# Localised herpes zoster infection and SIADH

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Localised herpes zoster infection ('shingles') in older patients is a common presentation to primary, and sometimes secondary, care physicians. However, symptoms of hyponatraemia, caused by the rare complication of 'syndrome of inappropriate antidiuretic hormone secretion' (SIADH), may be mistaken for constitutional symptoms of the infection itself. Such patients may require closer monitoring or hospitalisation.

**Case study – Mrs HS**

Mrs HS, aged 86 years, presented to an emergency department with a 2 day history of left sided abdominal pain and nausea. Examination was reported as normal and she was diagnosed with constipation and discharged on the same day. No blood tests were taken at this time.

Mrs HS returned to hospital 5 days later with additional symptoms of anorexia, fatigue and intermittent mild headache. Although she reported no constipation, diarrhoea or vomiting, her left sided abdominal discomfort had increased in severity and a rash had developed in the same area.

**On examination, Mrs HS was haemodynamically stable with a soft abdomen. Tenderness was elicited only over a prominent vesicular rash in the area of the ninth and tenth left thoracic dermatomes, consistent with localised herpes zoster infection ('shingles').**

Initial blood results revealed hyponatraemia and low potassium (*Table 1*). As there were no likely causative drugs such as thiazide diuretics or antidepressants to explain the hyponatraemia, and the patient denied excess fluid intake, syndrome of inappropriate antidiuretic hormone secretion (SIADH) was suspected. Syndrome of inappropriate antidiuretic hormone secretion is the continued antidiuretic hormone secretion despite plasma hypotonicity and a normal or expanded plasma volume. The diagnosis was subsequently confirmed in Mrs HS by low serum osmolality with inappropriately high urine osmolality and an elevated urinary sodium excretion (*Table 2*). (Normal serum sodium of 135 mmol/L had been recorded 23 months previously.) Urine osmolality need not exceed serum osmolality to diagnose SIADH, as is often perceived.

Fluid restriction of 1200 mL per day was commenced for hyponatraemia, with acyclovir 800 mg five times per day orally for herpes zoster. Further investigations were undertaken to exclude other possible causes of SIADH:

- infection (from any source, but commonly pulmonary and central nervous system [CNS])
- malignancy (primarily small cell lung, CNS and pancreas), and
- other CNS disorders such as stroke or haemorrhage.

Chest radiograph and contrast enhanced computer tomography brain scan were unremarkable as were C-reactive protein, full blood count and liver function tests. Although not causes of SIADH, metabolic causes of hyponatraemia such as Addison disease and hypothyroidism were also excluded with normal morning serum cortisol (351 mmol/L [normal range >140]) and thyroid function tests respectively.

Four days later, serum sodium had risen to 134 mmol/L and serum osmolality to 274 mmol/L. Mrs HS was allowed to drink freely and on discharge 3 days later, after discontinuation of a 1 week course of acyclovir, serum sodium remained stable at 135 mmol/L. The rash had started to dry and crust, with significantly less discomfort than on admission. Mrs HS's other presenting constitutional symptoms had resolved.

## Discussion

Herpes zoster is caused by reactivation of latent varicella zoster virus in dorsal root ganglia. In immunocompetent individuals, it commonly causes a localised cutaneous eruption limited to one or two dermatomes. Immunosuppression and increasing age are associated with an increased risk of re-activation, the exact mechanism of which is unknown.[1] Incidence rises sharply with age, 14.2 per 1000 person years in those aged over 75 years compared to 2.15 per 1000 overall.[2]

Well documented complications, predominantly neurological, include postherpetic neuralgia, encephalitis, arteritis and myelitis.[1,3] Of these postherpetic neuralgia is the most common, commonly defined as dermatomal pain persisting more than 30 days after cutaneous healing. Pain can persist for months or years, and the incidence and duration of the neuralgia are again correlated with increasing age.[4] Immunocompromise confers the additional risk of cutaneous and visceral zoster dissemination, with a mortality rate of 5–15%, even with intravenous acyclovir.[3]

There are several reports in the literature of SIADH associated with disseminated varicella-zoster virus infection in the immunocompromised.[5,6]

Syndrome of inappropriate antidiuretic hormone secretion, however, is a rarely reported complication of localised herpes zoster, the mechanism of which is unclear. There are only four other documented cases in which serum sodium has been recorded as low as 95 mmol/L.[7–10] Previous authors have speculated that neural connections between the hypothalamus and breast and chest wall dermatomes, which stimulate prolactin and oxytocin secretion during suckling, may be responsible.[7,8] Another, where the antidiuretic hormone secretion level alone was felt insufficient to account for the degree of hyponatraemia, suggested a role for other peptides such as brain natriuretic factor.[9] However, this seems unlikely in the absence of cerebral involvement.

## Conclusion

Most patients with localised herpes zoster are managed in the primary care setting where biochemical testing in a seemingly uncomplicated case is unlikely. As the association with SIADH is not widely recognised, many of the symptoms of hyponatraemia – as demonstrated in our case report – might be attributed to the constitutional symptoms of virus reactivation. We postulate that although the association has been rarely reported in the literature, this is likely to be an under representation of its true incidence.

## Summary of important points

- Dermatomal pain caused by herpes zoster may precede constitutional symptoms and rash by a number of days, thus mimicking other causes of abdominal and chest pain.
- SIADH is a rarely reported complication of herpes zoster and may be under recognised.
- Symptoms of hyponatraemia may be mistaken for constitutional symptoms of virus reactivation.
- Physicians should be aware of the association and have a low threshold for biochemical testing as a diagnosis of SIADH may alter management.

**Table 1. Initial blood results of Mrs HS**

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|---|---|
| Sodium | 122 mmol/L (NR 135–145 mmol/L) |
| Potassium | 3.4 mmol/L (NR 3.5–5.0 mmol/L) |
| Urea | 2.3 mmol/L (NR 3.0–8.0 mmol/L) |
| Creatinine | 46 mmol/L (NR 50–90 mmol/L) |

**Table 2. Results of investigations confirming SIADH**

| | |
|---|---|
| Serum osmolality | 248 mmol/kg (NR 275–295 mmol/kg) |
| Urine osmolality | 216 mmol/kg |
| Urinary sodium excretion | 81 mmol/L (NR 20–40 mmol/L) |

Conflict of interest: none declared.

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