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Superior Vena Cava Syndrome Associated with Left Subclavian Thrombosis Secondary to Anaplastic Thyroid Carcinoma: A Case Report

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ABSTRACT

Superior vena cava syndrome results from obstruction of the venous flow or the return of the superior vena cava. Increased venous pressure in the upper body causes edema of the face, neck, and upper extremities occasionally accompanied by vessel distension, cyanosis, cough, dyspnea, stridor, and dysphagia. Cerebral edema causes headaches, confusion, coma, and even death. Lung cancer and non-Hodgkin lymphoma are the most frequent malignant causes (85%) of superior vena cava syndrome, and thyroid cancer is a rare cause (< 5%).

KEYWORDS: superior vena cava syndrome, anaplastic thyroid carcinoma, subclavian thrombosis

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INTRODUCTION

Superior vena cava syndrome (SVCS) is a potentially life-threatening complication caused by obstruction, thrombosis, or tumor invasion of the superior vena cava resulting, from malignancy in 60% to 70% of cases.[1,2,5] Another major cause of SVC is the use of intravascular medical devices, such as thrombosis associated with venous catheters (central or hemodialysis), and cardiac devices (defibrillators and pacemakers).[2] The superior vena cava begins after the union of the brachiocephalic veins, and the venous flow from the head and upper extremities empties directly into the right atrium.[1,2,3] Any obstruction in the flow of this vein leads to collateral circulation determined by the azygos vein and the veins of the mediastinum increasing the venous pressure from 20 to 40 mmHg at the point of the obstruction.[2,3] The most common malignant cause of such obstructions is non-small cell lung cancer (up to 50%), followed by small cell lung cancer (25%), lymphomas (10%), and other cancers (germinal, mesothelioma, and breast cancer), including metastatic tumors.[2,3,5] Thyroid cancer is a rare cause of SVCS, with some 30 cases having been reported.[4] A diagnosis of superior vena cava syndrome is made based on the clinical picture and imaging studies. Facial and arm edema are the most frequent symptoms. Other symptoms include facial plethora, dyspnea, stridor, and dilation of the veins in the neck.[3] Computed tomography with contrast administration is the study of first choice to assess the level of obstruction in the venous system. Other imaging studies used for this purpose include ultrasound, digital subtractive or magnetic resonance angiography, and positron emission tomography.[2,3,10] The treatment strategies for primary thrombosis are based on the etiology, the severity of the symptoms, and access to invasive procedures. Usually, for grades 1 and 2, the preferred interventional therapy involves stenting, thromboaspiration, thrombectomy, and/or thrombolysis. For grades 3 and 4, the treatment is multidisciplinary, the aim always being to treat the symptoms, which occasionally require intensive care.[1]

CLINICAL CASE

This case report focuses on an 82-year-old woman, a housewife residing in Zacatecas, Mexico with a history of

anaplastic small cell thyroid cancer diagnosed five months previously, hypothyroidism, and hypoparathyroidism secondary to total thyroidectomy for which she was being treated with levothyroxine 150 mcg/day, calcitriol 0.25 mcg/day, and calcium gluconate 2.94 gr/day, respectively. The patient had received liposomal doxorubicin-based chemotherapy for 6 cycles with an initial treatment response and tumor volume reduction of 70%. She presented in the emergency department with tumor enlargement, facial edema, headache, cough, and progressive dyspnea. Her vital signs were blood pressure 140/78 mmHg, heart rate 102 bpm, respiratory rate 22 rpm, and 90% oxygen saturation. Physical examination revealed facial plethora, tumor and edema in the cervical area and upper limbs (Figure 1).

