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3 **Herpes Zoster infection in young adult in the Nabdam district of Ghana: a case report**

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5 **ABSTRACT**

6 The varicella zoster virus belongs to the family of alpha herpes viridae which is responsible for
7 both primary and recurring infections. Herpes zoster is a condition caused by the reactivation of
8 the varicella zoster virus. The reactivation follows a suppressed immune system or aging.
9 Herpes zoster mostly affects the aged population, however, it can also occur at any age. Herpes
10 Zoster is capable of affecting any sensory ganglia and its cutaneous nerve. The infections
11 mostly affect the dermatomes of T3 to L2, however, about 13% of the patients manifest
12 infections relating any of the three branches of the trigeminal nerve. Prodromal symptoms may
13 include malaise, neuropathic pain, headache and interrupted sleep. Herpes zoster causes
14 pruritic, confined, blisters which usually occur unilaterally in the distribution of either one or more
15 adjacent sensory nerves that comes along with neuropathic pain in the affected dermatome.
16 This case of herpes zoster is reported on a 32-years-old man who **was** managed based on the
17 symptoms he presented.

18 **1. INTRODUCTION**

19 Herpes zoster (HZ), also called shingles, is caused by varicella zoster virus and is usually
20 presented by painful, blistering dermatomal rash [1,2]. It is a neurodermotropic virus which
21 remains quiescent in the sensory ganglion and upon reactivation, it causes herpes zoster.
22 Reactivation of the virus may occur when the immune system of the host is incompetent [3].
23 Herpes zoster usually erupts in one or two adjacent dermatomes; 50-60% being thoracic, 10-
24 20% being cervical and trigeminal (10-20%) being most commonly affected areas. Meanwhile,
25 the lumbar (5-10%) and the sacral (5%) are far less commonly affected dermatomes. In patients

26 with competent immune system, the involvement of non-contiguous dermatomes is never
27 observed, however, intersecting of adjacent dermatomes can be observed in 20% of the cases.
28 The estimated lifetime risk of HZ in the general population is between 10-30%, with the risk
29 increasing sharply in people over 50 years of age.[4] The incidence of HZ in immunocompetent
30 individuals is 1.4 per 1000 years in people below 40 years, 4.2 per 1000 years in people up to
31 50 years and 10.5 per 1000 years for people above years old.[5] This case is being reported to
32 highlight the rarity of the incidence of HZ in individuals less than 40 years.

33 **2. PRESENTATION OF CASE**

34 A 32-year-old man reported to the outpatient department with fever, pain and blisters on the
35 right thoracic, right upper arm, dorsum and neck for 6 days. He developed the vesicular
36 eruptions 3 days after the fever, followed by itching, which subsided after 2 days. The patient
37 also complained of severe and continuous pain which radiates to the neck and head. There was
38 no history of physical or mental stress, trauma, ill health, radiotherapy. However, there was a
39 history of exposure to herpes zoster viral infection about ten years ago.

40 Examination showed ruptured vesicles which merged to form large, dried ulcers that differed in
41 sizes from 1-2cm in diameter on the thoracic, upper arm, neck and dorsum (figure 1). The ulcers
42 were irregular in shape and covered with pseudomembranous sloughs at the base. The ulcers
43 were tender on palpation. No vesicles were on the floor of the tongue or palate during intraoral
44 examination. Based on the clinical manifestation, a diagnosis of herpes zoster was given and
45 patient immediately started antiviral medications and pain relievers.

46 A prescription of acyclovir tablets 600mg every 6 hours a day for one week was given to patient
47 to control the active viral phase. Diclofenac tablets 50mg thrice daily for 3 days was also
48 prescribed as a pain reliever and to suppress the inflammation. Vitamin C supplement was
49 given to promote healing of ulcers and topical application of acyclovir cream (5%) was also
50 prescribed to help soothe the ulcers. The patient was reviewed every week for four weeks. By
51 the fourth week, all lesions were healed and no new ones formed (figure 2).

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54 **Figure 1: Dried ulcers on the dorsum, neck, thoracic and upper arm**



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56 **Figure 2: Ulcers completely healed by the fourth week**

57 **3. DISCUSSION**

58 Varicella zoster virus is an alpha herpes virus which causes primary infection called chicken pox
59 and then becomes dormant, usually in the dorsal root ganglia or ganglia of the cranial nerves.
60 On reactivation, it produces herpes zoster infection, known as shingles. [6] The virus enters its
61 host through the respiratory canals and begin to replicates at an undefined site (probably the
62 nasopharynx). The virus then infiltrates the reticuloendothelial system where it eventually finds
63 its way into the bloodstream.[7]

64 Herpes zoster infection (shingles) mostly occur in adults and starts with a prodromal of deep,
65 achy and burning pain. It is usually accompanied with slight to no fever and crops of vesicles

66 begin to appear within 2-4 days in a dermatomal pattern.[6] The lesions then begin to dry up 3-5
67 days after appearing. The duration of the disease is generally between seven and ten days,
68 although complete healing may last up to four weeks or more.[8]

