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REVIEW ARTICLE

A RARE CASE OF POST SURGICAL STRESS INDUCED APPENDICITIS

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ABSTRACT

Acute appendicitis is common surgical disease most common in children and adolescent.Althoughits dominant cause is luminal obstruction and other less common cause includes emotional stress, blunt abdominal trauma. Post surgical acute appendicitis is rare event and cause effect relationship between them is not well established yet.

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INTRODUCTION

The stress response to surgery, critical illness, trauma, and burns encompasses derangements of metabolic and physiological processes which induce perturbations in the inflammatory, acute phase, hormonal, and genomic responses. Hypermetabolism and hypercatabolism result, leading to muscle wasting, impaired immune function and wound healing, organ failure, and death. The surgery-induced stress response is largely similar to that triggered by traumatic injuries; the duration of the stress response, however, varies according to the severity of injury (surgical or traumatic). This spectrum of injuries and insults ranges from small lacerations to severe insults such as large poly-traumatic and burn injuries. Following surgical or accidental trauma, the nervous system activates the stress response by sending impulses from the injured site to the hypothalamus. The hypothalamus either removes its inhibitory tone on the pituitary or releases hormones which stimulate the production and/or release of pituitary hormones. Pituitary hormones act on their respective target organ causing the release of hormones such as the stress hormone, cortisol. Elevations of cortisol, glucagon, catecholamines, and a host of inflammatory cytokines, also exacerbate the stress response to surgery. Afferent nerve signals from the injured site and proinflammatory cytokines have the net effect of increasing the secretion of hormones from the pituitary gland. Increased secretion of the anterior pituitary hormones corticotrophin (ACTH) and growth hormone (GH) have particularly significant metabolic consequences. Other anterior pituitary hormones such as thyrotrophin (TSH) and the gonadotropins (follicle stimulating hormone (FSH) and luteinizing hormone (LH)) are not as significantly affected.

Agents Generating the Stress Response to Surgery

Cortisol: Corticotrophin-releasing hormone (CRH), released by the hypothalamus, stimulates the anterior pituitary release of ACTH into the bloodstream.

ACTH is a by-product of the breakdown of pro-opiomelanocortin. To complete the hypothalamic-pituitary (HPA) axis, cortisol (the stress hormone) is produced by the adrenal glands following ACTH stimulation. The HPA axis is regulated by a negative feedback mechanism in which cortisol suppresses the release of both CRH and ACTH. Cortisol is a catabolic glucocorticoid hormone that mobilizes energy stores to prepare the body for the fight or flight response to stressors. It promotes gluconeogenesis in the liver, leading to raised blood glucose levels. Hyperglycemia reduces the rate of wound healing and is associated with an increase in infections and other comorbidities including ischemia, sepsis, and death. During and after surgery the negative feedback mechanisms fail and high levels of both ACTH and cortisol persist in the blood. In the presence of raised cortisol levels in a severe stress response, the rate of protein breakdown exceeds that of protein synthesis,[1] resulting in the net catabolism of muscle proteins to provide substrates for gluconeogenesis. Further substrates for gluconeogenesis are provided through the breakdown of fat. Triglycerides are catabolized into fatty acids and glycerol, a gluconeogenic substrate.

Growth Hormone: Growth hormone-releasing hormone (GHRH) from the hypothalamus stimulates the anterior pituitary to release GH. Propagation of the GH initiated signal occurs via the insulinlike growth factors which regulate growth. Signaling via these effectors regulates catabolism by increasing protein synthesis, reducing protein catabolism, and promoting lipolysis. Like cortisol, GH increases blood glucose levels by stimulating glycogenolysis. The hyperglycemic effect is also increased due to the anti-insulin effects of GH. However, the increased secretion of GH after surgery is not thought to be important in the perioperative period (Burton, 2004).

Vasopressin: Vasopressin is a major antidiuretic hormone which released from the neurohypophysis. After surgery, it acts on arginine vasopression (V2) receptors in the kidneys, leading to the insertion of aquaporins into the renal wall. Aquaporins allow the movement of water from the renal tubule back into the systemic circulation. Vasopression concentrations can be raised after surgery by pain alone.

CASE REPORT:

We present a case of 24 year old male patient pre morbidly healthy, admitted with complaints of pain in abdomen with complaint of pain in abdomen with h/o RTA with polytrauma on 11/11/2021. Patient underwent surgery outside hospital-pubic rami, pelvic diathesis andsacro-iliac joint fixation-shifted to Noble hospital on 17/11/2021. For this he underwent CT-abdomen+pelvis s/o-sigmoid perforation, peritonitis. Exploratory laparotomy-sigmoidectomywith colostomy done by Dr.AbhijitWhatkar on 18/11/2021. Post op patient shifted to ICU- required mechanical ventilation, transfused blood products and slowly weaning done-extubated on 22/11/2021. Patient required NIV support. Lab showed low potassium. USG thorax done-moderate right side hemothorax approx. volume 300-350cc. mild left hemothorax volume 40-50 cc. Patient treated all supportive treatment and starts tolerating orally. Patient hemodynamically stable, vitally stable functioning colostomy, no distention/tenderness hence planned discharge on 6/12/2021. Patient readmitted on 13th feb , 2022 for colostomy closure. Patient underwent surgery exploratory laparotomy with colostomy closure on 15/02/2022 by Dr.AbhijitWhatkar, entire procedure was uneventful. Post operatively patient was vitally stable on IV antibiotics, antiemetics, antacids, Dressings done regularly. Due to persistence of abdominal pain and fever patient advised CECT abdomen pelvis-s/o loculated fluid collection in left iliac fossa, mild pneumoperitoneum and possibility of anastomotic leak. Patient underwent surgery- exploratory laparotomy with ileostomy on 24/02/2022 by Dr. Abhijitwhatkar. Intraoperatively anastomosis was healthy. There was interbowel pus collection, extensive adhesions and burst appendix. Patient underwent appendicectomy and ileostomy was done. After all the post operative care patient was discharged on 1/3/2022.

DISCUSSION AND CONCLUSION

The diagnosis of appendicitis rests more on clinical examination of the abdomen than on any aspect of the history or laboratory investigations. The diagnosis of appendicitis or peritonitis in post op period may be difficult because there are no clinical sign because in patient already stressed by surgery, sepsis can escalate quickly and in early post op period sepsis can be difficult to differentiate from normal post op inflammatory response. The cardinal features are those of an unwell patient are low grade pyrexia , localised abdomen tenderness, muscle guarding and rebound tenderness. Since Sneve's early description of the catabolic response in burn patients (Wolfe, 1979), efforts to define and modulate the metabolic responses to injury and stress have continued. The characterization of a 2-phase hypermetabolic response to injury by Cuthbertson, divided the response to injury into quantifiable events (Cope, 1953). The initial ebb phase, occurring within several hours of the injury and lasting for 2-3 days, consists of reductions in cardiac output, oxygen consumption (VO2), the basal metabolic rate, and glucose tolerance. The second flow phase, beginning after the ebb phase and lasting for days to weeks depending on injury severity, is characterized by increases in cardiac output, respiratory rate, VO2, hyperglycemica, skeletal muscle catabolism, and a negative nitrogen balance. Although the ebb and flow phase model is very simple, and may not adequately describe the metabolic responses induced following severe or multiple insults, the correlations established between injury and hypermetabolism continue to guide advancements in surgical care. Postburn muscle catabolism occurs in conjunction with an increase in metabolic rates (Moore, 1959). Soon after this link was made, Moore (Cuthbertson, 1942) suggested that following a traumatic injury, proteolysis and muscle catabolism could be reduced by feeding the patient continuously. Reversal of preexisting protein deficiencies with long-term parenteral nutrition (PN) was explored to achieve a reduction of the risk of postsurgical complications (Jeschke, 2012).

The postsurgical hypermetabolic response is induced by increased serum catecholamine concentrations and maintenance of ambient temperature or administration of pharmacotherapeutic agents. Both burn and severe traumatic injuries induce a hypercatabolic response that, along with a heightened inflammatory response, leads to organ failure.[3] The attenuation of the hypermetabolic response through nutrition and pharmacologic interventions results in improved patient outcomes. Traumatic injury induces inflammatory and hormonal responses that alter metabolic processes, thereby changing nutrition requirements. The stress response to injury evolves temporally as the patient moves through the ebb and flow phases and eventually into the rehabilitative period. During each of the periods, nutrition requirements and nutrient intake, absorption and substrate utilization are different.

Major surgery, critical illness and sepsis, and traumatic and burn injuries induce elevations in the metabolic rate in an attempt to restore homeostasis. Although initially beneficial, the exaggerated and prolonged inflammatory, metabolic, and catabolic responses induce clinical complications, delay recovery, and increase mortality. Inflammatory, hormonal, and stress signaling mechanisms drive the hypermetabolic response including elevations of circulating catecholamines, glucocorticoids, and glucagon, with subsequent increases in gluconeogenesis, glycogenolysis, and protein catabolism. Insulin resistance and peripheral lipolysis develop as well. Surgical stress may impair metabolism, thereby negatively affecting the body's ability to grow, heal, maintain homeostasis, or adapt to the patient's surroundings. This may also affect the metabolic pathways through which absorption of nutrients and subsequent break down leads to generation of energy. Nutrition supplementation is needed to counteract dietary deficiencies, to augment the reduced function of the alimentary tract, and to support healing and recovery. Improved understanding of the pathophysiological response to injury has enabled the development of nutrition supplementation protocols that support recovery while reducing the effects of muscle catabolism, the major contributor to postsurgery or trauma adverse outcomes.

To heal following surgery and injury, increased availability of glucose, amino acids, and other nutrients is needed. Muscle proteolysis facilitates the release of glutamine and alanine following neuroendocrine and inflammatory signaling. These amino acids are required for protein synthesis and also serve as the building blocks fueling hepatic gluconeogenesis. Alanine and ammonia are derived from glutamine in the gut; these compounds are either used or converted to urea. To limit protein catabolism for the release of these amino acids, adequate nutrition supplementation is required.

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