

CASE REPORT

Acute treatment of uncompensated heart failure with 10% hypertonic saline and its subsequent effect on respiratory patterns

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INTRODUCTION

Uncompensated heart failure (UHF) is a leading cause of hospitalization. The available drugs to treat acute UHF include inotropes, diuretics and vasodilators. In recent years, hypertonic saline (HTS) has emerged as a new potential therapy for refractory heart failure, usually in association with high doses of diuretics. However, there have been no reports describing the use of HTS as a first-line treatment for UHF. Here, we report a case in which a 10% NaCl solution was used as the primary therapy for a patient with UHF. Almost immediately, hypertonic saline was able to restore the patient's hemodynamic profile and to eliminate Cheyne-Stokes respiration (CSR), which is associated with a low cardiac output state.

CASE DESCRIPTION

A 90-year-old man was admitted to the intensive care unit (ICU) because of dyspnea and lethargy. The patient had been diagnosed with prostatic cancer ten years before but had not received chemotherapy in the last two years. He also had chronic congestive heart failure with atrial fibrillation, probably related to arterial hypertension. His current prescriptions included furosemide (40 mg once a day) and captopril (25 mg twice daily). He had been institutionalized in a hospice for the previous six months and had symptoms of dyspnea after performing light to moderate activities.

Upon admission, the patient was somnolent (hypoactive delirium) and exhibited no focal neurologic signs. He complained of shortness of breath and lower back pain. His extremities were cold and had delayed capillary refill (about 4 seconds). The patient's blood pressure was 74/46 mmHg, and his heart rate was 120 beats per minute (atrial fibrillation). His central venous pressure was clinically estimated to be 17 cm of water. His breathing pattern was periodic and presented as apnea interspersed with faster and deeper breaths, which we determined to be Cheyne-Stokes respiration (CSR). He received supplementary oxygen through a nasal catheter at two liters per minute

with a peripheral arterial O₂ saturation of 99%. Rales were detected throughout the length of the patient's hemithoraces.

Laboratory exams showed a moderate level of metabolic acidosis, with a pH of 7.20, a low bicarbonate level of 17.8 mEq/L and a standard base excess of -10 mmol/L. His carbon dioxide level was slightly elevated at 46.3 mmHg, his lactate level was 2 mmol/L, and there were no electrolyte disorders detected. Initial troponin and creatine kinase-MB were both within normal ranges. Echocardiography was not available at the time.

A diagnosis of cardiogenic shock was established. After meeting with the patient's family, it was decided to treat the patient without invasive mechanical ventilation, dialysis, central venous puncture or vasoactive amines. The patient also refused noninvasive mechanical ventilation. Because the patient had already received 1L of 0.9% NaCl before ICU admission, only a small fluid challenge of 250 mL of lactated Ringer's solution was administered. However, no improvement in the patients parameters was observed. A low dose of dobutamine was administered, but the patient developed several episodes of non-sustained ventricular tachycardia and hypotension, so dobutamine administration was stopped. Additionally, nitrates were not administered due to the patient's low arterial blood pressure. Nesiritide was not available at the time. Therefore, we decided to try hypertonic saline infusion as a final, non-invasive approach to improve myocardial contractility. We infused 100 mL of 10% NaCl over a period of 10 minutes.

Less than three minutes after the infusion, the patient's breathing pattern improved, with the progressive reduction in the time of peak ventilation, cycle length, apnea length and ventilation length. Finally, the patient's breathing pattern returned to normal (figure 1). The patient's blood pressure increased to 110/70 mmHg, and his heart rate decreased to 106 beats per minute. His capillary refill time also improved. One hour later, intravenous infusion of a low dose of nitroprusside was performed, which was well tolerated by the patient. The patient was discharged from the ICU after being treated with oral vasodilators for an additional few days.

DISCUSSION

Recently, the use of hypertonic saline was proposed as a treatment for refractory heart failure associated with

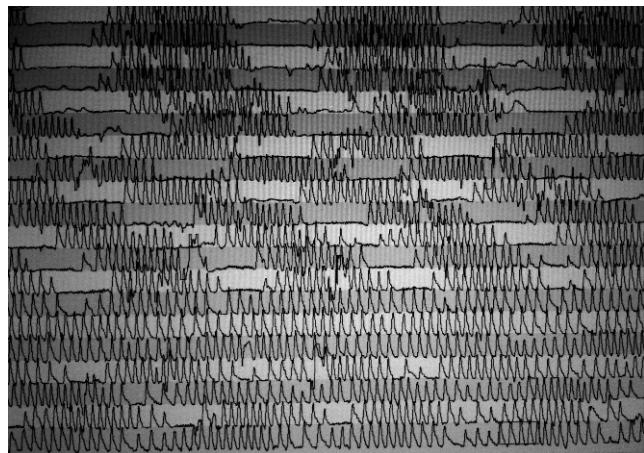


Figure 1

high-dose diuretic therapy, with promising results.¹ Most of the data available on the use of HTS are from chronic ambulatory patients, with only one report from an acute-care setting. Additionally, these data were generally from patients with cardiogenic shock resulting from right ventricular infarction.² Another report also evaluated the use of hypertonic saline as an adjunct therapy in uncompensated heart failure patients and indicated that hypertonic saline had positive effects on renal function.³ In experimental models, hypertonic saline improved myocardial contractility,⁴ but its immediate impact on the hemodynamic profile during acute UHF has not been studied or reported to date. In the present case study, hypertonic saline was the only available non-invasive option for the treatment of the patient. Its use improved the patient's blood pressure so that vasodilators could effectively be used. No side effects were observed after the infusion. To our knowledge, this is the first report of the use of HTS as the primary therapy in the management of UHF.

HTS infusion also restored the patient's normal breathing pattern. Periodic pattern breathing and CSR are both associated with UHF and low cardiac output states.⁵ In

ambulatory patients, CSR is associated with increased mortality, impeded exercise capacity and arrhythmias.⁵ The pathophysiology of CSR is complex and appears to involve circulation delay, increased chemoreceptor sensitivity and hyperventilation or hypocapnia.⁵

We hypothesize that HTS administration exerted a beneficial effect on the patient's myocardial contractility and improved his cardiac output, which reduced his periodic breathing pattern and improved his blood pressure. Interestingly, this improvement occurred in under three minutes (figure 1). Unfortunately, due to the quick action of HTS, we were unable to measure cardiac output before and after infusion. However, the possibility that the hypertonic solution exerted a direct effect on breathing control mechanisms (especially on chemoreceptors) cannot be excluded.

In conclusion, hypertonic saline was able to restore hemodynamic parameters and breathing patterns almost immediately after administration to a patient suffering from uncompensated heart failure. Therefore, hypertonic saline may be an attractive therapeutic option for the initial management of UHF. These data suggest that randomized controlled trials to evaluate the role of HTS early in the treatment of UHF are clearly warranted.

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