■ polio imaging

J Natl Med Assoc. 2012;104:110-115

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HISTORY

The patient was a 58-year-old right-handed male businessman who presented with transient ischemia of his left internal carotid artery with tingling-like sensation in his left shoulder and upper arm. He had experienced paresthesia in the shoulder girdle and the left upper extremity.

He had polio at age 2 years complicated by atrophy of the left shoulder muscles with weakness. For many years, he carried his brief case with left arm and hand, not aware he was stretching the left neck fascia and eventually dissecting the site of stenosis within the left common carotid artery. He presented to the emergency department acutely bleeding from the rupture of the left common carotid artery. His neurologist indicated he had undergone angioplasty and stent placement that subsequently restenosed and then reopened with stent placement 1 year later.

PHYSICAL EXAM

General

The patient was alert, active, in no acute distress. His vital signs were: height, 1.85 m; weight is 101.2 kg; blood pressure, 130/80 mm Hg, right arm, sitting; pulse 55 beats/min; and respirations at 12 breaths/min. He had chronic deformity of his left hand present for many years involving atrophy of his hand and wrist. He had had many surgery procedures of his left hand with substantial loss of the left arm function and certainly a complete loss of hand function. Carotid pulses were palpable. There were no bruits in the left neck. On the right neck, a bruit was heard radiating from the chest that appeared to be a systolic ejection murmur in listening to the apex of the chest.

Laboratory Data

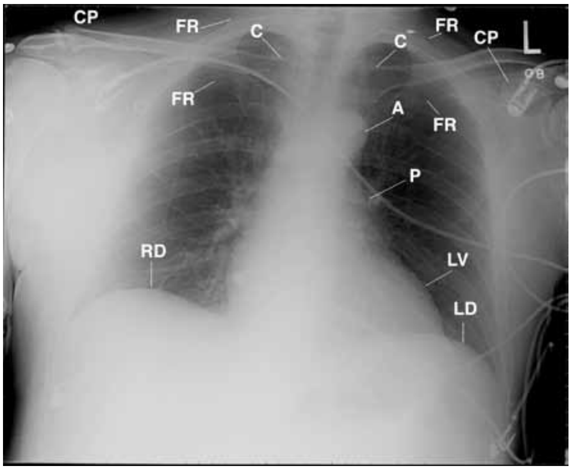
Preoperative lab findings were within normal limits, specifically a slightly elevated creatine kinase; normal findings from aldolase, liver tests, electrolytes, blood urea nitrate and creatinine, complete blood count, prothrombin time, and partial thromboplastin time.

Diagnostic Studies

Preoperative echocardiogram showed left bundle-branch block with a rate of 50 (this left bundle-branch block was old and had been documented for many years).

Preoperative SPECT thallium treadmill showed normal wall motion with no evidence for ischemia, with normal ejection fraction. There is some slight thickening to the ventricular wall on scan.

Figure 1. This anterior-posterior chest radiogaph displays the atrophy of the left neck and lateral chest wall muscles with increased radiolucency of the left lung as compared to the right, metal clip (not labeled) over the proximal left first rib (FR).



A, aorta; C, clavicle; CP, coracoid process; L, left side; LD, left diaphragm; LV, left ventricle; P, pulmonary artery; RD, right diaphragm.

DIAGNOSIS

- Polio at age 2 years with substantial loss of the left arm function with complete loss of hand function. Post left common carotid artery rupture with stenting.
- Post restenting left common carotid artery 1 year later, suspected thoracic outlet syndrome.

