Nebulised frusemide for the symptomatic treatment of end-stage congestive heart failure
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TO THE EDITOR: We report the use of nebulised frusemide for the symptomatic treatment of end-stage congestive heart failure (CHF). An 84-year-old man with New York Heart Association class IV CHF was referred to the Community Heart Failure Team at St Vincent's Hospital, Sydney, for ongoing management after a hospital admission for acute pulmonary oedema. His medical history included aortic stenosis, pulmonary hypertension, type 2 diabetes, chronic renal failure, atrial fibrillation, hypertension, chronic obstructive pulmonary disease (COPD) and hypercholesterolaemia.

The patient's medications were home oxygen via a concentrator at 2–4 L/minute; digoxin 5.625 μg three times a week; warfarin 5 mg daily; glyceryl trinitrate 25 mg daily (delivered via a patch); simvastatin 40 mg nightly; spironolactone 12.5 mg daily; frusemide 80 mg orally twice daily (flexible regimen); insulin/isophane (Protaphane; Novo Nordisk) variable dose twice daily; fluticasone 250 μg/salmeterol 50 μg (Seretide; GlaxoSmithKline) one dose twice daily; and omeprazole 20 mg daily. Previous trials of a β-blocker and angiotensin-converting enzyme inhibitor were not tolerated.

Two days after the patient was discharged, a home visit by the clinical nurse consultant (CNC) found him with grossly oedematous legs, jugular venous pressure (JVP) elevated above his ears, and crepitations from the bases to the upper mid zones of his lungs. On Day 1 and 2 of CNC care at home, the patient received intravenous bolus doses of frusemide 80 mg, resulting in good diuresis. On Day 3, the CNC was unable to gain intravenous access and, after consulting the Community Heart Failure Team cardiologist and pharmacist, administered frusemide 80 mg via a nebuliser.

The patient reported immediate improvement. Oxygen saturation increased from 88% to 97% on room air and his chest was clearer on auscultation. Increased diuresis occurred, with weight loss of 1 kg. Because the patient's JVP and leg oedema were unchanged, the dose was repeated daily for 5 days until respite admission (for social reasons and intravenous frusemide administration). Nebulised frusemide had provided symptomatic relief from dyspnoea for about 5 hours with no adverse effects for the patient, but did not provide sufficient diuresis to reduce his fluid overload symptoms. Ultimately, a central catheter (“long line”) was inserted to enable the CNC to administer frusemide intravenously at the patient's home.

CHF-associated dyspnoea causes significant morbidity and distress for patients and carers. Nebulised frusemide has been used for relief of dyspnoea associated with asthma, COPD and malignancy. Its precise mechanism of action is unknown, but is believed to be through local lung rather than renal effects. Our searches of MEDLINE, EMBASE, CINAHL and the internet found no reports of the use of nebulised frusemide for dyspnoea resulting from pulmonary oedema or CHF. Patients with CHF receiving palliative care have limited options for diuresis when oral administration is ineffective and intravenous access is unavailable. The use of nebulised frusemide could have potential in this setting, but requires further research.

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