Review Article

Renal endocrine manifestations during polytrauma: A cause of concern for the anesthesiologist

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ABSTRACT

Nowadays, an increasing number of patients get admitted with polytrauma, mainly due to road traffic accidents. These polytrauma victims may exhibit associated renal injuries, in addition to bone injuries and injuries to other visceral organs. Nevertheless, even in cases of polytrauma, renal tissue is hyperfunctional as part of the normal protective responses of the body to external insults. Both polytrauma and renal injuries exhibit widespread renal, endocrine, and metabolic responses. The situation is very challenging for the attending anesthesiologist, as he is expected to contribute immensely, not only in the resuscitation of such patients, but if required, to allow the operative procedures in case of life-threatening injuries. During administration of anesthesia, care has to be taken, not only to maintain hemodynamic stability, but equal attention has to be paid to various renal protection strategies. At the same time, various renoendocrine manifestations have to be taken into account, so that a judicious use of anesthesia drugs can be made, to minimize the renal insults.

Key words: Anesthesia, polytrauma, renal injuries, renal protection, renoendocrine manifestations

Introduction

Major trauma is a pathophysiological state that threatens the integrity of the internal environment, causing alteration in the sympathoadrenal activity. Major trauma continues to be the leading cause of death in the developed as well as developing countries. The deaths caused by major trauma can be classified into immediate, early, and late deaths, depending on the cause.[1] With severe trauma, various changes occur in the body, in order to maintain the homeostasis of the body, so that cardiovascular stability is maintained with the preservation of oxygen supply. The trauma may extend to the renal tissues as well, however, even normal renal tissues respond proportionally to the insult of trauma. The renal endocrine manifestations after trauma are also one of the major protective responses exerted by the body to maintain a normal metabolic and endocrine milieu. The caloric substitutes are mobilized so that supply of glucose to the vital organs is maintained.[2]

The endocrine and metabolic responses of the body to major trauma can be divided into two phases:

The Ebb phase: Lasts for two-to-three days and is represented by hemodynamic, endocrinal, and metabolic instability, characterized by hypotension, hypovolemia, reduced blood flow, increased vascular resistance, and increased secretion of catecholamines, glucocorticoids, and mineralocorticoids, with depletion of hepatic glycogen.[3]

The Flow phase: Is characterized by hyperdynamic responses within the body, such as, water retention, increased vascular permeability, decreased systemic vascular resistance, and growing levels of glucocorticoids and catecholamines, with resulting hyperglycemia and proteolysis.[4]

Renoendocrine Manifestations in Response to Trauma

In the initial phase following trauma, due to a sudden loss

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of blood volume, there is intense vasoconstriction in the body that diverts the blood volume toward vital organs like the heart and brain, at the expense of blood flow to the splanchnic and renal circulation. This phenomenon occurs due to the central sympathetic stimulation, followed by release of catecholamines from the adrenal medulla, causing these protective effects. Thereafter, the intravascular conservation of fluid occurs through pressure-sensitive baroreceptors, which are also found in the renal arteries, resulting in the secretion of various hormones from the pituitary, namely the Adrenocorticotropic hormone (ACTH), growth hormone, and vasopressin (posterior pituitary), thus causing retention of salt and water, to restore the lost blood volume.

Another important change of significant dimension is the stimulation of the renin-angiotensin system. This is governed by the stimulation of neurogenic receptors located within the juxtaglomerular apparatus present in the renal afferent arteriole. These are stimulated by the decreased circulating volume, ACTH, vasopressin, glucagons, and prostaglandins. This process releases renin, which converts angiotensinogen to angiotensin I in the presence of the enzyme angiotensin converting enzyme (ACE), which is further converted into angiotensin II in the lung tissue. Angiotensin is a potent vasoconstrictor, which also increases myocardial contractility and vascular resistance. Angiotensin further stimulates the release of aldosterone from the adrenal cortex, which stimulates salt and water retention from the distal convoluted tubules.