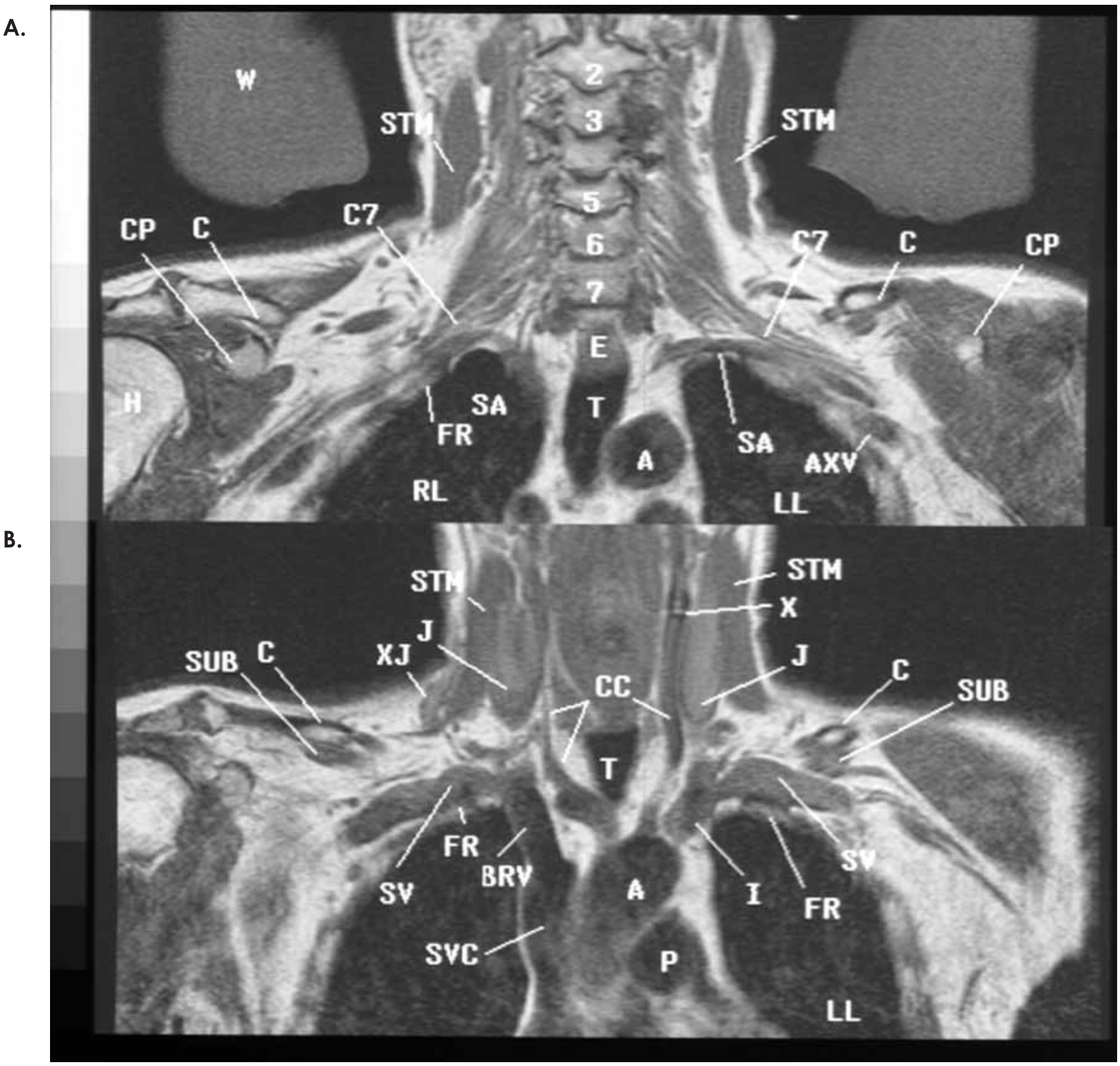
Bilateral MRI of the brachial plexus has been requested to determine the site of compromise of the brachial plexus and to discern the anatomic landmark anatomy.[1-3]

The anterior-posterior chest radiogaph (Figure 1) displays the atrophy of the left neck and lateral chest wall muscles, increased radiolucency of the left lung as compared to the right, metal clip over the proximal left first rib.

CONCLUSION FOR THE CHEST RADIOGRAPH

- Atrophy of the left thorax, shoulder, and arm muscles.
- Metal clip post stent placement.

Figure 2. Atrophy of the Left Shoulder, Neck, and Chest Wall Muscles



These coronal noncontiguous sequential magnetic resonance images display marked atrophy of the left shoulder (drooping), neck and chest wall muscles; low signal intensities marginating the stent (X) in the left common carotid artery in the lower image; intermediate gray proton signal intensity increased turbulence superior to the low signal intensity within the left common carotid artery; mixed signal intensities turbulent venous return in the dominant right internal jugular vein (J): left clavicle (C) with the subclavius muscle (SUB) compressing the bicuspid valve within the gray proton-dense bulbous expanded subclavian vein (SV); gray proton-dense compressed second divisions of the subclavian arteries (SA); gray proton-dense left brachiocephalic vein (BRV) reflecting decreased venous return as compared to the right (BRV); and increased venous return from the dilated right external jugular vein (XJ).

