

# A Case of Cervical Malignant Lymphoma with Carotid Sinus Syndrome Resoluted by Lymph Node Dissection and Subsequent Treatment

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**Abstract** A 79-year-old male presented with decreased blood pressure, weight loss, and episodes of transient loss of consciousness. Diagnosed with neurocardiogenic syncope and vagal nerve paralysis, he experienced a loss of consciousness. Despite initial treatments, syncope persisted. Repeated imaging revealed swelling of neck lymph nodes, diagnosed as carotid sinus syndrome. Lymph node biopsy confirmed malignant lymphoma, prompting thorough dissection. Following diagnosis, syncope episodes ceased. Steroid therapy and radiation were initiated due to weight loss and swallowing difficulties. This case underscores the importance of considering surgical interventions, even when non-invasive treatments fail, in managing carotid sinus syndrome caused by cervical malignant lymphoma.

**Keywords:** carotid sinus syndrome; malignant lymphoma; surgical intervention

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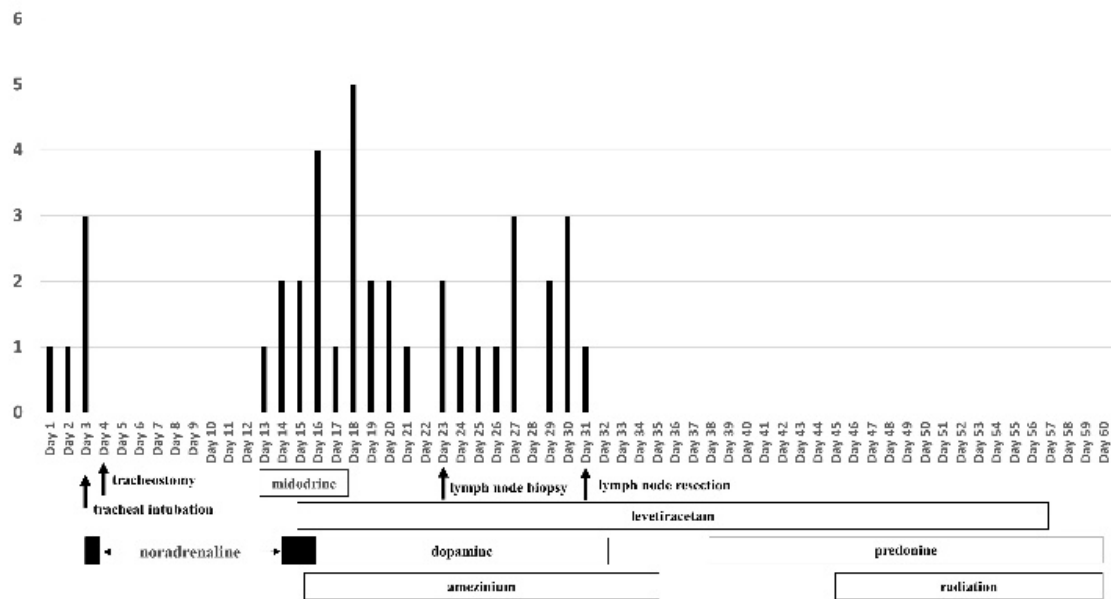
## 1. Introduction

It has long been observed that pressure at the site where the common carotid artery bifurcates produces a reflex that leads to slowing in heart rate and fall in blood pressure. In some individuals an abnormal response to carotid massage is observed. A ventricular pause lasting more than 3 seconds and/or a fall in systolic blood pressure of more than 50 mm/Hg defines carotid sinus hypersensitivity. When associated with spontaneous syncope, an abnormal response to carotid massage defines the carotid sinus syndrome. [1,2] We report a case of carotid sinus syndrome caused by treatment-resistant cervical malignant lymphoma, which was alleviated through lymph node biopsy and subsequent treatment. We provide a literature review and discuss the implications of this case..

## 2. Case Report

A 79-year-old male with a medical history of hypertension, renal stones, and gastroesophageal reflux disease presented with a decrease in blood pressure from around 140 mmHg one month ago to approximately 120 mmHg. During this time, he experienced progressive weight loss due to poor appetite, and episodes of transient loss of consciousness and

