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Internal Jugular Vein Stenosis is Common in Patients Presenting with Neurogenic Thoracic Outlet Syndrome

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Previous magnetic resonance imaging studies have shown abnormalities of the internal jugular veins in patients with thoracic outlet syndrome (TOS), but this finding has largely been ignored. We, thus, prospectively performed diagnostic brachiocephalic venograms in all patients with diagnosed neurogenic TOS from April 2008 to December 2011 (mean age, 42.6; *r*, 16–68; 77.8% women and 22.2% men). Stenosis of the left internal jugular vein, left subclavian vein, right internal jugular vein, and right subclavian vein were assessed, and significant stenoses of these vessels were seen in 63.49%, 65.08%, 60.32%, and 68.25% of patients, respectively. Internal jugular vein stenosis was not present in 23.81%, present unilaterally in 28.57%, and present bilaterally in 47.62% of patients. Subclavian vein stenosis was not present in 17.46%, present unilaterally in 28.57%, and present bilaterally in 53.97% of patients. Phi coefficients of correlation were 0.067 between left internal jugular vein and left subclavian vein stenoses, 0.061 between right internal jugular vein and right subclavian vein stenoses, and 0 between any internal jugular vein and any subclavian vein stenoses, indicating there is no correlation between jugular vein stenosis and subclavian vein stenosis in these patients. We conclude that right and left internal jugular vein stenosis is common in patients with neurogenic TOS symptoms. Treatment of internal jugular vein stenosis could potentially benefit these patients, and the implications of these findings warrant further study.

INTRODUCTION

Thoracic outlet syndrome (TOS) is a condition caused by compression of the neurovascular structures running to the arm through the superior thoracic outlet.[1] TOS can be broken down into neurogenic, arterial, and venous subtypes depending on the structure(s) compressed, including the brachial plexus, subclavian artery, and subclavian vein or a combination of these structures. Hypertrophic anterior and/or medial scalene muscles, narrowing between the clavicle and first rib, and pressure imposed by the pectoralis minor can contribute to the compression of these structures. Headaches, auditory and visual changes, pain and tingling of the extremities, and arm and hand weakness are common symptoms, and patients benefit through both noninvasive therapies such as stretching and surgical approaches, including first rib resection, scalenectomy, pectoralis minor tendon release, and vascular decompression.[2]

Traditionally, the fraction of patients with TOS with a venous etiology is small, comprising up to 5% of patients with TOS (compared with 95% neurogenic and 1% arterial).[1] Venous TOS is typically recognized with thrombus formation in the subclavian vein, but links to internal jugular vein stenosis have been described. Collins et al.[3] have demonstrated compression of internal jugular veins in magnetic resonance images of patients with kyphosis who had TOS symptoms. Classically, the internal jugular vein is not considered to be under the same anatomic constraints because the axillary subclavian vein in TOS and the phenomena of central venous compression have not been

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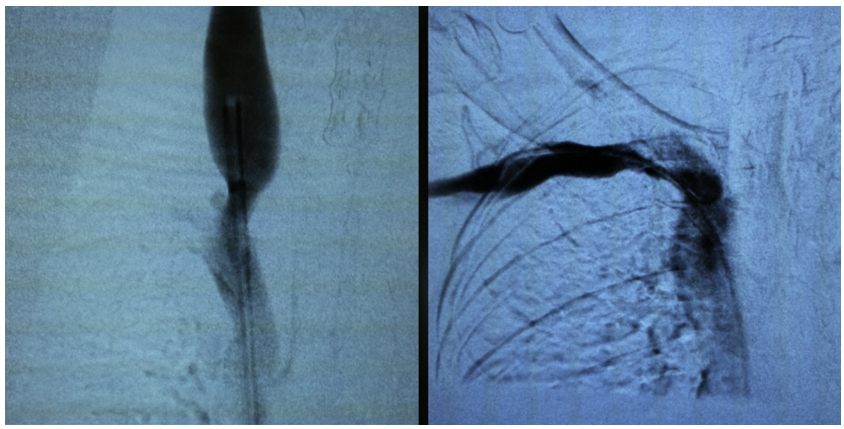

Fig. 1. Examples of significant stenosis in internal jugular and subclavian veins. Stenosis was considered to be high grade if the vein displayed >66% occlusion. High-grade stenoses were considered significant in our study. A highly stenosed internal jugular vein is seen *left*. A highly stenosed subclavian vein is seen *right*.

