CORRESPONDENCE

Herpes zoster with autonomic dysregulation presented as alteration in local microcirculation: a case report

Case report

A 68-year-old-man without any systemic disease presented at our dermatology outpatient clinic with the chief complaint of pain described as throbbing and burning along his right abdominal wall. This is followed by an eruption of clustered vesicles along the right T10–T12 dermatome. After admission and treatment with intravenous acyclovir for 3 days, the patient was discharged with further follow up. During a routine check up, 1 month after discharge, the patient showed full recovery of epidermal wound with subjective improvement of acute herpetic pain. However, mild postherpetic neuralgia was still noted 1 month after disease onset with residual postinflammatory hyperpigmentation. In addition, a conspicuous protruding mass measured at 7 cm × 7 cm over his right abdominal wall coinciding with the distribution of previous zosteriform skin eruption was noted during follow up physical examination (Figure 1). When palpated, there was absence of pain or apparent subcutaneous mass. The protruded abdominal mass was further accentuated during forceful expiration, a clinical sign suggesting focal muscle paralysis. Further survey with abdominal computed tomography showed normal finding without any apparent intra-abdominal lesions. Analysis with semiquantitative needle electromyography showed active denervation of the T10–T12 abdominal muscles on the zoster affected side, whereas normal nerve conduction was recorded on the contralateral T10–T12 dermatome. At this point, both sensory and motor nerve dysfunction had been confirmed. For further assessment of autonomic dysfunction, the method of cold recovery test was chosen. A circular area with a diameter of 2 cm was marked on both the affected dermatome and the contralateral normal dermatome. A baseline surface temperature and blood flow were recorded in these two areas before commencing the experiment. This was followed by simultaneous ice water packing (4°C) of both the control and experimental sides for 15 minutes. Cutaneous blood flow measured through Laser Doppler flowmetry (using the Periflux system 5000 model, Perimed AB, Sweden) was recorded at an increment of 1 minute for a total of 10 minutes following the removal of cold stress. The blood flow was determined by the product of the number of red blood cells moving in the measured volume (within the surface capillaries of the skin) and the mean velocity of these blood cells. This is expressed as perfusion unit (PU), which is arbitrarily determined by the Perimed analysis program of Periflux to allow direct comparison and documentation of blood flow within the experiment. The statistical analysis of autonomic nervous system function is described below. Briefly, the experiment was a repeated measures design from repeated observations of the zoster-affected side and the contralateral control side blood flow with skin surface temperature at each dynamic time point of 1 minute interval during cold stress recovery as described above. The Kolmogorov-Smirnov D tests for the lesional side (D = 0.16) and the control side (D = 0.14) were not significantly different assuming normal distribution. Therefore, the mixed linear model was used to estimate the lesional side and control side blood flow with the skin surface temperature at each time point during recovery process. Accordingly, at the baseline level, the surface temperature of the lesion and control were 32.9°C and 33.5°C, respectively (Table 1), whereas blood flow was 15.8 PU for the control and 7.8 PU for the herpes zoster affected side. The changes in temperature at each time interval were not significantly different between the lesional and the control sides (p > 0.05). On the other hand, the estimate of the lesional side intercept is −24.82, whereas the control side is −6.97, reflecting the initial changes (intercept) in blood flow for lesional side is lower than that for the contralateral control side (p = 0.0001). Secondly, the rate of blood flow increase (slope) is significantly higher in the lesional side as compared with the contralateral control side (p = 0.0115) (Figure 2).

In a subsequent follow up 3 months after discharge, the patient has recovered from the previous muscle paralysis and postherpetic neuralgia was barely perceptible. Unfortunately, we were unable to perform a second laser Doppler flowmetry examination to assess the recovery of autonomic dysfunction.

Figure 1 A conspicuous protruding mass over the right abdominal wall was presented during a 1-month follow up following zoster eruption. Mild residual hyperpigmentation was still visible.
Discussion
The exact pathogenesis of herpes zoster and postherpetic neuralgia is still under investigation. It is postulated that after a primary infection with Varicella zoster virus causing Varicella or more commonly known as “chicken pox,” the virus passes from the skin or mucosal surface into the contiguous endings of sensory nerves. These virus particles then transport centrifugally along the sensory nerves and establish a latent infection within sensory ganglions. During reactivation, Varicella zoster virus replicates within the ganglionic cells producing virions, which are subsequently transported along the axons producing zosteric lesions along the dermatome innervated by that sensory ganglion. Head and Campbell showed that in performed autopsies, Varicella zoster viruses were not only identified in dorsal root ganglion but also within sympathetic ganglion. Owing to the close proximity between the two types of ganglion, it is reasonable to hypothesize that a spread from dorsal root sensory ganglion through the white communicating rami to the sympathetic ganglion is possible.