choking emerged. He sought consultation with neurology and otolaryngology, where he was diagnosed with neurocardiogenic syncope and idiopathic recurrent vagal nerve paralysis. While waiting for a follow-up appointment at the otolaryngology outpatient clinic, the patient experienced a loss of consciousness, prompting a code blue. As healthcare providers were unable to palpate the carotid pulse, chest compressions were initiated, leading to the prompt restoration of cardiac activity. The patient was subsequently transported to the emergency department. There is no significant family medical history. Upon arrival at the emergency department, the vital signs were as follows: Glasgow Coma Scale, E4V5M6; blood pressure, 131/81 mmHg; heart rate, 73 beats/min; respiratory rate, 16 breaths/min; percutaneous saturated oxygen, 99% with 4L of oxygen; and body temperature, 36.5°C. The patient's height was 163 cm, and weight was 51 kg. Physical examination revealed findings related to the loss of consciousness: right eyelid swelling and subcutaneous bleeding due to the fall during the fainting episode, as well as right anterior chest pain attributed to chest compressions. Blood test results are presented in [Table 1](#), showing mild malnutrition and an elevation in lactate dehydrogenase. A whole-body computed tomographic scan (CT), aside from age-related changes, did not reveal any significant abnormalities. In order to investigate the recurrent episodes of syncope, the decision was made to admit the patient for a thorough examination. The course of syncope frequency and the main treatment details after admission are illustrated in [Figure 1](#).



Treatment-resistant syncope and hypotensive episodes persisted until the 31st day of hospitalization.

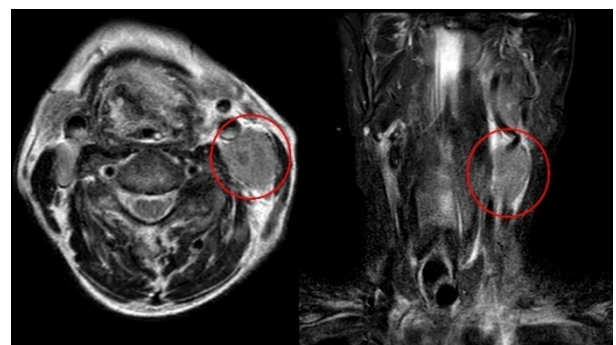
**Figure 1.** The course of syncope frequency and the main treatment details after admission

**Table 1. Blood test results**

variables	data	unit
white blood cell count	7.3	$10^3\mu\text{L}^{-1}$
hemoglobin (Hb)	13.5	gdL-1
platelets	177	$10^4\mu\text{L}^{-1}$
total protein	5.8	gdL-1
albumin	3.5	gdL-1
total bilirubin	1.9	mgdL-1
aspartate aminotransferase	23	UL-1
alanine aminotransferase	23	UL-1
cholinesterase	173	UL-1
gamma-glutamyl transpeptidase	17	UL-1
alkaline phosphatase	64	UL-1
lactate dehydrogenase	263	UL-1
glucose	130	mgdL-1
HbA1C	5.7	%
blood urea nitrogen	11.5	mgdL-1
creatinine	0.82	mgdL-1
creatinine kinase	91	UL-1
amylase	49	UL-1
uric acid	5.5	mgdL-1
sodium	140	mmolL-1
potassium	3.5	mmolL-1
chloride	100	mmolL-1
C reactive protein	0.72	mgdL-1
prothrombin time	11.9	second
activated partial thrombin time	30.2	second
fibrinogen	453	mgdL-1
fibrinogen degradation product	3.9	$\mu\text{gmL}^{-1}$

Initially, both epilepsy and autonomic dysfunction were considered, and simultaneous treatments for both conditions were initiated. However, treatment-resistant

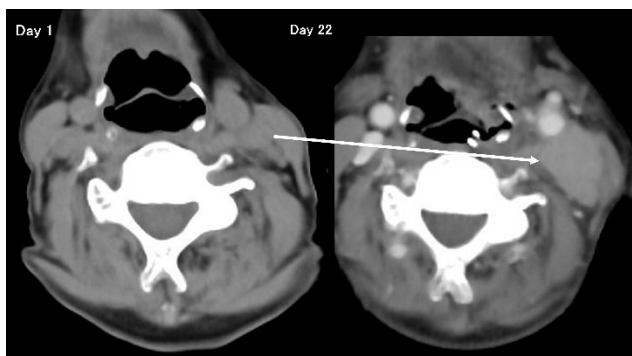
syncope and hypotensive episodes persisted until the 31st day of hospitalization. During this period, investigations into the cause of syncope were conducted. On the 2nd day, echocardiography, tilt-table testing on the 5th day, Holter electrocardiography, and electroencephalogram on the 8th day, and head magnetic resonance image (MRI) on the 8th day were performed. Additionally, a lumbar puncture was conducted on the 9th day. Except for age-related changes detected on the head MRI, no abnormalities were found. On the 19th day, a neck MRI revealed swelling of lymph nodes around the carotid artery from the upper part of the clavicle, leading to the diagnosis of carotid sinus syndrome caused by this swelling (Figure 2).



The MRI revealed swelling of lymph nodes around the carotid artery from the upper part of the clavicle (circle), leading to the diagnosis of carotid sinus syndrome caused by this swelling.