A, dilated aorta; AXV, axillary vein; CC, common carotid artery; CP, coracoid process; E, esophagus; FR, first rib; H, humerus; I, left brachiocephalic vein; LL, left Lung; P, pulmonary artery; RL, right lung; SPC, spinal cord; STM, sternocleidomastoid muscle; SVC, Superior vena cava; T, trachea; TRP, trapezius muscle; 2, 7, second through seventh cervical vertebrae; C7, seventh cervical nerve root.

MAGNETIC RESONANCE IMAGING FINDINGS

Coronal sequence displayed marked atrophy of the left shoulder, neck and chest wall (Figure 2); left pectoralis major and minor muscles and the latissimus dorsi muscle were virtually absent; the left trapezius, deltoid, supraspinatus, infraspinatus, subscapularis, coracobrachialis, rhomboid, middle scalene and sternocleidomastoid muscles were small compared to the right; anterior-lateral rotated left shoulder); laxity and acute descent of the left clavicle compressing the left subclavian vein on the first rib; high proton-dense signal intensities within the dominant right internal jugular and smaller left internal jugular veins; horizontal mixed-signal intensity demarcates the suspected junction of the stent in the left common carotid artery; a conically shaped, pointed intermediate gray signal was present within the lumen of the left common carotid artery; and the right subclavian artery arched normally over the first rib, compressed by the larger anterior scalene muscle.

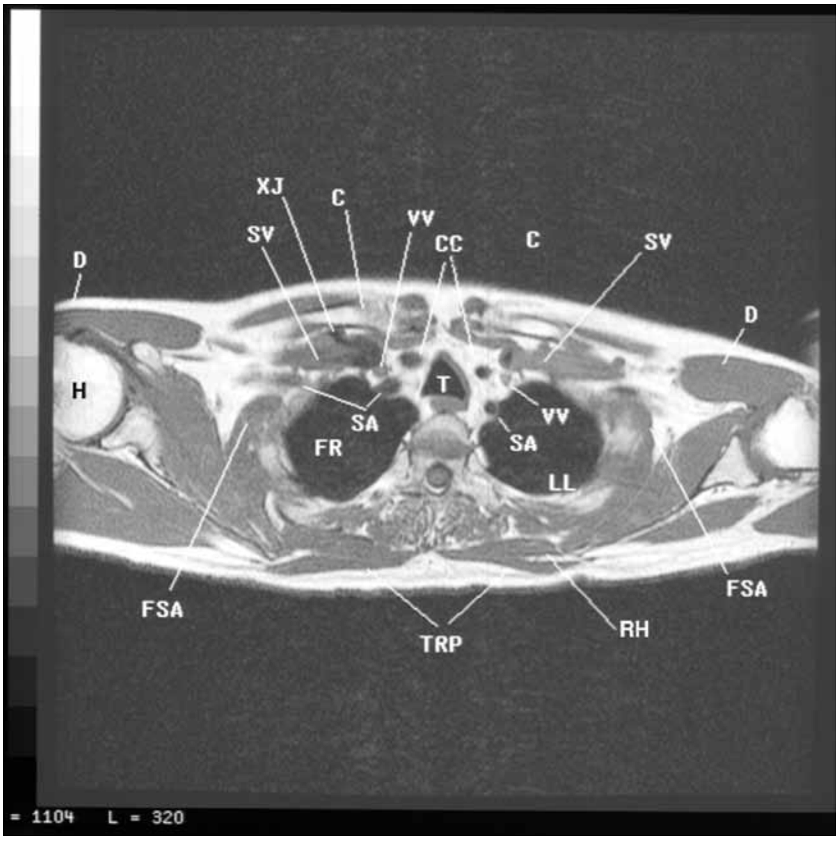
Transverse Sequence

This sequence confirms the atrophic left chest wall shoulder girdle and neck muscles and manubrium sternum sloping to the left and pectoralis muscles virtually absent throughout images acquired (Figure 3). Atrophy of the deltoid, supraspinatus, and infraspinatus muscles are demonstrated nicely; and acute angulation of the C8-T1 nerve roots and C7 nerve root, reflecting costoclavicular compression, and the unprotected left spinal accessory nerve is displayed within the fascial planes of fat common with the dilated left common carotid artery as compared to the right common carotid artery.[4,5]

Left and right transverse oblique sequences cross-reference the coronal sequence. Left and right sagittal sequences cross-reference the coronal and transverse sequences to display costoclavicular compression of the left subclavian vein (Figure 4). The anterior scalene muscle descends vertically, effacing the subclavian artery and nerve roots on the compression of the binding roots on the second division of the left subclavian artery.