Although the most frequent presentation of herpes zoster involves sensory nerves, motor, and autonomic symptomatology is also known to occur in this disease. There are at least 18 cases of isolated muscle paralysis of the abdominal wall related to herpes zoster reported in medical literature since 1895. Other examples of herpes zoster involving the S2–S4 dermatome with associated autonomic dysfunction presented as acute urinary retention and constipation have also been reported in rare cases. Therefore, it is reasonable to postulate that herpes zoster can also potentially affect other branches of the autonomic nervous system resulting in change in skin surface microcirculation as demonstrated in our case.

Temperature regulation involves a complex interaction between the preoptic anterior hypothalamus, autonomic nervous system, and the cutaneous arterioles. The body’s thermoregulatory reflexes, which facilitate body surface temperature regulation, are activated in the time of heat and cold stress. Various neurotransmitters and cotransmitters, such as vasoactive intestinal polypeptide, acetylcholine, and nitric oxide, work to regulate surface temperature through alteration of local microcirculation. Therefore, a disruption of autonomic innervation can potentially alter the regulation of microcirculation within the affected area. Following cold stress, the body attempts to restore normal surface temperature through vasodilatation to increase blood flow in the superficial dermis. This regulatory mechanism governed by autonomic nervous system is seen in effect through an immediate increase of blood flow following removal of cold stress in the control side. However, in the herpes zoster-affected dermatome, there was an initial decrease in blood flow as demonstrated by a significantly lower starting point or intercept value of −24.82 as compared with a more positive value of −6.97 on the control side (p = 0.0001). This difference in initial blood flow changes suggests a certain degree of autonomic dysregulation. Similar results were also seen in studies measuring blood flow recovery time of extremities in Type 2 diabetes patients with autonomic neuropathy. A longer vasodilatory response as compared with normal control from cold stress is seen regularly in severe diabetics and particularly in those with autonomic neuropathy. Interestingly, when comparing the average slope of differences in blood flow between the control and the lesional side, the lesional side showed a significantly higher slope value of 0.88 versus 0.52 of the control side (p = 0.0115). This can, therefore, be interpreted as the body’s attempt to compensate for the initial decrease in blood flow by increasing localized blood flow at a greater rate. This alteration in blood flow, however, resulted only in insignificant changes in surface temperature, which can be easily overlooked or masked by the excruciating pain associated with postherpetic neuralgia. However, the result of this article suggests that in a certain subset of patients, some form of autonomic dysregulation occurs at the herpes zoster-affected dermatome. Currently, no published literature has reported the incidence rate of autonomic dysregulation in herpes zoster-affected dermatome, but inferring from the incidence of herpes zoster associated acute urinary retention, the frequency is approximately 3.5%. Yet, this can be because of a combination of sensory, motor, and autonomic dysfunction. Therefore, further investigations are warranted to determine the frequency of autonomic dysregulation in herpes zoster-affected dermatome in other patients with herpes zoster and to clarify its incidence rate. In this report, we describe a rare case of combined sensory, motor, and autonomic nerve dysfunction along the affected dermatome following zoster infection.

Tommy Chi-H Chieh Chang, Yi-Ying Chin, Cheng-Che Eric Lan* Department of Dermatology, Kaohsiung Medical University, Chung-Ho Memorial Hospital, Kaohsiung Municipal Ta-Tung Hospital, Kaohsiung, Taiwan
* Corresponding author. Department of Dermatology, Kaohsiung Medical University, Chung-Ho Memorial Hospital, Kaohsiung Municipal Ta-Tung Hospital, 100 Shih-Chuan 1st Road, Kaohsiung, Taiwan E-mail address: laneric@gmail.com (C.-C. Lan)
References


Received: Mar 3, 2010
Revised: Apr 15, 2010
Accepted: Jun 29, 2010