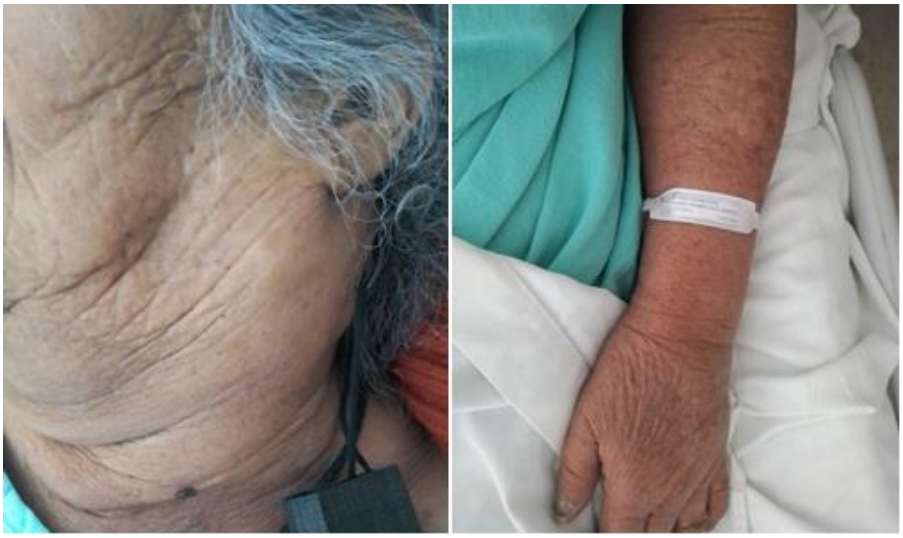

Figure 1. Photograph of the patient showing the thyroid tumor in the left cervical area and the presence of edema in the right upper limb.

Both carotid pulses were palpitated, and dilation of the upper veins of the left thorax and neck was observed in addition to audible laryngeal stridor detected through auscultation. Because of the progression of malignant disease, intravenous contrast tomography was requested, and coronal section showed the presence of thrombosis in the left subclavian vein and compression of the airway by the tumor and the right collateral veins (Figure 2).

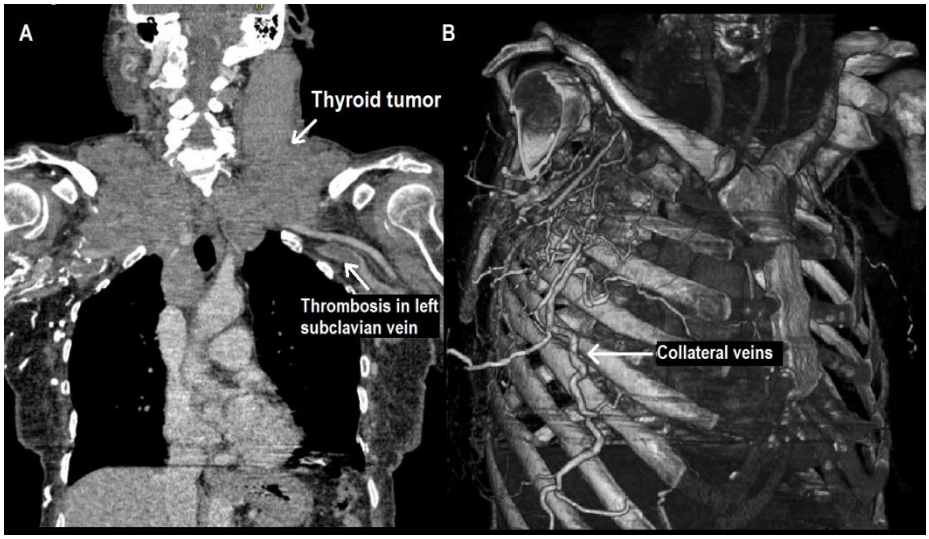

Figure 2. Tomography of the chest with intravenous contrast: (A) coronal projection showing thyroid tumor compressing vascular structures and left subclavian vein thrombosis. (B) Three-dimensional reconstructed image showing the collateral veins.

CONCLUSION

Superior vena cava syndrome secondary to thyroid neoplasia is a complication of tumor invasion and vascular thrombosis. Brain edema and hemodynamic involvement are factors indicating a poor prognosis, and facial edema is the symptom most frequently observed (80-100%).[2,3,4] The clinical presentation is progressive and varies according to the location of the obstruction and the establishment of

collateral venous circulation.[2] The diagnosis is based on clinical signs (neurological, facial, laryngeal, and venous) and an imaging study that demonstrates vascular obstruction, invasion, or thrombosis. The patient in this case study presented with dyspnea, cough, stridor, and edema with vascular obstruction and thrombosis of the left subclavian vein developing collateral circulation in the right chest wall. The occlusion affected the circulation of the left brachiocephalic vein and was classified as grade 3 to 4 because of the presence of stridor. The treatment in this case was elevation of the head and administration of glucocorticoids and loop diuretics because the patient was not a candidate for endovascular therapy.

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