


CASE REPORT

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Herpes Zoster on a Giant Arteriovenous Fistula Aneurysm in an End-Stage Renal Disease Patient Undergoing Hemodialysis: A Case Report

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ABSTRACT

Introduction: Herpes zoster results from reactivation of the varicella-zoster virus. Patients with end-stage renal disease (ESRD) and those with physical trauma to the affected area are at increased risk of developing this condition. **Case**: A 31-year-old woman with ESRD and herpes zoster developed around a giant arteriovenous fistula aneurysm on her left arm. She was treated with acyclovir tablets and cream for 10 days, and her condition improved. **Discussion**: Immune dysfunction, especially immunodepression, contributes to the high incidence of infection in patients with end-stage renal disease. Decreased VZV-specific cell-mediated immunity is a pivotal factor for herpes zoster. Venous aneurysm of a hemodialysis access fistula is a relatively common complication of long-term arteriovenous fistula use. This is caused by high pressure in the fistula and frequent same-site puncturing. Mechanical trauma is reported to increase the risk of herpes zoster at the trauma site. **Conclusions**: Prolonged trauma to a specific site may predict the location of herpes zoster infection. Alternatively, herpes zoster at a specific site may suggest an unknown underlying abnormality in that dermatome.

Keywords: Arteriovenous fistula aneurysm, case report, end stage renal disease, hemodialysis, herpes zoster, trauma.

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INTRODUCTION

Herpes zoster is a localized cutaneous condition caused by reactivation of the varicella zoster virus (VZV), when immunity falls below a certain level. Herpes zoster lesions are typically unilateral and confined to the skin region innervated by a single sensory ganglion.[1] Herpes zoster disseminates interneurally and is confined to the skin area innervated by the affected nerve, as opposed to disseminating through viremia like varicella.[2] Risk factors for herpes zoster are age, decreased immunity, history of physical trauma in the affected dermatome, hemodialysis, and family history of herpes zoster.[1,2]

End-stage renal disease (ESRD) patients are more likely to contract herpes zoster due to their immunocompromised state.[3] Two needles are used to create a vascular access pathway in hemodialysis.[4] On account of the repeated puncturing of the fistula in the same location, the connective tissue fibers of the vein are destroyed and lose their mechanical strength, causing the vein wall to become flaccid and prone to develop aneurysmal dilatation.[5]

We report the case of a 31-year-old female with ESRD who developed herpes zoster at the site of a giant arteriovenous fistula aneurysm. She was treated with acyclovir tablets and acyclovir cream for 10 days, and her condition improved. We reviewed the relationship between the incidence of herpes zoster in patients with ESRD and the predilection of herpes zoster in site of the arteriovenous fistula aneurysm. ESRD patients should be more aware of the possibility of herpes zoster infection.

CASE

A 31-year-old female came to the internal medicine clinic for her monthly check-up for ESRD. She receives regular hemodialysis on Wednesdays and Saturdays and has had an arteriovenous fistula in her left arm for 7 years. Several large arteriovenous fistula aneurysms formed about 4 years ago. During this visit, she presented with itchiness and a stinging sensation on her left arm that began 5 days prior. Visible herpetiform papules had also formed along her left arm **(Figure 1).**

The patient was diagnosed with herpes zoster and treated with oral acyclovir 200 mg three times per day and topical acyclovir cream for 10 days. One month later, the patient still complained of itching, but the stinging was gone. Crusts and post-inflammatory hypopigmentation were seen along the patient's arm, while the arteriovenous fistula aneurysm remained unchanged **(Figure 2).** The patient was given additional oral cetirizine. The patient continued her hemodialysis therapy.

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*Photo documentation: Lidya Renaningtyas Setia Budi.

Figure 1. Herpes zoster on giant arteriovenous aneurysm. Herpetiformic papules with several large arteriovenous fistula aneurysms along the upper arm involving C6–C8 dermatome.



*Photo documentation: Lidya Renaningtyas Setia Budi.

Figure 2. Healed herpes zoster on giant arteriovenous aneurysm. Crustae and post-inflammatory hypopigmentation were seen along the patient's arm, while the arteriovenous fistula aneurysm still appeared the same.

