Case Report
Posterior reversible encephalopathy syndrome following multiple wasp stings: a case report

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Abstract: Posterior reversible encephalopathy syndrome (PRES) is characterized by neuroimaging findings of symmetric changes of vasogenic edema predominantly in the posterior subcortical regions of the brain. The cause of PRES remains unclear. We describe a 66-year-old female patient who developed PRES with normal blood pressure on the first day after being attacked by a swarm of wasps. Over the next few days, she suffered from intravascular hemolysis, rhabdomyolysis, acute renal failure and hepatic dysfunction. Her outcome was favorable with prompt treatments including steroids, diuretics and plasma exchange. Mechanism of PRES in this patient may be systemic immune mediated reaction to the stings rather than arterial pressure exceeding the limits of cerebral vascular autoregulation.

Keywords: Posterior reversible encephalopathy syndrome, wasp, sting, pathophysiology

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiologic entity with headache, seizures, blindness and altered mental status [1]. A variety of underlying co-morbid conditions are associated with PRES, such as systemic lupus erythematosus, postchemotherapy, eclampsia, uremic encephalopathy and severe hypertension [1-3]. But those due to wasp stings are quite rare. We here present a case of PRES due to multiple wasp stings.

Case report

A 66-year-old woman presented with headache and pain at the sites of sting after being stung by a swarm of wasps. Twenty hours later she was agitated and then suffered from generalized seizures with loss of consciousness. The oxygen saturation was 70% on room air and intubation was conducted. She was afebrile, heart rate was 132 beats/min, blood pressure was 135/85 mmHg, and respiratory rate was 30 breaths/min. She was treated with bicarbonate infusion, steroids, antihistamines, and diuretics. An urgent CT showed bilateral hypodensities in the occipital and posterior parietal lobes. Following T2-weighted MRI (Figure 1A) and fluid attenuated inversion recovery (FLAIR) images showed hyperintense abnormalities of the bilateral parietooccipital area (Figure 1B). Diffusion weighted imaging (DWI) confirmed vasogenic edema (Figure 1C). At the same time, a scar was left on her skin (Figure 2).

Over the next few days after admission, the patient had one episode of dark urine. Repeated laboratory test showed a drastic drop in haemoglobin from 146 g/l to 55 g/l, platelet counts from 143,000/μL to 26,000/μL, albumin (ALB) from 44.6 g/l to 18.3 g/l. Plasma exchange was performed on hospital day 2 and lasted for 4 days. The highest level of blood BUN and serum creatinine was 23.47 mg/dl and 236 µmol/l separately. Laboratory findings also showed her impaired liver function: creatine phosphokinase (CPK) 4738 IU/l, lactate dehydrogenase (LDH) 4467 IU/l, aspartate alaninetransferase (AST) 621 IU/l, alanine aminotransferase (ALT) 624 IU/l. Liver enzymes and blood count normalized within 13 days of the incident, while renal function returned to normal on the sixteenth hospital day. Reaction of the skin lesions is 5 to 8 cm in diameter, with necrosis and scar formation.
Discussion

The wasp has venom dangerous to kidneys, blood, muscle and liver [4]. Human disease can result from toxic or allergic reactions after being stung. Acute renal failure with wasp bites is rare. It may be directly related to the insect venom or indirectly due to hemolysis and rhabdomyolysis [5].

This syndrome can occur in a broad spectrum of clinical entities such as severe hypertension, pre-eclampsia and hyperthermia, but only one case was reported about the relationship between PRES and wasp stings [7]. The patient also suffered from serious complications, such as renal failure, rhabdomyolysis and hemolysis. About one month after being stung, she had abrupt increases in blood pressure (160-190/88-104 mmHg) and an episode of unconsciousness with urinary incontinence. PRES was verified by a noncontrast CT scan. It seemed that sudden rise of blood pressure exceeded the autoregulatory capacity of brain and led to disruption of blood-brain barrier. However, renal failure or hemolysis may also be causative. For our patient, PRES manifested on T2-weighted MRI as hyperintense signal of the bilateral parieto-occipital. FLAIR sequences improve detection of injury. Facilitated DWI suggested vasogenic (increased ADC) rather than cytotoxic (reduced ADC) edema [2, 3]. PRES in this case occurred at the first day of onset with normal pressure, so it was not caused by hypertension. It was well known that vasoconstriction and endothelial dysfunction can be caused by systemic immune reaction after being stung [8]. So in my opinion, the underlying mechanism of PRES in this case may be mediated by systemic immune reaction. The antigen-anti-
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body complexes may play an important role in cerebral vasoconstriction and lead to brain edema. This case may support the view that pathophysiology of PRES is complex and not uniform [9].

In conclusion, our patient highlights the pathophysiological diversity of PRES. Immune factors may be important in PRES.

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Disclosure of conflict of interest

None.

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References