Two-dimensional time-of-flight magnetic resonance angiography and venography (MRA and MRV) displayed

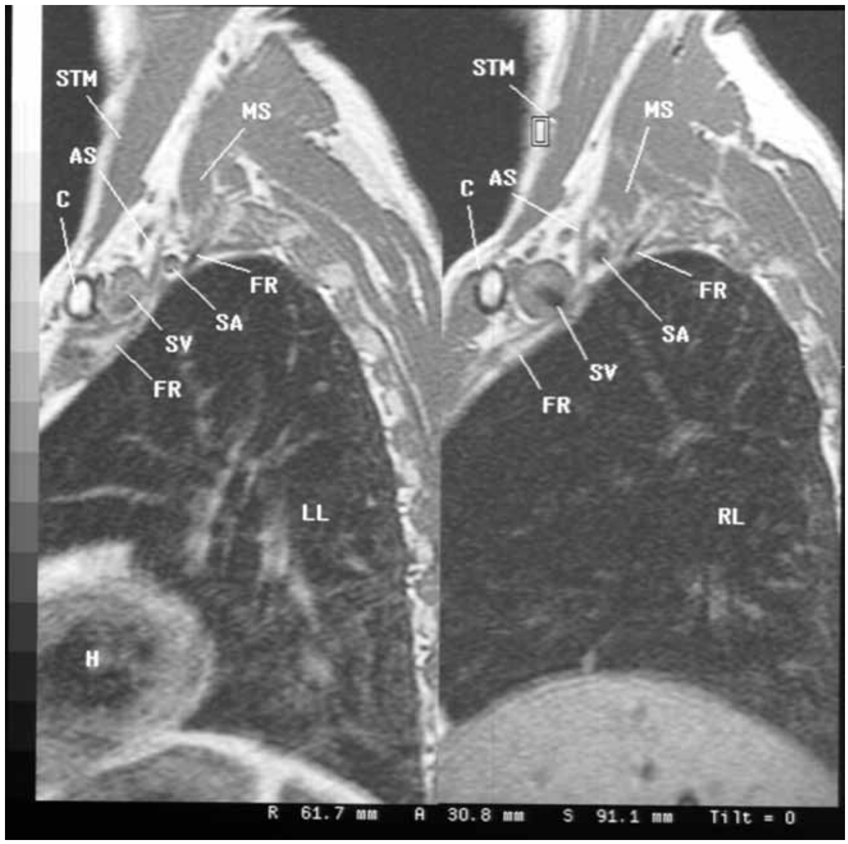
Figure 3. Atrophic Smaller Left Shoulder Muscles, Left Clavicle, and the Subclavius Muscle



Transverse magnetic resonance image cross-references the coronal sequence of images to display the atrophic smaller left shoulder muscles, left clavicle (C) with the subclavius muscle (not labeled) compressing the left gray proton-dense external jugular vein (XJ) and the subclavian veins (SV) left greater than right; dilated vertebral veins (VV) reflecting impedance to venous return; smaller left trapezius (TRP) and rhomboid muscles (not led the left). Observe the greater high signal intensity fat (not labeled) over the left shoulder as compared to the right.

CC, common carotid artery; D, deltoid muscle; FR, first rib; H, humerus; LL, left lung; RH, rhomboid muscle; SA, subclavian arerty; T, trachea.

Figure 4. Anterior Scalene Muscle



The left sagittal image displays the near vertical left anterior scalene muscle (AS) compressing the subclavian artery (SA) and nerve roots on the first rib (FR) in the scalene triangle. The left clavicle compresses the external jugular vein (not labeled) against the smaller subclavian vein (SV) as compared to the right external jugular vein (not labeled) against the right subclavian vein. The right sagittal image displays the anterior scalene muscle compressing the larger subclavian vein on the first rib (FR) posterior to the clavicle and the subclavius muscle diminishing the signal intensity within the lumen of the vein.