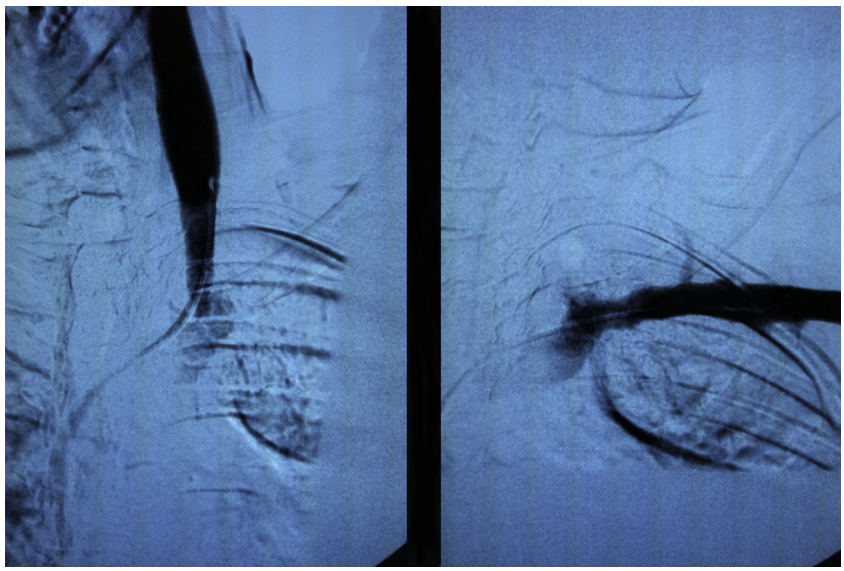

Fig. 2. Examples of nonsignificant stenosis in internal jugular and subclavian veins. Stenosis was considered to be low grade if the vein displayed <33% stenosis. Stenosis was considered medium grade if the vein displayed 33–66% stenosis. These stenoses were not considered significant in our study unless they displayed collateral flow (Fig. 3). A medium-grade stenosis of the internal jugular vein is seen *left*. A low-grade stenosis of the subclavian vein is seen *right*.

thoroughly explored in patients with TOS. We, thus, undertook this prospective study to determine the incidence of internal jugular vein stenosis in these patients.

MATERIALS AND METHODS

We prospectively performed diagnostic brachiocephalic venograms in consecutive patients with neurogenic TOS from April 2008 to December 2011. All patients presented with signs and symptoms consistent with neurogenic TOS, and all cited headaches and numbness and tingling of the head, neck, and extremities as major components of their presenting symptoms. There were 63 unique patients with an average age of 42.6 years (*r*, 16–68) with 77.8% women and 22.2% men.

For venograms, all contrast injections were by hand with minimal force with 5 cc of half-strength contrast. Patients were instructed to hold their breath after a normal inspiration for the duration of the venogram.

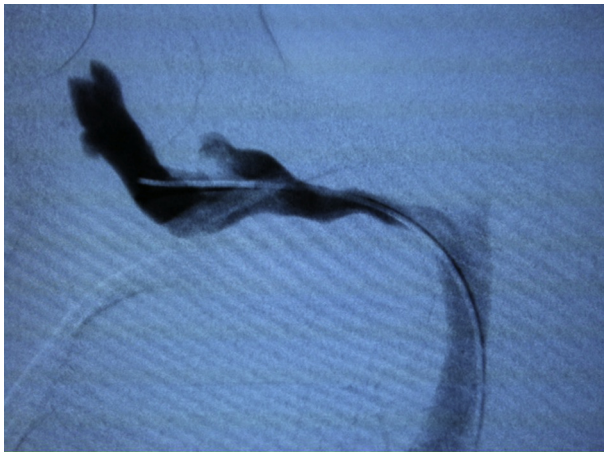

Fig. 3. Example of jugular vein stenosis with visible collaterals. Medium-grade stenosis is seen, but the presence of collaterals implies profound impedence of venous flow. This example of stenosis would thus be considered significant in our study.