69 Postherpetic neuralgia (PHN) is considered the most common complication associated with
70 herpes. This is a condition where the pain persist for a number of months and even sometimes
71 years after the rash has been resolved.[9,10] About 20% of elderly patients reports with PHN
72 after acute phase of herpes zoster infection. [11] The pain can be described as a brief recurring
73 and shooting, with a constant, typically deep pain and it is a major cause of morbidity. Other
74 possible complications of HZ may include myelitis, peripheral nerve palsies, encephalitis and
75 forms of contralateral hemiparesis. A rare complication of herpes zoster involving geniculate
76 ganglion is termed Ramsay Hunt syndrome. It is characterized by the development of Bell's
77 palsy, blisters of the auris externa as well as loss of taste in the two-third anterior of the
78 tongue.[8] There was no complication in this case report.

79 Acyclovir is the first medication of choice for the management of HZ. It is prescribed as tablets
80 acyclovir 800mg six hourly a day for 7-10 days. Famciclovir is a newer form of antiviral drug
81 developed to specifically tackle the acute of phase of herpes zoster infection. It is administered
82 in dosage of 500mg thrice daily for 7 days. Topical acyclovir cream (5%) treatment is applied as
83 topically four times daily for one week. Vitamin C is also given as 200mg thrice a day for 3
84 days.[12]

85 Oral corticosteroids have frequently been used for pain management in herpes zoster, however,
86 clinical trials have generated inconsistent outcomes for decreasing development of PHN. In a
87 study using the combination of prednisone and acyclovir indicated a significant decrease in pain
88 associated with herpes zoster. Narcotics use may be indicated in patients suffering severe pain.
89 Nerve block medications is also used as a pain management option in the standard medical
90 model. Anesthetic agents may also be injected locally in the affected nerves and this could
91 provide pain relief that lasts for 12-24 hours. [7]

92 **4. CONCLUSION**

93 It has been established that patients with HZ are contagious until the lesions have been healed.
94 Therefore, prompt diagnosis and management of the infection in the prodromal phase by using
95 the right antiviral medications should be the main aim of its management. Clinicians should
96 therefore be equipped with thorough knowledge about manifestation of the disease, its
97 treatment as well as its possible complications. Differential diagnosis is also very crucial to
98 ensure that the right treatment is provided.

99 **COMPETING INTEREST**

100 There is no competing interest.

101 **PATIENT CONSENT**

102 A written informed consent was obtained from the patient for publication of this case report and
103 accompanying his images.

104 **ETHICAL APPROVAL**

105 An approval was sought from Ghana Health Service through the Nabdam District Health
106 Directorate.

107 **REFERENCES**

- 108 1. JI C. Clinical practice: herpes zoster. *N Engl J Med*. 2013;369:255–63.
- 109 2. Dworkin R, Johnson R, Breuer J. Recommendations for the management of herpes
110 zoster. *Clin Infect Disinfect*. 2007;44.
- 111 3. Vineet D, Mithra R, Baskaran P. Oro-facial herpes zoster: a case report with a detailed
112 review of literature. *Oral Maxillofac Pathol J*. 2013;4:346–354.
- 113 4. Yawn B, Saddier P, Wollan P. A population-based study of the incidence and
114 complication rates of herpes zoster before zoster vaccine introduction. *Mayo Clin Proc*.
115 2007;82:1341–1349.
- 116 5. CDC. *Morbidity and Mortality Weekly Report:Prevention of Herpes Zoster*. Vol. 57. 2008.
- 117 6. Sook B, Martin S. *Ulcerative, vesicular and bullous lesions*. 11th ed. BC Decker; 2008.

- 118 46–49 p.
- 119 7. Mohan S, Prakash R, Verma S, Singh U, Agarwal N. Herpes zoster. *BMJ*. 2013;1.
- 120 8. Sampathkumar P, Drage L, Martin D. Herpes zoster (shingles) and post herpetic
121 neuralgia. *Mayo Clin Proc*. 2009;84:274–80.
- 122 9. Whitley R. Varicella-zoster virus infections. In: Fauci AS, Braunwald E, Isselbacher KJ, et
123 al. eds. *Harrison's principles of internal medicine*. 14th ed. New York: McGraw Hill; 1998.
124 1086–1089 p.
- 125 10. Blasberg B, Greenberg M. Orofacial pain. In: Greenberg MS, Glick M, eds. *Burket's oral
126 medicine*. 10th ed. Hamilton, Ontario: BC Decker; 2003. 330 p.
- 127 11. Mehta D, Thakkar B, Asrani M. Herpes zoster of orofacial region—a review. *Nat J Int Res
128 Med*. 2013;4:112–5.
- 129 12. Raj S, Verma P, Mahajan P, Puri A. Herpes Zoster Infection of the Face : A Case Report
130 with Review of Literature. 2017;2017–9.

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