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Case Report

A case of myxedema coma precipitated by herpes zoster: a rare manifestation of a common disease

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ABSTRACT

Myxedema coma is a disease state which is characterised by altered sensorium, hyponatremia, Bradycardia and hypotension. It is an emergency where prompt treatment is needed at the earliest, failure in identifying it is associated with fatal prognosis. The crisis is usually triggered by stressful events, the most common of which is infections like pneumonia and urinary tract infection. There has been sporadic case reports of myxedema coma associated with herpes zoster infection.

Keywords: Myxedema coma, Herpes zoster, Hypothyroidism

INTRODUCTION

Myxedema coma is a disease state which is characterised by altered sensorium, hyponatremia, bradycardia and hypotension. It mostly affects older people in winter months and is associated with a high mortality.[1] The crisis is usually triggered by stressful events, the most common of which is infections like pneumonia and urinary tract infection. It is an under recognised entity in tropical countries like India and most of the cases are from temperate regions. Myxedema coma is poorly recognised from the signs and symptoms and can only be identified with a high degree of suspicion by the treating clinician. It is an emergency where prompt treatment is needed at the earliest, failure in identifying it is associated with fatal prognosis.

Objective

The objective of this study was to consider myxedema coma as a differential diagnosis in a patient presenting with altered sensorium.

CASE REPORT

We presented an elusive case of a 80 years old gentleman who presented to us in the emergency department in a state of altered sensorium. There was no history of any co-morbidity.

At presentation, pulse was 52/minute (regular), blood pressure of 100/60 mm of Hg, saturation of 96% in ambient room air with a normal axillary temperature of 98°F (since we did not have the facility to check rectal temperature). There was no evidence of any focus of infection.

On further examination, a typical grouped painful herpetiform vesicles on an erythematous base was noted along the left T3-T5 dermatomes. There was non-pitting oedema around his orbits with loss of lateral 1/3rd of eyebrows (madarosis). Reflexes could not be elicited. Patient carried a set of reports done as a part of commercial package investigation the day. Table 1 shows biochemical

parameters before his presentation to the emergency which revealed.

Table 1: Showing all the biochemical parameters.

Parameters	Values
Hb	12.3 g/dl
Total count	8770
Platelet	1.50 lakh
Urea	70 g/dl
Creatinine	1.3 g/dl
Total bilirubin	0.4 g/dl
SGPT	87 mg/dl
Alkaline phosphate	117 mg/dl
TSH	>150 uIU/ml (0.7-4.2)
ESR	44 in 1st hour
Na	114 mg/dl
K	3.8 mg/dl
Albumin	4.6 g/dl
Globulin	4.9 g/dl
SGOT	68 mg/dl
Total T4	1.66 ug/dl (5.1-14.10)
Total T3	0.66 ng/ml (0.8-2.0)
FBS 110 mg/dl	PPBS 111 mg/dl

He was initially given 100 mg of IV hydrocortisone followed by 3 crushed tablets of 100 mcg of levothyroxine via the ryles tube as well as valacyclovir, pregabalin, levothyroxine daily and an emollient. No ventilatory support was required. By day 3, he started talking and was fully conscious. By day 7, his skin lesion improved and was discharged in a hemodynamically stable condition.

DISCUSSION

Myxedema coma is a form of severe hypothyroidism associated with a high mortality rate if prompt treatment measures are not initiated. Despite the term 'myxedema coma', it is a misnomer as the patients do not present with coma rather they come with altered sensorium.[2] In existing literature, the most important precipitating factors are infections. Among infections, pneumonia and urosepsis top the list.[3] There has been sporadic case reports of myxedema coma associated with herpes zoster infection.[4] In such cases, it was found to involve the dermatomal supply of trigeminal nerve. Herpes zoster remains a common disease in the elderly. Seldom does this disease go severe. In most of the countries including India, injectable T3 may not be available but T4 is easily available and so giving T4 via Ryle's tube is equally effective.[5] Circulatory collapse requiring pharmacological intervention or resuscitation and respiratory failure requiring mechanical ventilation are common complications.[6] Despite standard treatment of myxedema coma after detection, it is associated with poor outcomes. Several studies have shown that myxedema coma can present atypically, without the most common symptoms and clinical findings.[7-11]

CONCLUSION

Myxedema coma can be diagnosed only if there is a high degree of clinical suspicion by the treating physician and involves thorough history taking, physical examination and laboratory evaluation. As no definitive diagnostic tools are available to diagnose myxedema coma, it remains mainly a clinical diagnosis.

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