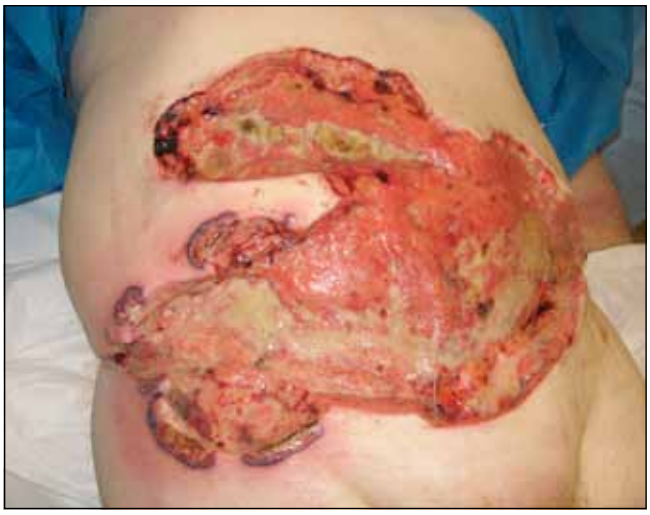

Fig 2. Extensive abdominal wall debridement with classical violaceous borders seen at the wound periphery

Intravenous antibiotics were stopped and high dose prednisolone was commenced in addition to the already prescribed somatostatin (Octreotide®). The patient was maintained on azathioprine (Imuran®) once the prednisolone had been tapered. His large abdominal defect was dressed with Activon tulle® honey dressings. He progressed well and was discharged. Follow up revealed satisfactory recovery of the wound.

DISCUSSION

The literature yields only one other case connecting PG with carcinoid tumour[1], while most reports correlate the occurrence of PG to trauma, typically surgery[2]. The delay in the recognition of this serious dermatological condition was associated with increased morbidity for our patient. PG is a serious and potentially fatal skin condition when correct treatment is not quickly commenced. Management is relatively simple once recognised with the use of corticosteroids and immunosuppressant. Surgery is not thought to be beneficial and in many circumstances can worsen the condition[3].

We recommend that in any significant skin condition, particularly post-operatively or in one not responding to treatment effectively, one must seek the early advice of a dermatologist and not be guided primarily by histology.

The authors have no conflict of interest.

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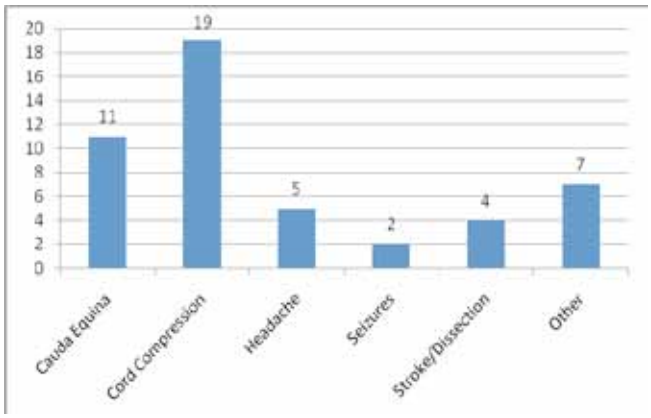
USE OF OUT OF HOURS MRI IN THE ROYAL VICTORIA HOSPITAL – A 6 MONTH RETROSPECTIVE REVIEW

Editor

Through the ongoing development of the Critical Care Centre, it is anticipated that the region's principal trauma receiving unit at the Royal Victoria Hospital will attain Level 1 Trauma Centre status. However an essential criterion for this is the provision of 24 hour access to MRI, as stipulated by the American College of Critical Care Medicine[1]. Out of hours MRI is currently provided as a time-limited, daily service on a consultant to consultant referral basis. Within the UK, it has been reported that only 32 out of 88 (36.3%) trauma units with MRI provide an out of hours service[2].

We undertook a 6 month retrospective review of all patients requiring out of hours MRI between November 2007 and May 2008. Records were assessed for referral information, imaging result and clinical outcome. 74 patients in total had out of hours MRI. Of these, 48 were regarded as emergency (scan performed <24 hours from referral).

Of the 48 emergency requests, the majority came from neurosurgery (n=27) and neurology (n=14), with orthopaedics (n=5), general medicine (n=1) and A&E (n=1) making up the remainder. Figure 1 illustrates the categories of clinical referral, with the majority for either suspected cauda equina syndrome or cord compression.