Thus, the duration of hypotension in the early phase, after a major trauma, determines the chances of the development of renal insufficiency, which further depends upon the measures taken to restore the blood volume. However, various risk factors that have been implicated in the development of renal insufficiency following major trauma are pre-existing renal insufficiency, type I diabetes, age more than 65 years and exposure to nephrotoxic agents (contrast dye).[5] Following the early changes of acute trauma, efforts should be made to protect the renal functions, to prevent the development of renal insufficiency.

Acute Kidney Injury in Polytrauma and Rhabdomyolysis

The incidence of acute kidney injury after polytrauma has not been clearly established, but the literary evidence accessed from various retrospective studies indicates figures of 0.5 – 8%. The incidence rises further if these patients undergo any surgical procedure and anesthesia or get admitted into the Intensive Care Unit (ICU).[6-8] The renal, endocrine, and metabolic functions get severely impaired during the episode of acute kidney injury (AKI), which can result from hypovolemia (hemorrhage), sepsis, nephrotoxic drugs and toxins, radio-contrast dyes, and rhabdomyolysis. However, no single cause can be attributed for such an acute insult, and invariably it is the association with multiple risk factors that predisposes the kidney to bear the systemic insults.[9-17] The underlying basic pathology in all types of insults, whether polytrauma or rhabdomyolysis, is the decreased renal perfusion, which leads to deranged renal, metabolic, and endocrine functions.[18,19]

The cellular mechanisms involved in AKI due to rhabdomyolysis in polytrauma patients involve sarcolemmal injury, depletion of ATP in the myocytes, uncontrolled increased calcium influx intracellularly, ischemic reperfusion, and infiltration of neutrophils[20-22] during recovery of various pathophysiological features, and they can pose to be diagnostic and therapeutic challenges. Hypercalcemia during recovery from AKI, due to polytrauma and rhabdomyolysis, can occur as a result of delayed resolution of secondary hyperparathyroidism.[23] Numerous biomarkers of renal injury are used to estimate the presence and extent of renal injury and failure, and include cystatin, kidney injury molecule, interleukin-18, and Neutrophil Gelatinase Associated Lipocalin (NGAL).[24]

The prevention of AKI with forced diuresis, with frusemide and other diuretics, has met with conflicting results, mostly negative, which is evident from various studies carried out on polytrauma patients. Rather diuresis with these pharmacological agents has impaired renal metabolic and endocrine functions, raising the mortality and morbidity.[25-27]

Various studies have advocated the use of intravascular volume expansion and alkaline diuresis (urine output > 200 ml/hour), which prevents the formation of myoglobin casts in the renal tubules. It has also successfully demonstrated that creatinine kinase > 5000 U/L warrants treatment with a combination of renoprotective strategies, including aggressive hydration, bicarbonate, and mannitol.[28-30]

Nowadays, another important scenario is encountered, during renal procurement from polytrauma brain-dead patients; utmost care is taken to preserve the hemodynamic stability of the brain-dead patient, and the patient is ideally treated in the ICU. The procedure has to be performed on an urgent basis because any delay in procurement increases the chances of complications and damage to the kidneys and other vital organs. Brain-dead donors are particularly vulnerable to numerous complications such as diabetes insipidus, cardiac arrhythmia, and endocrine

dysfunction. Therapeutic strategies for diabetes insipidus and its resultant complications, such as, hypovolemia, hypernatremia, hypokalemia, and hyposmolarity, include pitressin or desmopressin acetate (DDAVP).[31,32]

Renal Protective Strategies

Numerous strategies and techniques have been used from time to time for prevention of acute renal insult during polytrauma and renal injuries.

