

Ogilvie's syndrome, beta-blockers and burns

To the Editor: A 25-year-old HIV-negative man was admitted to the Burns Unit at Pelonomi Hospital, Bloemfontein. He had been involved in a domestic fire accident and had sustained 25% total body surface area burns, mostly full-thickness, involving the lower limbs. He had no inhalation injury and was otherwise in good health. On admission he was moderately oliguric.

The patient was adequately resuscitated and taken to theatre for burn wound excision and autografting. Postoperatively he failed to thrive, going into a hyper-metabolic state with progressive weight loss and persistent tachycardia despite a normal temperature and repeatedly negative inflammatory markers (pro-calcitonin (PCT), C-reactive protein (CRP) and white cell count).

Oral propranolol was introduced at a dose of 20 mg 8-hourly and was continued until the heart rate fell by 25% (from 140 to 110/min). After 3 days on propranolol there was considerable improvement in the patient's general condition. He became more alert and mobile whereas previously he had been bedridden, but at the same time he developed a colonic pseudo-obstruction.

Abdominal radiographs showed massive colonic distension with no signs of a volvulus or other mechanical obstruction. Infection markers (PCT, CRP) were repeatedly negative. His general state did not deteriorate, and the Ogilvie's syndrome was managed non-operatively (bowel decompression by rec-

tal tube, fluid and electrolyte correction, and withdrawal of propranolol).

The patient developed sepsis caused by *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, but these organisms were sensitive to piperacin and tazocin. He required repeated skin grafts because of loss due to sepsis but remained in reasonable condition, and the Ogilvie's syndrome did not compromise the burn treatment to any great extent. He was discharged 74 days after the burn.

Beta-blockers have become the accepted way to modulate burn-associated hypermetabolism.¹⁻³ The benefits of this therapy need to be weighed against the side-effects. It is hoped that this report will stimulate vigilance during modulation therapy for burn-associated hypermetabolism.

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REFERENCES

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