H, heart; LL, left lung; MS, middle scalene muscle; RL, right lung; STM, sternocleidomastoid muscle.

the posterior angulation of the left neurovascular blood supply as compared to the right, consistent with atrophy; anterior lateral rotation of the shoulder girdle and acute sloping of the ribs. Compressed left subclavian artery diminishes size into the supraclavicular fossa and axilla. The left subclavian vein is compressed and the axillary vein dilated. The right subclavian artery is mildly compressed within the scalene triangle proximal to the first rib, and the right subclavian vein is compressed by the overlying clavicle and subclavius muscle. The left common carotid artery signal intensity discontinues proximal to its bifurcation reflecting the stent placement as per history (Figure 5).

Bilateral abduction external rotation (AER) displayed enhanced costoclavicular compression without significant compression of the tense left neurovascular bundle and left subclavian vein (Figure 6).

Bilateral AER of the upper extremities did not trigger pain, tingling, and/or numbness. The pain and discomfort experienced improved on this maneuver in the supine position. The patient actually experienced no problem in assuming this position on the right and/or on the left.

CONCLUSIONS

- Incomplete left common carotid artery signal intensity 45 mm from the aortic arch secondary to the stent placement obscured signal within the left common carotid artery.
- Laxity of left neck, shoulder girdle and upper extremity, contributing to stretching of the neurovascular bundle, which triggered complaints per patient's history.[6]
- Marked atrophy and virtually absent left pectoral, infraspinatus, trapezius, and latissimus muscles.
- Fatty replacement of left chest wall muscles.
- Bilateral AER maneuver did not trigger complaints in the supine position, secondary to decreased tension and marked atrophy.

