



Spontaneous Early Recanalization of an Acute Internal Jugular Vein Thrombosis

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Acute internal jugular vein thrombosis (IJVT) is a rare vascular disease that can cause lethal complications and manifests with varying neurological symptoms. A 42-year-old woman presented with a progressively worsening headache over the left temporal-occipital area and pain in the left posterior nuchal; ultrasonography and magnetic resonance venography (MRV) revealed a thrombosis with critical stenosis in the left internal jugular vein (IJV). Seven days after treatment with an anticoagulant, she exhibited considerable improvement. Follow-up ultrasonography and three-dimensional computed tomography angiography (CTA) indicated spontaneous IJV recanalization. To avoid implementing inappropriate treatments and prevent adverse outcomes, acute IJVT should be considered as a cause of secondary headaches. To determine the prognosis and secondary prophylaxis strategy (i.e., medical or surgical), recanalization should be detected using color-coded duplex ultrasonography and confirmed using MRV or CTA; these are all safe and minimally invasive procedures.

Key words: Anticoagulants, headache, internal jugular vein thrombosis, recanalization

INTRODUCTION

Most people experience headaches, and patients presenting with primary headache should be differentiated from those who present with secondary headache. Secondary headache is linked to vascular, infectious, neoplastic, and elevated intracranial pressure causes, which are urgent medical problems that must be identified early. Internal jugular vein thrombosis (IJVT) is an elusive and rare condition that is related to secondary headaches and has an incidence of 0.5%–3.47% among patients with deep vein thrombosis (DVT),¹ although this may be underestimated because some patients are asymptomatic.² IJVT can cause lethal complications, including pulmonary embolism and intracranial propagation of the thrombus. The causes of IJVT are categorized as either primary or secondary, with the main cause of secondary IJVT being foreign bodies such as central venous catheters or pacemakers. Other secondary etiologies include radical neck dissection, malignancy, infection, coagulation abnormalities,

ovarian hyperstimulation syndrome,^{3,4} and autoimmune disorders.^{1,5,6} The clinical presentations of IJVT vary from common arm edema and pain to intracranial hypertension with severe headache, focal neurological abnormalities, and seizures. However, it can also be asymptomatic.⁷ Therefore, the diagnosis of IJVT is often challenging and requires an attentive and persistent physician.

CASE REPORT

A 42-year-old Asian woman, who was an office worker with a medical history of papillary thyroid cancer (which was in a stable posttreatment state) and migraine, presented with a progressively worsening headache over the left temporal-occipital area and pain in the left posterior nuchal that she experienced for 7 days. Symptoms such as pulsating pain, nausea, vomiting, photophobia, and phonophobia were not reported. However, the patient's condition worsened upon awakening and coughing.

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A physical examination indicated no cervical edema, carotid bruit, or erythematous changes. None of the laboratory studies that were performed (including clotting screen and blood viscosity tests, autoimmune marker screening, and metabolic deficiency testing) yielded any notable findings. The results of the laboratory studies were as follows: Human immunodeficiency virus Ab/Ag, negative; homocysteine, 5.58 $\mu\text{mol/L}$; rheumatic factor, 20 IU/mL; antistreptolysin O titer, 25 mg/dL; C3, 104 mg/dL; C4, 21.3 mg/dL; IgG, 834 mg/dL; IgM, 111 mg/dL; IgA, 182 mg/dL; D-dimer, 0.16 mg/dL; erythrocyte sedimentation rate, 5 mm/h; antithrombin III, 90.8%; lupus anticoagulant, 1.2%; protein C, 117.7%; protein S, 37.6%; rapid plasma reagin, nonreactive; thyroid-stimulating hormone, 0.03 uIU/mL; free T4, 1.84 ng/mL; antithyroid peroxidase antibody, negative; anti-Tg, negative; anticardiolipin IgG, 852 U/m; antinuclear antibody titer, 1:80; alpha-fetoprotein, 1.34 ng/mL; carcinoembryonic antigen, 1.14 ng/mL; cancer antigen 125, 10.56 U/mL; cancer antigen 153, 12.21 U/mL; cancer antigen 19-9, 8.24 ng/mL; squamous cell carcinoma, 0.5 ng/mL; and HbA1c, 5.8%. A chest X-ray revealed normal results. However, a color-coded duplex ultrasonography (CCDU) revealed a thrombosis with critical stenosis in the left internal jugular vein (IJV), which was confirmed using magnetic resonance venography [MRV; Figure 1]. All other potential causes were excluded, including recent intravascular intervention, pacemaker implantation, and contraceptive use. The patient was administered subcutaneous low-molecular-weight heparin (LMWH; enoxaparin) for 5 days, followed by oral warfarin as anticoagulant therapy (2.5 mg of warfarin was initially administered every day; subsequently, a titrated dosage per the effective international normalized ratio range was administered). Rapid regression of symptoms was observed by the 7th day. The recanalization of a previously highly stenosed IJV was observed in CCDU and confirmed through computed tomography venography [CTV; Figure 2]. The patient was discharged 2 weeks later and underwent an additional 28-day warfarin treatment. During the 1-year

follow-up, no recurrent thrombosis events or residual neurological sequelae were reported.

DISCUSSION

The median age at which patients, who are predominantly female, experience IJVT is approximately 40 years. The clinical features of IJVT are pain (76.5%) and cervical edema (85.7%, particularly in patients with underlying head/neck disorders); other signs include arm edema (12.2%), superficial varicose collateral veins (13.2%), and indurated vein (8.1%).¹ Idiopathic IJVT comprises about 17% of all IJVT cases,⁸ and complications include pulmonary embolism, postthrombotic syndrome, and death.³

For adults patients, the mnemonic SNOOP (i.e., systemic symptoms/signs and disease, neurologic symptoms or signs, sudden onset or onset after the age of 40 years, and change in headache pattern) should always be kept in mind when diagnosing secondary headaches. The patient in the present case was older than 40 years and presented with a new headache pattern relative to her history of migraine, all of which alerted clinicians to her condition. On the basis of the pattern change and with a focus on intracranial hypertension because the symptoms were exacerbated on the patient waking up and coughing, status migrainosus was ruled out. We also considered malignance given the patient's history of papillary thyroid cancer. After all other potential causes were ruled out, the patient was diagnosed as having idiopathic IJVT.

