Coma Conundrum

A case of coma blisters in non-dependent areas

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Introduction

Coma blisters are a well-described phenomenon, consisting of bullous skin lesions in the setting of loss of consciousness. (1)

Classically occurring in pressure dependent areas, (2) they commonly arise in patients with coma due to barbiturate overdose, develop within 48-72 hours of the onset of coma, and are generally self-limited. (1)

Case Description

We present the case of a 52-year-old lady, who was brought by ambulance to her local hospital.

On arrival GCS was 6 and she was intubated and ventilated, however the reason for her reduced level of consciousness unclear.

Past medical history was significant for epilepsy. Anaesthetic colleagues on arrival noted the presence of a number of tense, 0.5-1cm, serous fluid-filled blisters, with underlying erythematous plaques on the left dorsal hand, forearms, dorsal feet, and right ankle.

Of note, there was no peripheral oedema. She was transferred to ITU.

Investigations and histology

At time of dermatology consultation, the GCS remained at 6, despite weaning sedation & ventilation.

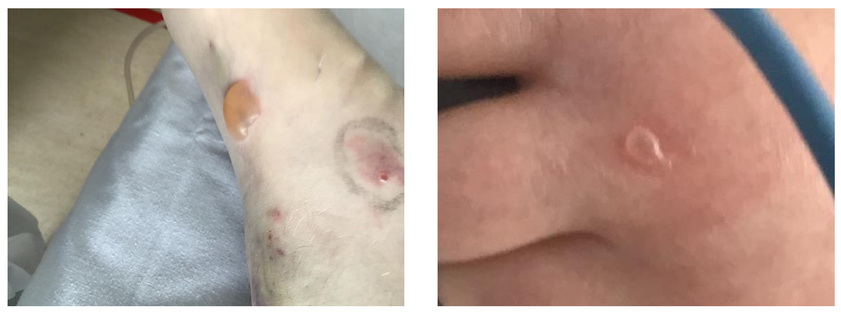
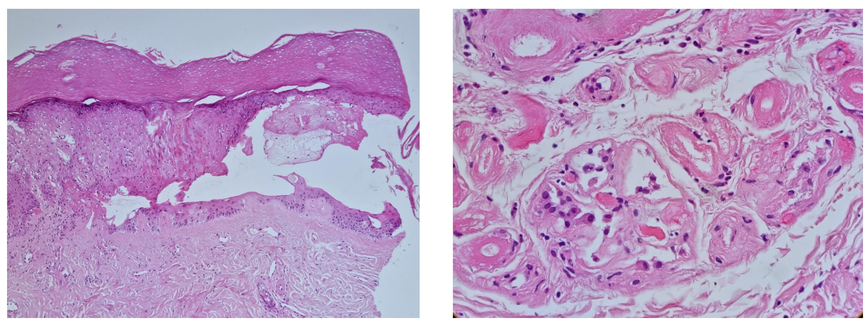


Fig 1: Bullous lesions on the dorsal foot (left)
Fig 2: Bullous lesions on the dorsal hand (right), noted on arrival in the emergency department

The lesions, as pictured, appeared consistent with coma blisters but given presence on non-pressure dependent areas and the fact that the reason for loss of consciousness remained unclear, a skin biopsy was taken. Barbiturate levels were requested in the context of epilepsy treated with phenobarbital.



Histological images from biopsy of dorsal foot.

Fig 3 (left): Focal epidermal detachment with fibrinoid necrosis and partial re-epithelialization. Mild accompanying inflammatory component.
Fig 4 (right): Detail of dermal tissue bearing focal fibrinoid necrosis of eccrine glands.

Skin biopsy showed sub epidermal blistering with partial re-epithelialization and focal keratinocytic necrosis. In the blister cavity, focal fibrinoid degeneration and mild neutrophilic infiltrate was observed. Underlying dermis showed focal neutrophilic inflammation and sweat gland ischemic necrosis – consistent with coma blisters.

Phenobarbital toxicity was confirmed on biochemical analysis.

Outcome and Discussion

The lesions self-resolved, and the patient gradually regained consciousness and was well enough for discharge home, with plan from neurology team to wean off phenobarbital.

While coma blisters are well described, their presence in non-pressure bearing areas is less common, and coma blisters were not considered as a possible cause. (4) The patient had initially been admitted to a local hospital, then transferred to a tertiary hospital, as cause for reduced level of consciousness was unknown and consult services are more accessible

Blisters had been noted on the initial admission, and dermatology had been informed allowing for prompt consultation following transfer

The presence of these blisters provided a clue to the aetiology of the patient's presentation before barbiturate toxicity was confirmed on biochemical analysis.

Following dermatology consultation, a uniting cause for skin lesions and loss of consciousness was identified, and ultimately led to a diagnosis, allowing for appropriate treatment of the patient and for measures to be taken to prevent further episodes of phenobarbital toxicity.

Literature cited

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Acknowledgments

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