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Deep Vein Thrombosis Following Herpes Zoster: A Rare Case Report

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Abstract | **Case Report**

Herpes zoster is a viral infection (HZ) caused by the reactivation of the varicella-zoster virus (VZV), commonly in older adults. While postherpetic neuralgia is the most frequent complication, venous thrombosis associated with HZ is extremely rare. We report a 59-year-old male with extensive upper limb deep vein thrombosis in the context of HZ reactivation. Despite anticoagulation, antiviral therapy, and antibiotics for bacterial superinfection, the patient succumbed. This case emphasizes the importance of considering thrombotic complications following HZ, even without traditional risk factors.

Keywords: Herpes zoster, Varicella-zoster virus, Deep vein thrombosis.

INTRODUCTION

Herpes zoster results from the reactivation of latent VZV and increases sharply after age 50 due to declining cell-mediated immunity [1–3]. It usually presents with unilateral neuropathic pain followed by a dermatomal vesicular rash. While postherpetic neuralgia is the most known complication, vascular complications such as deep vein thrombosis (DVT) are rarely reported. We present a case of extensive upper limb DVT associated with HZ in a 59-year-old man.

CASE PRESENTATION

A 59-year-old chronic smoker presented with painful swelling of the right upper limb for 20 days, associated with mild fever. Examination revealed grouped vesicles in a dermatomal distribution over the right intercostal area, with a warm, tender, erythematous plaque extending to the cervical region and upper limb. The clinical appearance is consistent with thoracic shingles (Figure 1).

Given the swelling and worsening condition of the limb, an emergency Doppler ultrasound revealed thrombosis of the right internal jugular vein, the brachiocephalic trunk, and the subclavian vein. The patient was started on therapeutic low-molecular-weight heparin after normal renal function and platelet count.

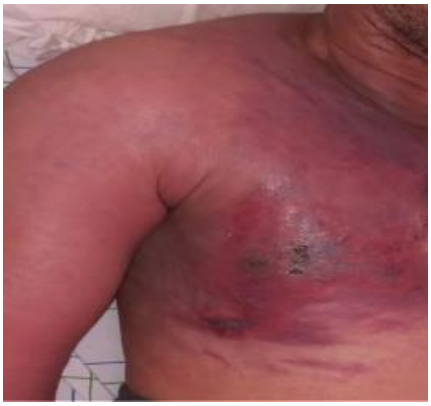

Figure 1: Vesicular lesions and erythematous plaque over the right hemithorax and upper limb, consistent with herpes zoster

Laboratory tests showed a hemoglobin level of 12 g/dL, leukocytosis (11,600/µL, predominantly monocytic), and an elevated CRP (72 mg/L). Tumor markers, viral serologies (HBV, HCV, HIV, syphilis), and autoimmune testing (ANA, antiphospholipid antibodies, ANCA) were negative. The patient received valacyclovir (1.5 g/day) and pregabalin (150 mg/day) for cutaneous herpes zoster.

The condition worsened, with the skin becoming infected and the lesions developing. The patient was started on triple antibiotic therapy (ceftriaxone, metronidazole, gentamicin). Despite treatment, the patient died from septicemia.

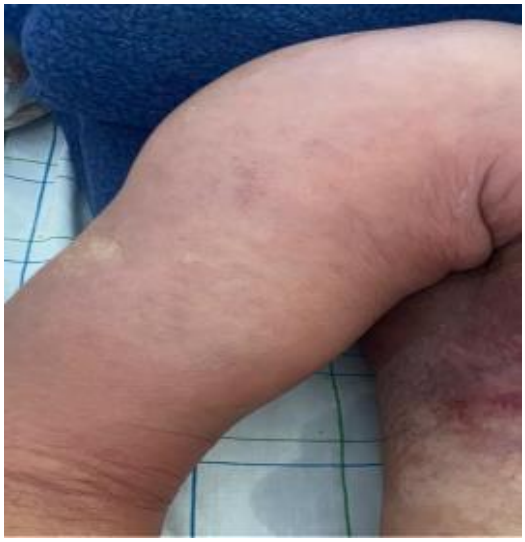

Figure 2: Marked swelling and erythema of the right upper limb, consistent with deep vein thrombosis

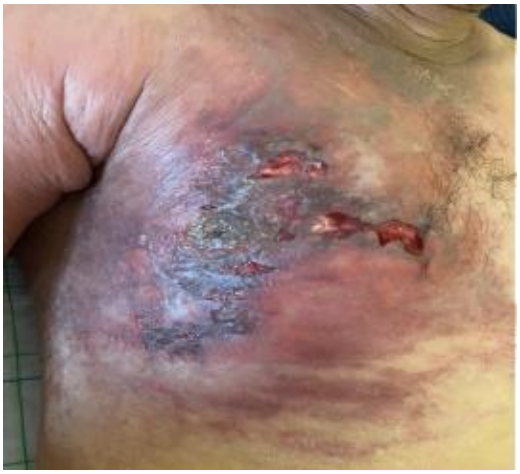

Figure 3: Extensive dermatomal vesicular eruption over the right intercostal and cervical area

DISCUSSION

The association between the reactivation of the Varicella-Zoster Virus (VZV) and the subsequent development of thrombotic complications—whether arterial (primarily manifesting as ischemic strokes) or venous (deep vein thrombosis or cerebral venous sinus thrombosis)—represents a significant clinical entity. While increasingly documented in the literature, it remains frequently underdiagnosed in clinical practice. This discussion evaluates the underlying pathophysiological mechanisms, the temporal relationship of clinical manifestations, and current diagnostic and therapeutic strategies based on contemporary evidence.