**Figure 2.** Neck magnetic resonance image (MRI) on the 19th day.

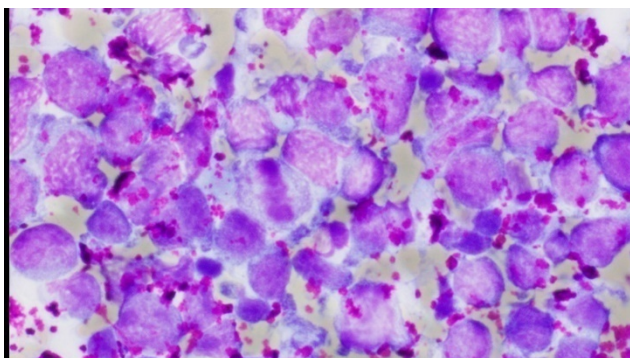
On the 22nd day, contrast-enhanced CT was performed, revealing a rapid progression of swelling in the neck lymph nodes compared to the neck CT findings on the 1st day (Figure 3).



The CT on the 22nd day revealed a rapid progression of swelling in the neck lymph nodes compared to the neck CT findings on the 1st day (arrow).

**Figure 3.** Neck computed tomography (CT) on the 22nd day

On the 23rd day, a lymph node biopsy was performed, revealing the presence of large lymphoma cells with monotonous appearance, nuclear irregularities, and fine mesh-like chromatin (Figure 4).



On the 23rd day, a lymph node biopsy was performed, revealing the presence of large lymphoma cells with monotonous appearance, nuclear irregularities, and fine mesh-like chromatin. Prominent nucleoli were also observed, suggesting a cellular pattern indicative of malignant lymphoma.

**Figure 4.** Results of a biopsy of a neck lymph node (hematoxylin and eosin stain, x 400)

Prominent nucleoli were also observed, suggesting a cellular pattern indicative of malignant lymphoma. On the 31st day, a Rapid Efflux of Epithelial Detritus biopsy of the lymph node was conducted, and during this procedure, thorough lymph node dissection was performed. Following the diagnosis of diffuse large B-cell lymphoma, the episodes of syncope ceased. Due to weight loss associated with swallowing difficulties, steroid therapy was initiated, and radiation therapy commenced on the 45th day. After treatment for malignant lymphoma in the neck, he was transferred for rehabilitation purposes in 103 days due to persisting dysphagia and disuse atrophy.

### 3. Discussion

This case involved treatment-resistant carotid sinus syndrome induced by malignant lymphoma occurring in the cervical lymph nodes. Similar reports have been documented in the past, and it is not uncommon for malignant lymphoma affecting the cervical lymph nodes to be a causative factor for carotid sinus syndrome. [3,4,5,6]

Carotid sinus syndrome is a type of vasovagal syncope. Vasovagal syncope is a very common form of fainting. Treatment begins with patient education, non-pharmacological approaches including increases in dietary salt and water intake, the use of compression garments, physical counter-maneuvers and tilt-training. When these approaches are inadequate, medications including alpha-1 agonists, mineralocorticoids, selective serotonin reuptake inhibitors or beta-blockers can sometimes be effective. [7,8] While the present case showed no effective for occurrence of vasovagal syncope by both non-pharmacological and pharmacological approaches.

In this case, a refractory carotid sinus syndrome was induced, but the syncope episodes disappeared following lymph node dissection and subsequent treatment. In the past, Dziekiewicz and others have compiled reports summarizing cases where surgical treatment led to improvement in symptoms of carotid sinus syndrome. In their study, all patients with CSS who received more extensive unilateral carotid artery denervation than demonstrated in other studies were symptom free at 30 days after surgery and, importantly, showed no clinically relevant impairment of cardiovascular autonomic control. [9] In a study by Toorop et al also reported that carotid denervation by adventitial stripping of the ICA in patients with CSS, 93% of the subjects were free from symptoms. [10] In this case, although denervation of the internal carotid artery was not performed, the lymph node dissection in the neck led to a form of denervation of the branches of the vagus nerve. It is speculated that this denervation, combined with subsequent treatment, contributed to the control of abnormal vagus nerve activity. When faced with a non-responsive carotid sinus syndrome to non-invasive treatments, including cases like this, consideration of interventions, including surgical approaches, may be necessary.

For the treatment of the current case, we opted for steroid and radiation therapy. This decision was based on the patient's Lugano classification (2014) of stage II and an international prognostic index score of 3, indicating a poor prognosis. Additionally, the patient experienced weight loss associated with swallowing difficulties. In the future, it may be necessary to consider completing the therapy as per guidelines (chemotherapy + anti-CD20 monoclonal antibodies) due to the tendency of this lymphoma type to recur. [11]

### 4. Conclusion

This sentence describes a case presentation where syncope episodes disappeared in a patient with treatment-resistant carotid sinus syndrome caused by cervical malignant lymphoma. The resolution was achieved through lymph node dissection and subsequent treatment. The statement emphasizes the consideration of surgical interventions, including in cases where non-invasive treatments are ineffective for carotid sinus syndrome.

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## Conflicts of Interest

The authors declare no conflicts of interest in association with this study.

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