1. Intravascular volume expansion: Correction of volume status should continue with intravascular fluids during the initial as well as later phases following trauma. The role of crystalloids compared to colloids in intravascular volume expansion remains unclear; however, the first fluid used in patients with multiple trauma is usually crystalloids. The colloids used in these patients include blood and blood products, dextran solution, albumin, and starch solutions. These remain in the intravascular space for a longer duration and increase the cardiac output more than crystalloids, but on the other hand, they have some drawbacks like more chances of allergic reactions, nephrotoxicity, and coagulopathy. Fully cross-matched whole blood is ideal for volume replacement, but unfortunately is not available readily and may have the risk of transmission of infections. The controversy between crystalloids and colloids will continue and greater number of patient outcome studies is required to prove either side of argument.
2. Maintain renal perfusion pressure: Maintenance of optimal cardiac output and systemic arterial pressure is essential for preserving renal perfusion pressure. This is achieved initially by maintenance of normovolemia by intravascular volume expansion. Inotropes and vasopressors may be required for low cardiac output and persistent arterial hypotension, with the aim of keeping the minimum mean arterial pressure between 65 and 75 mmHg. Norepinephrine is considered as the first-line vasopressor, as there is no firm evidence that it compromises renal, hepatic or gastrointestinal blood flow at the doses used.
3. Renovascular vasodilatation: The renal vessels are rich in dopaminergic receptors both DA_1 and DA_2. The DA_1 receptors are post synaptic, cause renal vasodilatation, and thus increase renal blood flow. The DA_2 receptors are post synaptic, cause renal vasoconstriction, and thus reduce renal blood flow.[33] Some clinicians do advocate the use of low-dose dopamine (0.05 – 2.5 ug / kg / minute) in maintaining renal perfusion, due to stimulation of renal dopamine receptors, but recently various literary evidences suggest that low-dose dopamine does not provide any renal benefits,[34,35] and there are reports that this low-dose dopamine may be associated with increased incidence of cardiac arrhythmia and worsening of renal functions;[36] this may be attributed to its receptor non-specificity.
   Fenoldopam is a selective DA1 receptor agonist that has been studied recently. It has been seen to improve creatinine clearance, improve renal blood flow, and reverse the effects of cyclosporine A toxicity. By selective vasodilatation of renal and mesenteric vessels, it enhances perfusion to the gut and kidneys in hypovolemic states, as in hemorrhagic shock following multiple trauma.[37,38]
4. Maintenance of tubular flow: Obstruction to the flow through renal tubules due to shed casts or cellular debris can cause cell ischemia and death, thus causing renal dysfunction. The use of loop diuretic and mannitol has been advocated in maintaining the tubular flow and thus preventing renal dysfunction, but randomized trials are lacking.[39]
5. Other pharmacological agents: Recombinant atrial natriuretic peptides (ANP) have been found to be useful due to their natriuretic and diuretic effects, by increasing glomerular perfusion pressure and thus filtration, but the literary evidence is still inconclusive
   Other agents like N-acetyl cysteine (NAC), Insulin like growth factor-1 (IGF-1), Erythropoietin, and mesenchymal stem cells are being studied for their renal protective effects.
6. Glycemic control: Strict glycemic control, using intensive insulin therapy (IIT), has been found to reduce chances of renal dysfunction, especially in previously diabetic patients as multiple trauma induces a hyperglycemic state, which may aggravate the renal injury.

Thus, there are alterations in each and every system of the body, and the kidney tissue also bears a major brunt, resulting in various metabolic and endocrine manifestations in response to the trauma. The renal tissue exerts its protective mechanisms through various endocrine and metabolic pathways, which are aimed at restoring the homeostasis of the body in patients with multiple trauma. These patients often come for surgical procedures that may be definitive or damage control and the attending anesthesiologist is faced with the management of such unstable patients. Renal protective strategies should be continued in the intraoperative period also, which involve the following procedures:

Preoperative Concerns and Evaluation

Preoperative assessment

Preoperative assessment of trauma patients starts with the evaluation of airway, breathing, and circulation (ABC).