presence of collaterals around the obstructions of the right and left internal jugular and subclavian veins that would implicate significant hemodynamic disruption in these vessels. For the analysis, high stenosis was considered significant (Fig. 1). Medium and low stenoses were also considered significant only if they presented visible collaterals (Figs. 2 and 3). Patients who exhibited significant stenosis were tallied, and rate of incidence was calculated. Phi values of correlation between internal jugular vein stenosis and subclavian vein stenosis were calculated using a 2 × 2 contingency table for binary variables.

RESULTS

Left internal jugular vein, left subclavian vein, right internal jugular vein, and right subclavian vein stenoses were seen in 63.49%, 65.08%, 60.32%, and 68.25% of patients, respectively. Internal jugular vein stenosis was not present in 23.81%, present unilaterally in 28.57%, and present bilaterally in 47.62% of patients. Subclavian vein stenosis was not present in 17.46%, present unilaterally in 28.57%, and present bilaterally in 53.97% of patients. Phi coefficients of correlation were 0.067 between left internal jugular vein and left subclavian vein stenoses, 0.061 between right internal jugular vein and right subclavian vein stenoses, and 0 between any internal jugular vein and any subclavian vein stenoses, indicating there is no correlation between jugular vein stenosis and subclavian vein stenosis in these patients. Observed stenoses with prominent collaterals were distributed in 62.50% high stenoses, 27.78% in medium, and 9.72% in low. The results are summarized in Table I.

Table I. Rates of internal jugular vein stenosis and subclavian vein stenosis

	Internal jugular vein stenosis (%)	Subclavian vein stenosis (%)
None	23.81	17.46
Unilateral	28.57	28.57
Bilateral	47.62	53.97

	Left internal jugular vein (%)	Left subclavian vein (%)	Right internal jugular vein (%)	Left subclavian vein (%)
Rate of stenosis	63.49	65.08	60.32	68.25

	Left internal jugular and left subclavian veins	Right internal jugular and right subclavian veins	Any internal jugular stenosis with any subclavian stenosis
φ value	0.067	0.061	0

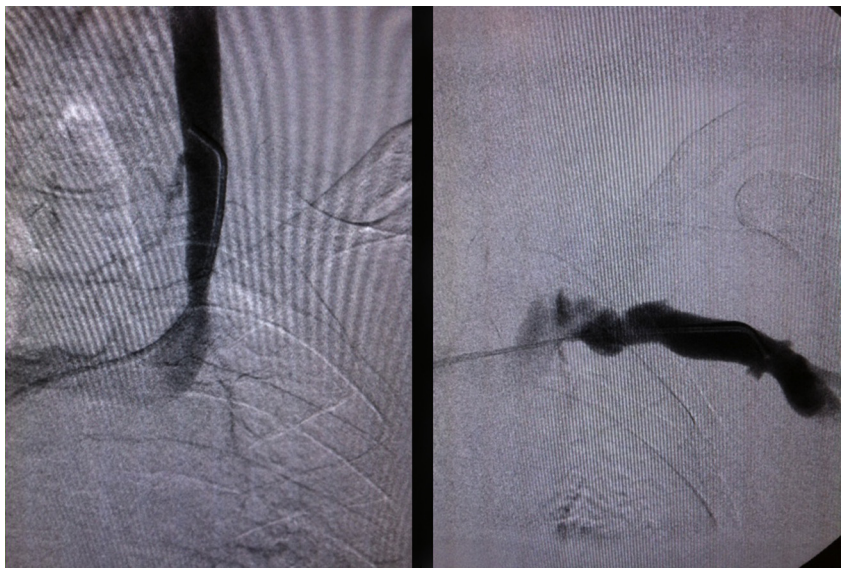

Fig. 4. Internal jugular vein stenosis is not dependent on subclavian vein stenosis. The figure illustrates a patient who has a significant stenosis in the left internal jugular vein yet an unremarkable left subclavian vein.

