Published in final edited form as:

Am J Kidney Dis. 2008 July ; 52(1): 140–143.

Challenges of Treating a 466 Kilogram Man with Acute Kidney Injury

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Abstract

Caring for super obese patients (body mass index >50) presents a number of complex and unique clinical challenges, particularly when acute kidney injury is present. We describe our experience treating the heaviest individual with acute kidney injury requiring renal replacement therapy reported to date. A 24 year old black man was admitted to our hospital with fevers, vomiting, progressive weakness, shortness of breath, and hemoptysis. Admission weight was 1024 lbs (466 kg), height was 6 ft 4 in (1.9 m), and body mass index was 125. During hospitalization the patient suffered oligoanuric acute kidney injury and required initiation of continuous and, subsequently, intermittent renal replacement therapy. This clinical scenario, which identifies many of the kidney and non-kidney related challenges surrounding the care of super obese patients with acute kidney injury, may be a harbinger of what awaits the nephrology community in the obesity pandemic era.

Keywords

super obesity; acute kidney injury; dialysis; renal replacement therapy; drug dosing

INTRODUCTION

Despite intense interest by the nephrology community in the clinical implications of obesity, relatively little attention has been given to individuals with clinically severe obesity, such as the “super” obese (body mass index (weight (kg)/height (m)²) >50), the fastest growing obesity subgroup (1). Caring for these patients in a hospital setting presents a number of complex and unique challenges, especially when management of kidney injury is involved. This report describes our experience in treating the heaviest individual with acute kidney injury requiring renal replacement therapy ever documented in the medical literature.

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RESEARCH SUPPORT

Friedman: NIH; Dialysis Clinics, Inc.; no financial conflict of interest

Decker: no outside support; no financial conflict of interest

Seele: no financial conflict of interest

Hellman: no outside support; no financial conflict of interest

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CASE REPORT

A 24 year black man was brought to our emergency ward with fevers, vomiting, progressive weakness, shortness of breath, and hemoptysis. The patient weighed 343 lb (156 kg) and 600 lb (273 kg) at ages fourteen and twenty one, respectively. On admission, physical exam revealed a weight of 1024 lbs (466 kg), height of 6 ft 4 in (1.9 m), and body mass index of 125. He was febrile to 102.7°F and tachypneic at 28 breaths/minute. Admission blood urea nitrogen and serum creatinine were 10 (3.6 mmol/L) and 1.3 mg/dl (99 µmol/L), respectively (serum creatinine was 1.1 mg/dl (84 µmol/L) three years prior), and toxicology screen was positive for cocaine, opiates and tetrahydrocannabinol. Initial urine and blood cultures were negative.

He immediately developed prolonged hypotension and respiratory failure. Presumptive diagnoses of cocaine-induced lung injury and sepsis were established. A nasotracheal intubation was performed. Oligoanuric acute kidney injury promptly followed, requiring initiation of renal replacement therapy. Urinalysis revealed a specific gravity of 1.015, pH of 5.0, trace protein, 8 red blood cells, 54 white blood cells with positive leukocyte esterase, and 4 squamous epithelial cells. Admission to the intensive care unit was delayed for two days until a room large enough to fit the patient was identified. A standard 19.5 cm temporary dialysis catheter was placed in the right internal jugular vein by the bedside under ultrasound guidance. Radiological assessment of the dialysis catheter position post-placement as well as the urinary system were not performed because of limited interpretability due to the patient's body habitus. An industrial grade lift was used during the hospitalization to help position and move the patient. Enteral support (Peptamen AF, Nestlé) at 50 cc/hr was initiated.

Assessment of volume status and dialysis and medication dosing were based entirely on empiric trial and error. Continuous venovenous hemofiltration was initiated using 6 L of replacement fluid hourly until room plumbing could be modified for intermittent hemodialysis. Dialysis was then performed for four to six hours five to six days/week using a high flux membrane (Polyflux 210 (2.1 m²), Gambro), which adequately controlled small solute and electrolyte levels. Urea reduction ratio for a typical session was 0.27 and single-pooled Kt/V was 0.31. Ultrafiltration of at least 120 L was successfully performed over the ensuing weeks. Pharmacokinetic studies based on an infusion of vancomycin 4000mg were performed. The corresponding peak level, considered to be the serum concentration at time zero (C_{p0}), was 30.5 mg/dL. This resulted in a calculated volume of distribution (V_d) of 131.1 L and a volume of distribution per total body weight of 0.29 L/kg.