DISCUSSION

VZV causes both varicella and herpes zoster. In varicella, VZV spreads from skin and mucosal surface lesions to the endings of adjacent sensory nerves, where it is transported up nerve fibers to the sensory ganglia. There, it establishes latent infection in neurons. The virus cannot be suppressed when immunity falls below a certain level.[1] The virus then multiplies and spreads throughout the ganglion, resulting in neuronal necrosis and inflammation. The virus then travels and is released from the skin nerve endings, forming the distinctive clusters of herpes zoster vesicles.[1,6] The key feature of herpes zoster lesions is their distribution and location. These lesions are typically unilateral and confined to the skin region innervated by a single sensory ganglion.[1] Herpes zoster lesions typically present as clusters of vesicles on an erythematous base. They do not propagate in the manner of chickenpox because herpes zoster disseminates interneurally and is confined to the skin area innervated by the affected nerve, as opposed to disseminating through viremia as in varicella. In 60%–90% of cases, skin eruptions are accompanied by pain.[2] The majority of people with herpes experience a range of sensations, from mild to severe, that are described as "burning", "deep aching", "tingling," or "stabbing".[1,7]

Herpes zoster is a condition associated with two main risk factors: age and decreased VZV-specific cell-mediated immunity. History of physical trauma in the affected dermatome, hemodialysis, female sex, psychological stress, and family history of herpes zoster are also factors linked to the risk of developing herpes zoster.[1,2,6] Herpes zoster is primarily associated with older age, but it has been demonstrated that immunosuppression in individuals with herpes zoster is linked to a substantially higher rate of complications.8 The risk is increased by immunosuppression, particularly in patients with hematological malignancies or HIV infection. The incidence is 30 per 1,000 persons per year in patients with HIV infection.[6] Immune dysfunction, particularly immunodepression, is a key factor in the high incidence of infection among patients with ESRD3. In ESRD patients, loss of kidney function leads to the buildup of cytokines and uremic toxins, creating an inflammation-prone environment that affects both innate and adaptive immunity. Immunological abnormalities related to viral infections include decreased natural killer cell numbers and activation, decreased antigen presentation and CD86 expression by dendritic cells, increased apoptosis, and decreased T and B cell function. This contributes to inadequate responses to viral infections in ESRD patients, such as herpes zoster.[8] Patients with ESRD are subject to a higher risk of herpes zoster. In the general population, the incidence is estimated at 3.9 per 1,000 person-years, while in patients with ESRD it is nearly 20 times higher, at 73.3 per 1,000 person-years.[8]

The primary objectives of antiviral therapy for patients with herpes zoster are twofold: first, to minimize the extent, duration, and severity of pain and rash within the primary affected dermatome; and second, to halt disease progression.[1] Acyclovir, valacyclovir and famciclovir are antiviral agents used to treat acute herpes zoster infection.[3] First-line treatment options for normal individuals include acyclovir (5 x 800 mg) for seven days, famciclovir (3 x 500 mg) for seven days, or valacyclovir (3 x 1 g) for seven days. For immunocompromised patients, continue treatment for seven to ten days **(Table 1)**.[1,6] Oral acyclovir, famciclovir, and valacyclovir are recommended for adult patients. Famciclovir and valacyclovir are preferred because they are taken only three times a day and have greater bioavailability, maintaining higher, longer-lasting antiviral levels in the blood.[2] In this case, the patient is being treated with acyclovir tablets, which are a commonly used and generally well-tolerated antiviral agent for the treatment of VZV reactivation in the general population.[3] In patients with ESRD, acyclovir's half-life increases significantly from 3 to over 20 hours, which increases the risk of neurological side effects and requires a dose adjustment.[3] In clinical practice, valacyclovir is the preferred choice because it requires less frequent dosing than acyclovir and is cheaper than famciclovir. But patients with impaired renal function may be particularly susceptible to the neurotoxicity associated with valacyclovir, as it is renally excreted.[3]




Table 1. Herpes zoster antiviral treatment in the normal and immunocompromised host.[1]

Patient Group	Regimen
Normal	
Age < 50 years	Symptomatic treatment alone, or • Famciclovir 500 mg orally every 8 h for 7 d OR • Valacyclovir 1 g orally every 8 h for 7 d OR • Acyclovir 800 mg orally 5 times a day for 7 d
Age ≥ 50 years, and patients of any age with cranial nerve involvement (e.g., ophthalmic zoster)	• Famciclovir 500 mg orally every 8 h for 7 d OR • Valacyclovir 1 g orally every 8 h for 7 d OR • Acyclovir 800 mg orally 5 times a day for 7 d
Immunocompromised	
Mild immunocompromise, including HIV-1 infection	• Famciclovir 500 mg orally every 8 h for 7–10 d OR • Valacyclovir 1 g orally every 8 h for 7–10 d OR • Acyclovir 800 mg orally 5 times a day for 7–10 d
Severe immunocompromise	• Acyclovir 10 mg/kg IV every 8 h for 7–10 d
Acyclovir resistant (e.g., advanced AIDS)	• Foscarnet 40 mg/kg IV every 8 h until healed