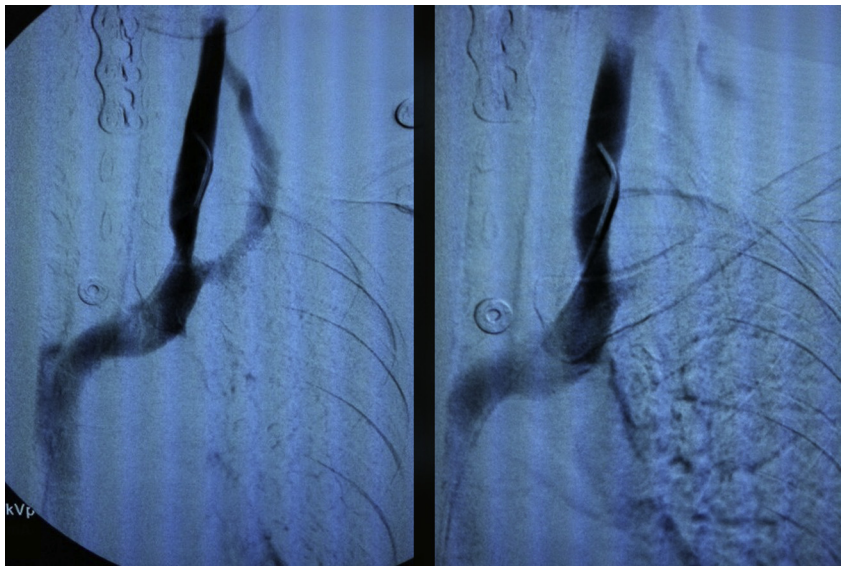

Fig. 5. Internal jugular stenosis in patients with TOS in response to ballooning. The mentioned patient with a significant internal jugular stenosis underwent a ballooning procedure, leading to increased patency of the vessel and resolution of collateral circulation as well as temporary resolution of symptoms.

DISCUSSION

Our data suggest that significant internal jugular vein stenosis is common in patients with neurogenic TOS. Although the venous component of TOS is considered to be only due to subclavian vein stenosis, our data suggest that the incidence of internal jugular vein stenosis is only slightly less than that of subclavian vein stenosis in patients presenting with neurogenic TOS symptoms. Interestingly, we did not see a correlation between the incidence of internal jugular vein stenosis and subclavian vein stenosis in these patients, suggesting that the occurrence of these stenoses is not dependent on each other (Fig. 4). Thus, despite the classical paradigm that the internal jugular vein is not anatomically at risk for compression at the superior thoracic outlet, internal jugular vein stenosis is

common in patients with neurogenic TOS, and it may be appropriate to expand the etiology of TOS to include internal jugular vein compression. Anatomically, bilateral internal jugular and subclavian stenosis are similar to bilateral innominate stenosis that is similar to superior vena cava (SVC) compression. Thus, such compression could account for symptoms similar to low-grade SVC syndrome, such as headaches, head and neck swelling, and dizziness.[4]

The implications of these findings and their involvement in TOS development and morbidity require further study. It would be helpful to determine direct consequences of these lesions, such as determining if these stenoses in patients with TOS lead to cephalic venous hypertension. Some presenting symptoms of TOS, including intractable headaches and light-headedness, may certainly have contributions from internal jugular vein stenosis, and, anecdotally, patients with TOS with these symptoms experience temporary relief with ballooning of these stenoses (Fig. 5). Temporary relief in symptoms from relieving internal jugular compression also lends weight to the idea that lesions in these vessels form a component of TOS. A comprehensive clinical correlation for the effect and treatment of internal jugular stenosis in patients with TOS is necessary, and we do not advocate angioplasty or stenting as the standard of care for these lesions as this time.

CONCLUSIONS

Right and left internal jugular vein stenosis is common in patients with neurogenic TOS symptoms. The stenosis of these vessels occurs with or without subclavian vein stenosis and may call for an increase in the scope of TOS etiology. Patients may benefit from the treatment of these stenoses, but the implications of internal jugular stenosis in TOS is not clear and require further study.

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