Pathophysiology: From Viral Reactivation to Thrombosis

The primary mechanism by which VZV induces thrombotic events is VZV vasculopathy (or granulomatous vasculitis). VZV exhibits unique neurotropism and vasculotropism in humans.

Following reactivation within the sensory ganglia (cranial or spinal), the virus undergoes transaxonal migration along afferent nerve fibers to reach the adventitia of adjacent blood vessels (*Nagel et al.,2014*). The infection then spreads transmurally, progressing from the adventitia through the media to the intima.

This direct viral infiltration triggers a cascade of pro-thrombotic pathological alterations:

- **Intimal Hyperplasia and Endothelial Dysfunction:** Viral infection disrupts the integrity of the endothelial barrier, promoting platelet adhesion and activating the coagulation cascade
- **Disruption of the Internal Elastic Lamina:** This leads to the depletion of smooth muscle cells, weakening the vascular wall and predisposing the vessel to both thrombotic occlusion and aneurysmal remodeling (*Gilden et al.,2002*).
- **Transmural Inflammatory Infiltration:** Characterized by an initial influx of neutrophils, followed by CD4+ and CD8+ T lymphocytes and macrophages (*Nagel et al.,2011*). These cells secrete pro-inflammatory cytokines that sustain a localized hypercoagulable state.
- **Pro-thrombotic Role:** Severe local inflammation induces the expression of tissue factor on the surface of endothelial cells and infiltrated macrophages, thereby triggering the extrinsic pathway of coagulation and culminating in *in situ* thrombosis (*Livieratos et al.,2025*).

Chronology and Clinical Spectrum: A Diagnostic Challenge

The primary clinical hurdle in post-zoster thrombotic vasculopathy is the temporal dissociation between the cutaneous eruption and the vascular event. Epidemiological data indicate that the risk of ischemic stroke or thrombosis peaks within the first two weeks following the zoster episode; however, this risk remains significantly elevated for up to 6 months, and in some cases, a year post-infection (*Lin et al.,2010; Kawai et al.,2014*).

Furthermore, the literature describes cases of thrombotic vasculitis occurring in the absence of a preceding rash (zoster sine herpete), a phenomenon that frequently complicates the diagnostic process (*Gilden et al.,2015*).

Thrombotic risk is particularly pronounced in cases of herpes zoster ophthalmicus (V1). Due to the anatomical proximity of the trigeminal ganglion to major cerebral arteries (internal carotid and middle cerebral arteries), the hazard ratio for an ipsilateral ischemic stroke can increase more than fourfold in the weeks following cutaneous involvement (*Lin et al.,2010*).

While arterial involvement is the hallmark, venous complications such as Cerebral Venous Sinus

Thrombosis (CVST) have also been reported, illustrating VZV's capacity to disrupt hemostasis across the entire cerebral vascular network (*Wu et al.,2025*).

Therapeutic and Preventive Implications

Management necessitates a dual strategy targeting both viral replication and the destructive inflammatory response:

- **Antiviral Therapy:** Early administration of intravenous acyclovir (10 mg/kg every 8 hours for 10 to 14 days) is the cornerstone of treatment to halt VZV replication within the vascular walls.
- **Adjuvant Corticosteroids:** The addition of corticosteroids (e.g., methylprednisolone or prednisone) is strongly recommended to dampen transmural inflammatory infiltration, stabilize the vascular wall, and reduce the risk of thrombosis extension or recurrence (*Livieratos et al.,2025*).
- **Antithrombotic Management:** The use of antiplatelet agents or anticoagulants should be determined on an individual basis, carefully weighing the benefit-to-risk ratio, especially given the potential for aneurysmal formation.

Finally, regarding prevention, recent data emphasize the critical role of zoster vaccination (specifically the recombinant subunit vaccine). By preventing VZV reactivation, vaccination significantly reduces the incidence of post-herpetic neuralgia and has been shown to lower the long-term risk of secondary cardiovascular and cerebrovascular events (*Livieratos et al.,2025*).

Literature Summary

	Site of thrombosis	Treatement	Evolution
Pranab Kumar Maity1, *et al.,*	Thrombosis of the left common iliac vein and the external iliac vein, the common femoral vein; left superficial femoral vein, and also in the right superficial femoral vein and right popliteal vein	Heparin Oral warfarin Oral acyclovir	Good
Syeda Sahraa *et al.,*	Segmental pulmonary embolism in the anterior segment of the right lower lobe	Dexamethasone and valacyclovir were initiated. Anticoagulant therapy with low molecular weight heparin 50 mg twice daily was started. The antiviral was replaced with IV acyclovir.	Good response to treatment with moderate residual facial dysfunction
Woo-Yeon Choi, *et al.,*	Segmental pulmonary embolism in the anterior segment of the right lower lobe	Dexamethasone and valacyclovir were initiated. Anticoagulant therapy with low molecular weight heparin 50 mg twice daily was started. The antiviral was replaced with IV acyclovir.	Good progress in skin lesions
Our cas	nternal jugular vein; of the innominate trunk and of the right subclavian vein	Low molecular weight heparin at therapeutic doses; valacyclovir 1.5 g daily combined with intravenous triple therapy: ceftriaxone 2 g daily; metronidazole 500 mg three times daily; gentamicin sulfate 240 mg daily. Pregabalin 150 mg daily for postherpetic neuralgia.	Death

CONCLUSION

Although rare, DVT can complicate herpes zoster and may be life-threatening. Clinicians should be aware of this possibility, especially in elderly patients, and consider early imaging, anticoagulation, and antiviral therapy.

Ethics Statement: Informed consent for publication of clinical details and images was obtained from the patient's family.

Conflicts of Interest: The authors declare no conflicts of interest.

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