Chapter 21
Endocrine and Metabolic Management in the Cardiothoracic ICU

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ABSTRACT

In this chapter, we review several important endocrine disorders frequently encountered in patients undergoing cardiothoracic surgery. Hyperglycemia, common in patients with and without diabetes mellitus (DM) in the perioperative period, has been linked to poor outcomes. Use of an intravenous insulin infusion early in the postoperative course, followed by transition to subcutaneous insulin, with maintenance of moderate glycemic targets (100-180 mg/dL) is currently the standard of care. Oral intake should be encouraged in the postoperative period, but if not possible, nutrition support with enteral nutrition should be considered. Critical illness related corticosteroid insufficiency (CIRCI) should be suspected in critically ill patients with refractory hypotension requiring vasopressors, especially in the setting of septic shock. Although diagnositic criteria are controversial, if suspected, empiric treatment with corticosteroids should be initiated. Nonthyroidal illness syndrome (NTIS) is common in critically ill patients and thyroid function tests should be interpreted with caution in this population.

INTRODUCTION

Endocrine and metabolic problems are among the many complex medical issues that face the cardiothoracic surgical patient. While at first glance these issues may appear minor, compared to obvious imperatives such as cardiac and pulmonary function, a deeper understanding of endocrine/metabolic pathophysiology will enhance overall care and clinical outcomes. The first objective of this chapter is to review the metabolic effects of stress endured by the CTICU patient, with an understanding of the various stages

DOI: 10.4018/978-1-4666-8603-8.ch021

of critical illness that may be traversed depending on the severity of illness. Subsequently, the chapter will explore four key topics: 1) tight glycemic control, 2) nutrition support, 3) adrenal insufficiency, and 4) abnormal thyroid function. For each of these important areas, the relevant literature will be reviewed and optimal management strategies presented.

BACKGROUND

While significant and recent advances in cardiothoracic surgical care have optimized patient outcomes, these patients sustain varying degrees of critical illness in the post-operative period. From a metabolic perspective, patients can be understood in terms of four sequential stages of critical illness, each with a unique hormonal and metabolic milieu: acute critical illness (ACI), prolonged acute critical illness (PACI), chronic critical illness (CCI), and recovery from critical illness (RCI) (Hollander & Mechanick, 2006; Schulman & Mechanick, 2012).

ACI follows a stressor (e.g., surgery, infection, or trauma) that triggers the "stress response," characterized by enhanced secretion of "stress hormones" (e.g., cortisol, glucagon, and growth hormone), catecholamines, and cytokines (tumor necrosis factor-α [TNF-α], interleukin-1 [IL-1], and interleukin-6 [IL-6]) (Singer, De Santis, & Vitale, 2004). Increased levels of cytokines shift reverse-phase reactants (e.g., albumin, transferrin, and cortisol-binding globulin) to acute-phase reactants (e.g., C-reactive protein and immunoglobulins). This hormonal milieu diverts substrates from anabolic to catabolic pathways, stimulating gluconeogenesis, lipolysis, and proteolysis of skeletal muscle, while inhibiting peripheral glucose uptake, to ensure an adequate supply of substrates (Chang & Bistrian, 1998; Marques, & Langouche, 2012). These metabolic changes create a state of insulin resistance, frequently leading to hyperglycemia in critically ill patients (Mechanick, 2006).

PACI, beginning at around day 3 of critical illness, is characterized by a persistent stress response, with maintenance of inflammation, catabolism, and insulin resistance. While ACI is considered an adaptive response, conferred by evolutionary mechanisms, the persistence of critically ill patients in the intensive care unit (ICU), repeatedly avoiding death by artificial means, is largely due to technological innovations, which have no evolutionary precedent. Persistent immune-neuroendocrine (INA) output results in excessive tissue breakdown, hyperglycemia, and hypoalbuminemia.

CCI, beginning with tracheotomy at around day 10-14 of critical illness, represents a unique maladaptive physiological state characterized by prolonged mechanical ventilation, kwashiorkor-like malnutrition, stress hyperglycemia, neuroendocrine dysfunction, impaired wound healing, immune deficiency, metabolic bone disease, critical illness myopathy and polyneuropathy, neurocognitive dysfunction, and excessive symptom burden for patients (Hollander & Mechanick, 2006; Nelson, Meier, & Litke, 2004).

RCI is defined by successful liberation from mechanical ventilation and promotes amelioration of inflammation and a return from catabolism to anabolism. Survivors frequently suffer from persistent organ dysfunction and impaired quality of life. Patients in the cardiothoracic ICU ideally recover from critical illness within a few days postoperatively, directly following ACI. However, some patients follow a more complicated course with prolonged mechanical ventilation.