In patients with headache, noninvasive neuroimaging techniques (such as CCDU, computed tomography, and magnetic resonance imaging) are used in secondary headache diagnoses that focus on vascular abnormalities, infection or inflammation, and mass lesion-related intracranial hypertension. Similar to the cause of symptoms reported in previous cases, the main symptoms of our patient were caused by increased intracranial pressure, and secondary headache was diagnosed through CTV and MRV. The patient was then administered anticoagulants. We employed ultrasound only

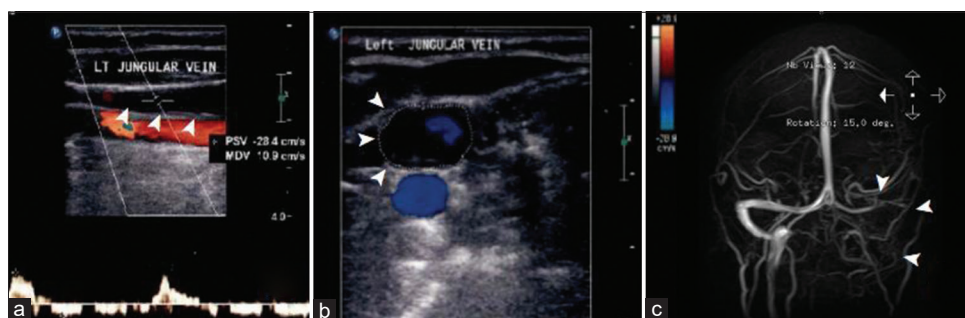


Figure 1: (a) Color-coded duplex ultrasonography: Right jugular vein exhibiting normal blood flow. (b) Left jugular vein engorgement exhibiting no blood flow. (c) Left jugular vein exhibiting partial blood flow indicative of jugular vein thrombosis

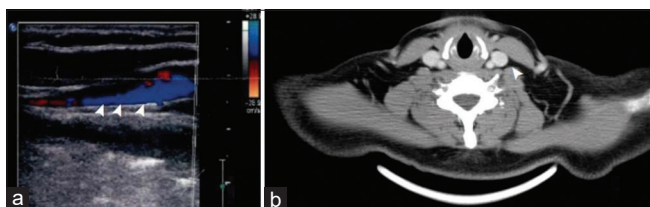


Figure 2: After 7 days of treatment, (a) the left jugular vein exhibited normal blood velocity on color-coded duplex ultrasonography. (b) Computed tomography with contrast indicated normal contrast opacification of the bilateral transverse-sigmoid sinuses and jugular veins

for early detection and followed up with the patient to ensure the timely resolution of her thrombosis. The patient recovered well without any neurological sequelae.

When applying therapeutic strategies, the underlying disease should be treated, and an anticoagulant should be administered. The benefits of anticoagulant administration have not been clarified due to the lack of randomized controlled trials with cases involving this rare thrombosis site. A case series conducted by Payrard *et al.*⁸ revealed that anticoagulants with intravenous unfractionated heparin or LMWH (i.e., enoxaparin, tinzaparin, and dalteparin) – sometimes accompanied by Vitamin K antagonists (acenocoumarol and warfarin), also, NOACs (i.e., edoxaban, apixaban, and rivaroxaban) and even aspirin were once used in respective case reports. The aforementioned treatments lasted from 2 weeks to 6 months.⁸ The decision to initiate anticoagulation treatment must be based on documented and confirmed acute DVT, involvement of multiple venous segments, and young age.⁹ In a few IJVT cases, thrombolysis, surgery, thrombectomy, and balloon dilatation with or without stent placement have been performed. Further studies are required to establish evidence-based guidelines for IJVT.

The main complication of IJVT is pulmonary embolism, followed by mortality caused by underlying malignancy and non-major hemorrhagic manifestations related to anticoagulant therapy. In a study with a 3–6-month follow-up, persistent thrombus was detected in 77.3% of patients through CCDU, and 41.4% of them developed postthrombotic syndrome but did not experience IJVT relapse.¹ However, our patient developed thrombosis and was symptom-free after undergoing 7 days of medical treatment. Furthermore, no further relapses were reported.

We emphasize the timely, noninvasive detection of IJVT and the early, complication-free recanalization of occluded vessels. Conservative treatment with systemic anticoagulants is recommended, and ligation and resection of the thrombus should be reserved for patients with contraindications and those who develop medication-related complications.¹⁰

Clinicians must differentiate the causes of IJVT, particularly in women; the causes are as follows: Malignancy,

infection, inflammation, coagulation abnormalities, ovarian hyperstimulation syndrome, and autoimmune or structural causes.

CONCLUSION

We recommend that physicians consider IJVT as a cause of secondary headache. Early detection (using CCDU, MRV, or CTV) and recanalization through systemic anticoagulant use are safe and minimally invasive procedures, and they are essential in ensuring appropriate and timely management and preventing complications and residual sequelae.

Ethical approval

The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee of the institute (IRB: C202005174). Informed written consent was obtained from all patients prior to their enrollment in this study.

Declaration of patient consent

The authors certify that they have obtained all the required patient consent forms. By signing the forms, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published, and due efforts will be made to conceal her identity, although anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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