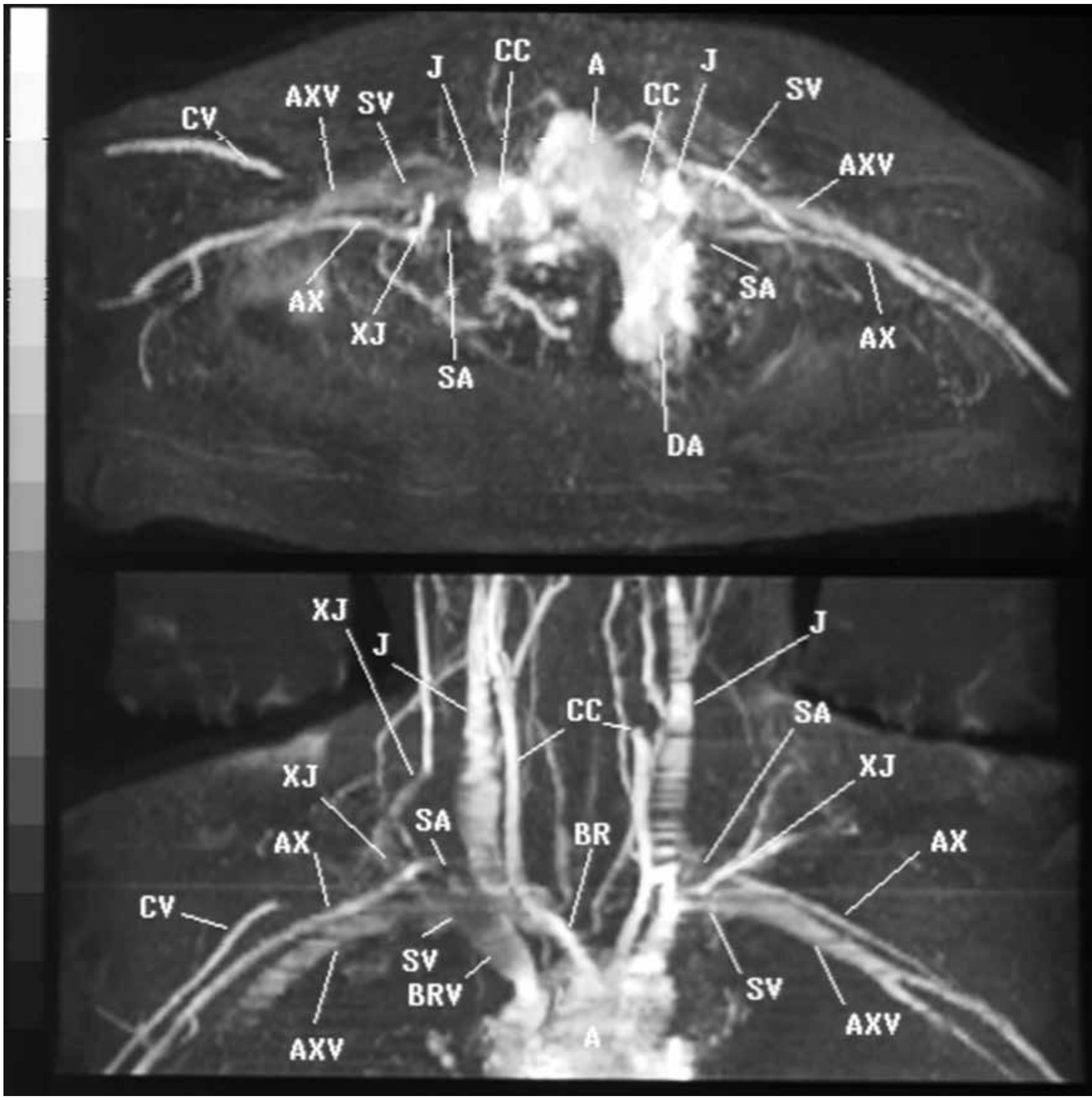
COMMENT

The sequences demonstrated laxity, considerable atrophy of the left neck, shoulder, upper-extremity and chest wall muscles replaced by fat as a complication of polio and the virtual absence of pectoral, latissimus

Figures 5. Subclavian Arteries and Veins

A.

B.



Coronal 3-dimensional reconstructed magnetic resonance angiography and venography images display diminished signal intensities of the compressed second division of the subclavian arteries (SA), right greater than left; bilateral compressed subclavian veins (SV), right greater than left; segmented decreased venous return within the left internal jugular vein (J) as compared to the right; dilated near vertical aorta reflecting mild kyphosis of the cervicothoracic spine; high signal proximal right external jugular vein as compared to near absence flow on the left; mild crimping of the brachiocephalic artery (BR) with the first division of the right subclavian artery (SA) compressing the region of the bicuspid valve within the right internal jugular vein diminishing venous return within the right brachiocephalic vein (BRV); increased collateral venous return over the right neck and shoulder as compared to the left; dilated high signal intensity left axillary vein (AXV) reflecting greater impedance to venous return as compared to the right axillary vein; higher signal intensity of the left brachiocephalic vein (not labeled) reflecting greater impedance to venous return from the left neck and shoulder than on the right, and the dominant right shoulder as compared to the drooping left.

A; aorta; AX; axillary artery; CC; common carotid artery; CV; cephalic vein; DA; descending aorta, XJ; external jugular vein.

muscles, and trapezius muscles exposing the left chest fascial planes of fat and nerves. The 2-dimensional time-of-flight MRA displays the incomplete course of the common carotid artery proximal to its division into the internal and external branches suggesting occlusion and/or loss of signal intensity secondary to stent placement, obscuring blood flow signal intensity as described. AER of the upper extremities and the 2-dimensional time-of-flight MRA and MRV sequence demonstrate posterior angulation of the left neurovascular structures, consistent with drooping and sloping of the left shoulder girdle and thorax. It seems reasonable that the upright position would accentuate the changes as described.

TAKE-HOME MESSAGE

The polio virus may cause lower-limb paralysis, abnormal gait, respiratory complications, and upper- and lower-extremity muscle atrophy as in our patient. This

Figure 6. Bilateral Abduction External Rotation of the Upper Extremities

Coronal bilateral abduction external rotation sequence (AER) of the upper extremities displaying the intermediate higher gray proton density within the compressed left subclavian vein (SV), axillary vein (not labeled) and brachiocephalic vein(s) (BRV) as compared to the right; right coracobrachialis muscle (not labeled) depressing the gray proton-dense right axillary vein (AXV). First rib (FR) is as compared to the absent left coracobrachialis muscle; dilated inferior vena cava (not labeled) reflecting impedance to venous return, and the higher gray proton density within the region of the stent (X) in the left common carotid artery (not labeled). Observe the smaller left deltoid and fascicules of the left serratus anterior muscle as compared to the right.

A, aorta; T, trachea; LD, left diaphragm; RD, right diaphragm.

case displays the complication of stent placement. The signal intensity of arterial blood with or without contrast is obstructed by the stent and thereby is not registered on the grayscale of MRI/MRA/MRV. This finding stresses the need for a complete history. Had this fact not have been provided by the history, physicians and health care professionals would not understand the loss of the signal intensity occurred secondary to stent placement on the 2-dimensional time-of-flight MRA and MRV sequence.[2,6,7]

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