In the setting of recurrent sepsis, a non-operable, enlarging necrotic paraspinal ulcer and osteomyelitis, a decision was made with the family to withdraw dialysis support. The patient died two weeks later, nearly three months after being admitted to the hospital.

DISCUSSION

Though the body mass of the individual highlighted in this case study is clearly exceptional, nephrologists should not be surprised if they begin to encounter similar cases with more frequency, for several reasons. First, according to data obtained from a Centers for Disease Control (CDC) annual telephone survey, the prevalence of super obesity is increasing at a much faster rate among American adults than is that of moderate obesity (1) (see Figure 1). Specifically, the prevalence of super obesity increased by over 900% in the last two decades (i.e. 0.42% of the adult population in 2005) *versus* less than 200% for individuals with moderate obesity. Second, super obesity elevates the likelihood of acute kidney injury. The risk for end-stage renal disease rises with body mass index (2), and chronic kidney disease is itself an established risk factor for acute kidney injury. Furthermore, in the surgical literature super obesity is associated with higher rates of acute kidney injury as well as other serious

complications (3–5). Third, the super obese spend more time in-hospital compared with less obese individuals, increasing exposure to risk factors associated with acute renal failure (6).

The clinician and support team face many challenges in caring for the super obese. For example, the physical examination, normally a vital diagnostic tool, is much less helpful in these patients, particularly with regards to the assessment of volume status. In addition, tests such as the renal ultrasound that are normally a routine part of the clinical evaluation are simply not useful.

Management also becomes much more difficult, and the challenges begin with the great effort, expense, and logistic support required in transporting these patients to the hospital. As recently documented (7), special bariatric ambulances that can carry up to 1,000 lbs and pull a stretcher up a ramp via a winch are now required for some obese patients. In addition, limitations related to hospital infrastructure and equipment may delay or hinder treatment. Some of these can be overcome by avoiding certain rooms and hallways that are too small or narrow to transport such patients, by employing bariatric beds (though most actually support no more than 600 to 1000 lbs) and special lift systems, and by ensuring that the rooms reserved for such large patients contain plumbing compatible with hemodialysis.

Appropriate medication dosing is particularly important in persons with altered baseline kidney function. Unfortunately, few anti-microbial or other drug dosing guidelines exist for moderately obese individuals, much less for those with super obesity (8). Our patient's vancomycin volume of distribution (131.1 L) was somewhat lower than the urea volume of distribution as calculated using the conventional total body water ($0.6 * \text{weight(kg)}$), Watson (9), or Hume-Meyer formulas (10), though it was consistent with previous findings in obese subjects (11–13). This discrepancy highlights the limitations of available formulas and suggests that drug dosing strategies may not be substantially different between the obese and super obese.

In addition, the use of hemodialysis as a therapeutic modality in super obese individuals is an unexplored area, and the optimal strategies and goals are unclear. Our treatment plan was purely empiric in nature and involved 1) keeping small solute levels, reflected by the blood urea nitrogen, as low as possible, and 2) removing fluid without inducing hemodynamic instability. Our initial CVVH dose (approximately 13 ml/kg/hr) was arrived at empirically, and though much lower than the currently recommended dose of 35 ml/kg/hr (14), appeared to provide adequate solute clearance. Adhering to the accepted CVVH dose, which would mean 16 L/hr replacement fluid, would have been unfeasible. As noted in Figure 2, small solute levels were reasonably controlled with our strategy. Given the frequency and length of our patient's dialysis sessions, it is fair to conclude that despite the large amount of small solute generated, intermittent hemodialysis *is* able to effectively control solute levels even in extreme obesity. Had we not been successful, we could have employed other strategies, such as using two dialysis membranes in series or more continuous forms of dialysis. Though we believe that the patient's proximate causes for withdrawal of care were related to problems other than underdialysis, this is impossible to verify.

Finally, the unique psychosocial aspects of caring for super obese patients with acute kidney injury must be acknowledged. We were aware that if the acute kidney injury was not to improve, outpatient dialysis would not be a feasible option for any individual of this size, and we communicated this to the medical team and family from the outset. We believe that this helped facilitate the decision by the health care team and family to accept the withdrawal of renal replacement therapy when the time came.

As daunting as it seems, our clinical case, which identifies many of the renal and non-renal challenges surrounding the care of super obese patients with acute kidney injury, may be a harbinger of what awaits the nephrology community in the obesity pandemic era.

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Figure 1.

Percentage increase in body mass index (BMI) categories since 1986. Reprinted with permission (1).

Figure 2.

Changes in serum BUN and creatinine over first month. * signifies initiation of continuous renal replacement therapy. † signifies initiation of intermittent renal replacement therapy.