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# DEEP VENOUS THROMBOSIS - A RARE COMPLICATION OF HERPES ZOSTER

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## ABSTRACT

**Thrombosis is a rare complication of varicella zoster virus infection and this occurs secondary to endothelial cell damage and coagulation abnormalities. A twenty-eight years female with no underlying comorbidities presented with Deep vein thrombosis of left popliteal vein following Herpes zoster infection. Working up for hypercoagulable states revealed, Antiphospholipid IgG and IgM antibodies were positive and protein C levels was mildly decreased. She was started on anti-coagulation and responded well after 3 months of follow up, the repeat testing for antiphospholipid antibodies turned negative and protein C levels were normalized. Herpes zoster infection associated with an acquired hypercoagulable state, exhibiting transient protein C and S deficiency and transient antiphospholipid antibody positivity. Early initiation of anticoagulant therapy in these patients results in decreased morbidity and good prognosis.**

**KEYWORDS:** Deep Venous Thrombosis, Herpes zoster, Antiphospholipid antibodies

Varicella Zoster Virus also known as Human Herpes virus – 3, is a double stranded DNA alpha herpes virus. It infects humans without animal reservoir. It primarily manifests as varicella infection (Chicken pox) more commonly in childhood. Virus later becomes latent in cervical and spinal ganglionic neurons. Declining in cell mediated immunity leads to reactivation of virus which manifests as Zoster also known as Shingles (Gershon *et al.*, 2015). Complications of Herpes Zoster include post herpetic neuralgia, pneumonia, meningoencephalitis, zoster paresis, ophthalmic disease and vasculopathy (Nagel and Gilden, 2013). Thrombosis is a rare complication of varicella zoster virus and this occurs secondary to endothelial cell damage and coagulation abnormalities (Nicholson and Hajjar, 1999). Here we discuss a case report of young immunocompetent female with no underlying comorbidities presented with Deep vein thrombosis of left leg following Herpes zoster infection.

## CASE REPORT

A twenty-eight years old female presented with subacute onset of pain, swelling and redness over her left lower limb from below the knee upto foot since one month. No trigger factors were present for the current episode except her history of painful vesicular rash over the left hypochondriac area 10 days prior to the leg symptoms. No history of breathlessness, trauma, fever. On examination, general condition is good, she is afebrile, Pulse rate is 92beats/min, Blood pressure is 116/70mmHg, Respiratory rate is 16breaths/min. She had mutiple hyperpigmented patchy lesions with few scabs and crusts over the left T8,9,10 dermatomes. Mild erythema and diffuse swelling of left lower limb from below knee to ankle. On palpation mild induration and warmth over left leg present. Homans sign was positive. Venous doppler of left leg revealed Deep vein thrombosis of popliteal vein. On further workup for hypercoagulable states, Anti thrombin III was normal, Protein C was 60 (70 – 140) mildly decreased, protein S was normal, Lupus anticoagulant was negative, Anti cardiolipin antibodies IgM and IgG was negative. Antiphospholipid antibodies IgG 146 (<10) and IgM 32 (<10) were positive. She was started on rivaroxaban and 3 weeks later the pain, swelling and erythema of left leg improved. After 3 months of follow up, she is doing good and the repeat testing for antiphospholipid antibodies turned negative and protein C was normal. Based on the history and evidence of healed herpetic lesions with no other triggers, her past thrombotic episode was attributed secondary to herpes zoster infection.

## DISCUSSION

The complications resulting from varicella zoster virus reactivation was complex and many atypical presentations was noted. One among them was thrombotic manifestations. Thrombotic complications secondary to varicella infection were mainly described in children, however few case reports were documented in adults. Examples of them are cortical venous thrombosis, pulmonary thromboembolism, tibial artery thrombosis, Deep venous thrombosis of legs (Mehkri *et al.*, 2022) (Salvotti *et al.*, 2023) (Sahra *et al.*, 2021) (Peyton *et al.*, 1998) (Maity *et al.*, 2014). The underlying pathology for

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the increased risk of thrombosis was due to endothelial cell damage and procoagulant state. Herpes infections leads to increased binding of inflammatory cells to virus infected endothelial cells which establishes a procoagulant state (Nicholson and Hajjar, 1999). The coagulation abnormalities like protein S deficiency or generation of protein S antibodies secondary to viral cytotoxicity and inflammation was described (Deviley *et al.*, 2019). The positivity of antiphospholipid antibodies was also described (Uthman and Gharavi, 2002). Mancho Johnson et al reported that seven varicella infected children with purpura fulminans and or thrombosis, all had transient protein S deficiency and lupus anticoagulant and 4 out of 7 had antiphospholipid or anticardiolipin antibodies (Manco-Johnson *et al.*, 1996). Peyton *et al.* (1998) reported 2 cases of varicella pneumonia with thrombosis of profunda femoris and tibial arteries, free protein S deficiency was observed in both the cases and one patient had antiphospholipid IgG and IgM antibodies and one had lupus anticoagulant.