Once the A, B, and C are taken care of, then further evaluation must be done for the exact nature and extent of the injury. A complete history must be taken about the nature and type of injury sustained. A thorough evaluation must be conducted for the presence of occult injuries like cervical spine injuries with focal neurological deficits, pneumothorax, cardiopulmonary contusion, and so on. These types of patients are likely to have renal trauma as well, with resultant endocrine and metabolic features of renal decompensation.

Laboratory investigations

Preoperative laboratory investigations should be done immediately, which should include (if possible) a cervical spine radiograph, chest radiograph, electrocardiogram, and complete blood count, and typed, cross-matched blood should always be in hand before a surgical procedure. A coagulation profile, renal functions, and electrolytes are also desirable, but should not delay the surgical procedure. Whenever renal injury is suspected, a computed tomography (CT) scan is the investigation of choice, especially in cases of retroperitoneal hemorrhage.

Premedication

Premedication in these cases is usually sparingly used due to the danger of excessive sedation; however, anticholinergic drugs can be used to reduce the secretions. Antacids are very useful prophylactic agents if the patient is cooperative, to prevent aspiration.

Operating Room protocols

The Operating Room should be kept ready by warming to a specific temperature in advance, the airway equipment should be checked and other necessary equipments like fluid warmer, drugs, body warmer should also be arranged accordingly. When shifting the patients to the Operating Room, supplemental oxygen should always be administered and the neck should always be stabilized if the cervical spine is not cleared.

It should always be remembered that the resuscitation of a traumatized patient should never be discontinued during the preparation of the patient for surgery.[40]

Perioperative Monitoring

All multiple trauma patients should have a minimum monitoring involving five lead electrocardiograms, automated blood pressure, pulse oximetry, core temperature probe, and capnograph. Capnography is particularly important as it can detect changes in the alveolar dead space, which in turn reflects changes in the volume status. An indwelling Foley's catheter should be in place to monitor hourly urine output. An arterial catheter for monitoring invasive blood pressures may be helpful in hemodynamically unstable patients undergoing major surgeries.

Central venous pressure monitoring is essential, especially in cases of renal trauma with significant blood loss, to guide the fluid therapy. The internal jugular vein is usually preferred (due to less chances of pneumothorax), however, the subclavian vein is preferred in head trauma, to prevent obstruction of the venous return from the brain. The femoral vein is usually not preferred in patients with potential intra-abdominal hemorrhage, such as, massive renal or hepatic trauma, but may be used in isolated limb injuries or head trauma. A pulmonary artery catheter may be useful in cases with massive blood loss or transfusion, impaired left ventricular function, pulmonary edema or sepsis.

Another important monitor is the transcutaneous oxygen monitor ($P_{tc}O_2$), which monitors tissue oxygen delivery. It can detect a fall in the arterial oxygen content and skin perfusion.

Administration of Anesthesia

An ideal induction agent for patients with multiple injuries should rapidly induce anesthesia and amnesia, provide excellent intubating conditions, and be devoid of cardiovascular, respiratory, and cerebral side-effects. However, no single inducing agent meets all the above-mentioned criteria. Nonetheless, few anesthesia-inducing agents can be used in trauma patients owing to their less hemodynamic side-effects and ability to provide optimal intubating conditions. The various agents that can be used are:

a. Etomidate: It is an ultra-short acting inducing agent, which is used as the preferred inducing agent in emergency trauma patients, since 1983.[41,42] It is quickly redistributed to the inactive tissues and is hydrolyzed by plasma esterases into inactive substrates. It has the most favorable hemodynamic profile, with minute alterations in the mean arterial pressure.[43,44] It also has cerebral protective effects, as it reduces cerebral blood flow, oxygen consumption, and intracranial pressure. Etomidate has also been found to suppress the adrenal release of cortisol, especially during prolonged use, for sedation in the Intensive Care Unit and there is a mild propensity to cause dose-related myoclonic jerks.[45,46]
b. Propofol: It is a short-acting agent, which produces better intubating conditions as compared to Etomidate,[47]but is not preferred in trauma patients due to its cardiac depressive effects, which produce a precipitous fall in the mean arterial blood pressure. Hence, these depressant

effects of propofol can be counterproductive to the resuscitation of the trauma patient.