Abbreviations: HIV: Human immunodeficiency virus; e.g.: exempli gratia; AIDS: Acquired immunodeficiency syndrome; IV: Intravenous; mg: milligrams; h: hours; g: grams; d: days; kg: kilograms.

Using acyclovir cream in this case is inappropriate because topical antiviral treatment is not effective. Applying compresses or calamine lotion during the acute phase may reduce local symptoms and speed up the drying of vesicular lesions. Avoid occlusive glucocorticoid ointments, creams, or lotions.[1]

The consequences of ESRD are multifaceted and have the potential to induce dysfunction in multiple organs, including the skin.[9] Skin manifestations are common in patients with ESRD. At least one associated skin change occurs in 50%–100% of patients with ESRD.[9,10] These clinical manifestations may occur before or after the start of dialysis. In some cases, they may be the first sign of kidney disease.[9] According to Malkud, *et al*., and Falodun, *et al*., common complaints in uremic patients are xerosis, pruritus, and pallor.[9,10] Patients with ESRD are more prone to skin infections due to depressed neutrophil function, impaired phagocytosis, reduced T and B lymphocyte function, and decreased natural killer cell activity.[9] A study by Jeswani, *et al.,* reported skin infections in 17.0% of ESRD patients. 7.2% were fungal, 4.4% bacterial, 3.0% viral, and 3.6% parasitic.[11] A study by Malkud, *et al*., has found that viral infection is experienced by 8 out of 60 ESRD patients **(Table 2).**[9]

Two needles create a vascular access pathway for hemodialysis. The arterial line supplies blood to the extracorporeal circuit, incorporating the dialyzer, and the venous line returns blood to the patient within the ECC. The arterial line should supply at least 300 mL/min of blood through the artificial kidney. The preferred site for AV access is autogenous distal wrist radiocephalic access in the non-dominant arm.[4] In this case, the AV access was also located in the non-dominant forearm.

Because of elevated pressure and repeated puncturing of the fistula in the same location, the vein's connective tissue fibers are destroyed and lose their mechanical strength. This causes the vein wall to become flaccid and prone to develop aneurysmal dilatation. Plonski, *et al*.,[5] reported fistula aneurysms in 129 of 400 patients receiving dialysis via an arteriovenous fistula. The occurrence of a venous aneurysm of a hemodialysis access fistula is a relatively common complication of long-term use of an arteriovenous fistula (AVF). We define a venous aneurysm in the AVF as a diameter increase of 2 to 3 times the surrounding normal veins or a venous dilatation of 2 cm or more.[12] The term 'giant venous aneurysm' is used by some authors to refer to enormous venous aneurysms, although there are no specific criteria for size.[12]

The study by Zhang, *et al*., indicates that local factors, including physical trauma, contribute to the occurrence of herpes zoster. Reactivation of latent VZV by local factors suggests a completely different molecular mechanism than reactivation by suppression of cell-mediated immunity. Trauma can reactivate latent viruses by stimulating sensory nerves or disrupting cutaneous immunity. This can reactivate neurons and transport the virus to cutaneous sites, resulting in herpes zoster.[13] Physical trauma, including surgical trauma, is implied as a potential risk factor for herpes zoster. It has been demonstrated that "significant traumas may precipitate localized changes that may facilitate VZV reactivation" and that impairment to nerve fibers resulting from physical trauma or surgical intervention may predispose herpes zoster patients to more intense pain. The proposed molecular mechanism of reactivation is the suppression of cell-mediated immunity. There are documented cases of herpes zoster occurring after surgical and medical procedures, including liver biopsies, total joint arthroplasty, cystoscopic removal of ureter stones, incisions for central venous access ports, cryosurgery for solar keratosis, and shock wave lithotripsy.[14] The area most commonly affected by trauma-related shingles is the trunk and cranial regions, though the limbs may also be affected.[14]