Out of hours MRI had the greatest impact in suspected cauda equina syndrome, as all scan positive patients (n=5) had surgery on the day of scanning, and made good neurological recovery, with only 1 having ongoing pain at 6 month follow-

up. Early surgery (<24hours) is felt to be of most benefit to those presenting with incomplete cauda equina syndrome[3]. However, it should be noted that suspected cauda equina syndrome contributed to 15% (11/74) of the total out of hours MRI caseload.

Of the 19 patients investigated for cord compression, 7 were confirmed on MRI. A further 2 patients were diagnosed with cord ischaemia. The remainder were either normal, had degenerative change or disc protrusion not causing compromise of the cord or nerve roots. 2 patients with confirmed cord compression were treated conservatively. Of those who had decompressive surgery, 2 were operated upon within 24 hours of their scan but neurological deficit persisted upon discharge.

It is anticipated that a modern, safe and comprehensive out of hours MRI service to Northern Ireland could be achieved with the 4 district general hospitals which have MRI capacity adopting an out of hours service similar to the current at the Royal Victoria Hospital, coupled with expansion of the Royal Victoria Hospital service to provide 24 hour access. Demand for out of hours MRI is anticipated to further increase with full implementation of NICE guidelines for stroke imaging and suspected metastatic cord compression.

The authors have no conflict of interest

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OLANZAPINE INDUCED HYPONATRAEMIA

Editor

We report a case, a 48 years old woman, presenting with life threatening severe hyponatraemia caused by the Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH) secondary to Olanzapine use. A Medline search revealed no publications of Olanzapine induced SIADH or hyponatraemia. However, online, there were three cases with hyponatraemia been reported at a Dutch pharmacovigilance centre[1].

A 48 years old Caucasian female, obese (BMI 32), smoker with medical history of mixed bipolar affective disorder, schizoid personality disorder and hypercholesterolaemia was admitted to the hospital in a postictal confusional state following an episode of generalised tonic clonic seizure at home with biting of the tongue and urinary incontinence. There was one day history of generalised muscle aches, anorexia, lethargy, irritability, confusion and unsteady gait prior to the episode. There was no history of polydipsia or polyuria. Shortly after admission, she had respiratory arrest for which she was intubated, started on mechanical ventilation and transferred to ICU.

She was on Olanzapine 20 mg daily for last two years. Her concomitant medications included Diazepam 5mg and Simvastatin 40 mg per day. She had not used any other medication known to cause SIADH during the previous two years. Laboratory investigations revealed hyponatraemia with sodium value of 114 mmol/l, serum osmolality 240 mos/kg, urinary sodium 49 mmol/l and urinary osmolality 220 mos/kg.

Diagnosis of SIADH was made. Olanzapine was incriminated as the causative agent since no other apparent cause of SIADH was found. With discontinuation of Olanzapine and treatment with hypertonic/ normal saline, her serum sodium levels normalised, her respiratory functions improved dramatically and soon, she was weaned off the ventilator, extubated and sent to general ward. In the ward, she continued to maintain normal sodium levels with the discontinuation of Olanzapine. Causality assessment using the Naranjo Nomogram revealed a probable association, with probability score of six.

DISCUSSION

Hyponatraemia (serum sodium concentration < 136 mEq/L) is a prevalent and potentially dangerous medical comorbidity in psychiatric patients[2]. Hyponatraemia is known to occur as a rare but clinically important adverse reaction to treatment with different psychotropic drugs[3]. In these patients, it is important to rule out psychogenic polydipsia, a clinical disorder characterised by polyuria and polydipsia, as it occurs in 6% to 20% of psychiatric patients and is more likely to be seen in schizophrenia[4].

In our patient, diagnosis of hyponatraemia secondary to SIADH was made as the biochemical blood and urine test results were consistent with SIADH. SIADH is suspected in any patient with hyponatraemia, hypoosmolality, and a urine osmolality >100 mOsm/kg. It causes hyponatraemia by preventing the excretion of ingested water[5].

Usually, rapid and complete recovery of drug-induced SIADH occurs when the offending agent is discontinued. In our patient also, the correction of hyponatraemia, combined with the discontinuation of her Olanzapine, resulted in resolution of hyponatraemia, without any further recurrence.

CONCLUSION

Clinicians should be aware that patients being treated with Olanzapine can develop hyponatraemia and it is important to check serum sodium levels when patients on Olanzapine develop symptoms suggestive of hyponatraemia.

The authors have no conflict of interest

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