Our patient developed deep vein thrombosis after herpes zoster infection and she had a transient positive antiphospholipid antibodies. Viral infections are known to induce development of antiphospholipid antibodies. In some patients these antibodies are transient and disappears by few months. In some susceptible individuals the antiphospholipid antibodies persist (Uthman and Gharavi, 2002). Thus making infection a trigger for the development of antiphospholipid antibodies in autoimmune diseases.

With regards to treatment prompt and early initiation of anti coagulation reduces the further morbidity and mortality (Paul *et al.*, 2016).

## CONCLUSION

Herpes zoster infection associated with an acquired hypercoagulable state, exhibiting transient protein C and S deficiency and tranisent antiphospholipid antibody positivity. In our patient we observed the protein C deficiency and transient presence of antiphospholipid antibodies. All the cases of herpes zoster infection should be screened for any evidence of thrombosis. Early initiation of anticoagulant therapy in these patients results in decreased morbidity and good prognosis.

## REFERENCES

Deviley J.A., Oldenburg D., Berg L.C. and Agger W.A., 2019. Recurrent strokes, central nervous system vasculitis, and acquired protein S deficiency secondary to varicella zoster in a child with AIDS. J. Neurovirol, **25**(1):137-140. doi: 10.1007/s13365-018-0697-2. Epub 2018 Nov 26. PMID: 30478796.

Gershon A.A., Breuer J., Cohen J.I., Cohrs R.J., Gershon M.D., Gilden D., Grose C., Hambleton S., Kennedy P.G., Oxman M.N., Seward J.F. and Yamanishi K., 2015. Varicella zoster virus infection. Nat. Rev. Dis. Primers., **1**:15016. doi: 10.1038/nrdp.2015.16. PMID: 27188665; PMCID: PMC5381807.

Manco-Johnson M.J., Nuss R., Key N., Moertel C., Jacobson L., Meech S., Weinberg A. and Lefkowitz J., 1996. Lupus anticoagulant and protein S deficiency in children with postvaricella purpura fulminans or thrombosis. J. Pediatr., **128**(3):319-23. doi: 10.1016/s0022-3476(96)70274-3. PMID: 8774497.

Maity P.K., Chakrabarti N., Mondal M., Patar K. and Mukhopadhyay M., 2014. Deep vein thrombosis: a rare signature of herpes zoster. J. Assoc. Physicians India, **62**(1):72-4. PMID: 25327102.

Mehkri Y., Valisno J.A., Rivas L.F., Hernandez J., Jain V., Elfasi A., De Prey J., Hu C., Tarhan B., Tuna I.S. and Shuhaiber H.H., 2022. Management of Cavernous Sinus Thrombosis Following Herpes Zoster Ophthalmicus. Cureus., **14**(1):e21036. doi: 10.7759/cureus.21036. PMID: 35155004; PMCID: PMC8824491.

Nagel M.A. and Gilden D., 2013. Complications of varicella zoster virus reactivation. Curr Treat Options Neurol. **15**(4):439-53. doi: 10.1007/s 11940-013-0246-5. PMID: 23794213; PMCID: PMC3752706.

Nicholson A.C. and Hajjar D.P., 1999. Herpesviruses and thrombosis: activation of coagulation on the endothelium. Clin. Chim. Acta., **286**(1-2):23-9. doi: 10.1016/s0009-8981(99)00091-1. PMID: 10511282.

Paul G., Paul B.S. and Singh G., 2016. Unseen face of varicella-zoster infection in adults. Indian J. Crit. Care Med., **20**(12):731-734. doi: 10.4103/0972-5229.195713. PMID: 28149032; PMCID: PMC 5225775.

Peyton B.D., Cutler B.S. and Stewart F.M., 1998. Spontaneous tibial artery thrombosis associated with varicella pneumonia and free protein S deficiency. J. Vasc. Surg., **27**(3):563-7. doi: 10.1016/s0741-5214(98)70335-0. PMID: 9546247.

Sahra S., Jahangir A., Gavica M.C., Mobarakai N. and Jahangir A., 2021. First case report of pulmonary embolism with Zoster Sine Herpete. Respir Med Case Rep., **33**:101462. doi: 10.1016/j.rmcr.2021.101462. PMID: 34401298; PMCID: PMC 8349081.

Salvotti F., Trapletti S., Chiarini G., Castellano M. and Muiesan M.L., 2023. Atypical varicella-zoster virus reactivation: a case report. E.J.C.R.I.M., **10**:doi:10.12890/2023_003945

Uthman I.W. and Gharavi A.E., 2002. Viral infections and antiphospholipid antibodies. Semin Arthritis Rheum, **31**(4):256-63. doi: 10.1053/sarh.2002.28303. PMID: 11836658.