A significantly elevated risk of herpes zoster was found to be associated with mechanical trauma in the study by Thomas, *et al*. The findings indicated that trauma elevated the risk of herpes zoster development at the site of the trauma, but not elsewhere. This suggests that traumatic nerve stimulation may provoke viral reactivation in the dorsal root ganglion of the affected nerve.[15] Foye, *et al*., reported herpes zoster affecting the right C5–C6 dermatomes of a hospital worker who suffered a traumatic hyperextension injury to her right wrist, hand and fingers.[16] Uchida, *et al*., reported a case of herpes zoster in a patient with a history of chronic hearing loss, cranial surgery following head trauma, coronary artery disease, and diverticulitis. The herpes zoster vesicles appeared on the right face near the ear, right chest, arm, and




Table 2. Various cutaneous manifestations in the study group.[9]

Clinical Manifestations		Number of Patients
Skin Changes	Xerosis	37
	Pruritus	35
	Pallor	28
	Pigmentary changes	14
	Acquired perforating dermatosis	3
	Gynecomastia	3
	Purpura and ecchymosis	1
	Bacterial infection	10
	Fungal infection	23
	Viral infection	8
Nail Changes	Koilonychia	14
	Half and half nail	8
	Nail dystrophy	6
	Onycholysis	6
	Subungual hyperkeratosis	6
	Beau's lines	5
	Leukonychia	3
	Pitting	2
	Clubbing	2
	Splinter hemorrhage	2
Oral Mucosal Changes	Glossitis	10
	Cheilitis	7
	Pigmentation of oral mucosa	8
	Xerostomia	6
	Macroglossia	1
Hair Changes	Dry lusterless hair	10
	Sparse body hair	4
	Diffuse alopecia	3

leg. This case suggests that trauma can trigger the spread of herpes zoster, even in an immunocompetent host.[17]

The herpes zoster vaccine can prevent herpes zoster and its complications. The target population for this vaccine includes healthy adults over 50 years of age, as well as individuals over 18 years of age with immunocompromised conditions.[18] Those with a history of varicella immunization are still at risk of developing herpes zoster. Two herpes zoster vaccines are available: the live attenuated zoster vaccine and the recombinant zoster vaccine. Both vaccines work by stimulating pre-immunization CD4+ T cells.[18]

The patient in this case was a 31-year-old female with ESRD who underwent routine hemodialysis through an AVF on her left arm and developed herpes zoster in the same dermatome. In addition to the immunosuppression associated with ESRD, additional factors including chronic local trauma, female sex, and hemodialysis are reported to increase the risk of herpes zoster.[1,2,6]

The appearance of herpes zoster directly over the AVF aneurysm in this patient is unlikely to be a coincidence. Several studies have demonstrated that localized trauma can act as a trigger for varicella-zoster virus reactivation within the dorsal root ganglion, as reported by Thomas, *et al*.,[15] and Zhang, *et al*.[13] In our patient, prolonged and repetitive needling and chronic pressure at the AVF aneurysm likely created local neural and tissue stress that predisposed the corresponding dermatome to viral reactivation. These findings support a plausible causal relationship between the AVF aneurysm site and the localization of herpes zoster in this case.

CONCLUSION

Patients with ESRD are more susceptible to herpes zoster infection due to impaired cell-mediated immunity. This case highlights an unusual presentation of herpes zoster involving a giant arteriovenous fistula aneurysm in a patient undergoing maintenance hemodialysis. Clinicians should maintain a high index of suspicion for herpes zoster in immunocompromised patients, particularly when atypical skin lesions develop around vascular access sites. Early recognition, prompt antiviral treatment, and appropriate wound care are essential to prevent further complications and preserve vascular access function. Furthermore, consider preventive strategies, including herpes zoster vaccination when clinically appropriate, in patients with ESRD.

DECLARATIONS

Consent for Publication

The patient provided written informed consent for publication of this case report.

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Author Contributions

LRSB and MPSB contributed to the conception and design of the case report, data collection, literature review, and manuscript drafting. ES contributed to the clinical management,




interpretation of findings, and critical revision of the manuscript. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of Interest

The authors declare no financial or non-financial conflicts of interest related to this case report.

Declaration on the Use of Artificial Intelligence (AI)

AI assistance was used during the preparation of this manuscript for language editing, wording refinement, and drafting assistance. The authors reviewed, verified, and approved the entire manuscript and remain fully responsible for its accuracy, originality, and final content.

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