c. Ketamine: It is one of the most popular inducing agents in trauma patients, owing to its sympathomimetic effects of increase in heart rate and mean blood pressure, which is desirable in trauma patients. It produces effective anesthesia, sedation, and analgesia, without compromising the airway protection or spontaneous respiratory efforts.[48] However, it should be kept in mind that ketamine is actually a cardiac depressant, but this effect is overshadowed by its propensity to release catecholamines from the adrenal medulla, and thus raise the heart rate and cardiac output. This effect of ketamine is important in multiple trauma patients with hemorrhagic shock, as they are in a state of depleted catecholamines, and the use of ketamine can further aggravate shock due to its cardiac depressant effects. It also has the propensity to cause increased intracranial tension, thus limiting its use in patients with traumatic brain injury. On the contrary, ketamine by its antagonistic effects on the NMDA receptors in the brain, has been found to prevent neuronal death in head injury patients.
d. Thiopentone: This ultra-short acting barbiturate is also used commonly for anesthesia induction, but is not very popular in trauma patients owing to its cardiodepressant effects of reducing the mean blood pressure.

Neuromuscular Blockade

The most common neuromuscular blockers used in the setting of a trauma patient are succinylcholine and rocuronium.

a. Succinylcholine: It has been utilized in trauma patients since many years and has been found to give optimal intubating conditions within 30 to 60 seconds, with a short duration of action. Although, its use is confronted with various side-effects like hyperkalemia, raised intracranial and intraocular pressures, and cardiac arrhythmias, it is still considered one of the best paralyzing agents in the rapid sequence intubation scenarios found in trauma patients.[49,50]
b. Rocuronium: It is also considered a good paralyzing agent in trauma patients, where succinylcholine is relatively contraindicated. In a dose of 0.6 – 1 mg / kg intravenously, it produces ideal intubating conditions within 55 – 70 seconds, but the action lasts for about 30 – 60 minutes. The main disadvantage with rocuronium is, its longer duration of action makes managing the failed airway more difficult.[51] However, with the introduction of a selective rocuronium antagonizing agent, suggamedex, it is possible to antagonize the rocuronium-induced muscle paralysis, making it advantageous in patients where succinylcholine is contraindicated.[52]

Most of the drugs used in the setting of multiple traumas are excreted by the kidneys, so in the setting of renal trauma it becomes very essential to closely monitor the functioning of the kidneys. Equally important is the dose adjustment for individual anesthetics in the setting of renal failure due to polytrauma. Drugs with predominant renal excretion, such as, benzodiazepines, opioids, pancuronium, vecuronium, and so on, require careful dose titration and adjustment, so as to prevent toxicity due to cumulative effects.

Trauma to the kidneys may be associated with significant retroperitoneal hematoma, which may present with sudden cardiovascular collapse, so any such suspicion during the conservative management of renal trauma should alert the clinician, and surgical intervention should be done to reverse it.

Conclusion

In conclusion, polytrauma is a state of acute stress to the body, and the body tries to achieve homeostasis by bringing about various physiological changes, which restore the disturbed systems of the body to a normal state. Renal and endocrine changes are the most important in restoring the disturbed homeostasis. Initial resuscitation of polytrauma patients is very essential for a positive outcome, and once initial resuscitation has been performed, definitive treatment must be carried out in the form of surgical procedures. Providing anesthesia in such multiple injured patients is a very daunting task, as the attending anesthesiologist is faced with a patient with disturbed hemodynamics, where there is no time to completely stabilize the patient as the surgical procedure is essential to save the patient's life.

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