Basics in Clinical Nutrition: Nutritional support in trauma

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Learning objectives

– To characterize metabolic changes specifically related to trauma
– To be able to prescribe nutritional support in trauma patients
– To be aware of new substrates used in trauma patients
– To characterize metabolic changes connected with severe head trauma and the consequences for nutritional support.

1. Pathophysiology of trauma

Trauma is defined as any physical damage to the body and often occurs in young patients, who have little or no protein-depletion. It includes an immediate cardiovascular response, an inflammatory response occurring several hours after the injury, and finally a metabolic response, which has to be taken into account, particularly during recovery (Fig. 1).

1.1. Cardiovascular response

The cardiovascular response associates hemorrhage, tissue damage, pain and anxiety and has three phases:

– First, heart rate and total peripheral vascular resistance increase to maintain blood pressure.
– After a loss of a third of blood volume, blood pressure falls and is accompanied by bradycardia and syncope.
– Finally, when about 44% of blood is lost, heart rate increases again massively.

On the other hand, tissue damage alone induces tachycardia, increased blood pressure, increased sympathetic efferent activity and redistribution of blood flow to skeletal muscle. This contrasts with hemorrhage, which results in diversion of blood flow to vital organs. When hemorrhage and tissue damage coexist, as in trauma, the response to tissue damage prevails. As a consequence, ischemia of gut mucosa may be initiated and translocation of bacteria and endotoxin from the gut, as demonstrated in animal experiments, can occur.

1.2. Inflammatory response

During the inflammatory response there is an increased production of cytokines (TNF-α, IL-1, IL-6, IL-10). These cytokines are probably produced in the gut (via stimulation of the gut associated lymphoid tissue) as well as locally at the wounded tissue. This change in cytokine secretion may be related to the development of multiple organ failure (MOF). The recognition that some patients early and some go into MOF late led to further subdivisions of the inflammatory response. One model describes a systemic inflammatory response syndrome (SIRS) followed by a phase of immunosuppression. Another model hypothesizes a “one-hit” or “two-hit” insult. The “one-hit” insult represents the MOF occurring immediately after severe trauma and the “two-hit” insult represents the MOF triggered by a secondary insult, as surgery, infection and/or ischemia a few days after trauma.
1.3. Metabolic response

Finally, the metabolic response consists mainly of hypermetabolism, mediated by the stimulation of catabolic hormones (glucagons, catecholamines and corticoids) and insulin resistance. Associated with inadequate nutrition, the administration of drugs as glucocorticoids and physical immobilization, this neuroendocrine response leads to protein breakdown to amino acids which are used to produce de novo glucose in the liver. It is suggested that this glucose, in turn, supplies energy anaerobically to cells with undeveloped mitochondria, e.g. granulation tissue of wounds. In trauma patients, wound healing is delayed, maybe as a result of altered lymphocyte function or drugs.

2. Wound healing and nutrition

Wound healing depends on nutritional state. Moderate or severe protein energy malnutrition, as often found in critically ill patients, impairs or delays wound healing. A protein deficiency interferes with new capillary formation, fibroblastic proliferation, production of proteoglycans, and collagen synthesis. Thus, protein intake is essential for wound healing. Among the amino acids, arginine has been reported to enhance wound healing and immune function. Its mechanism of action may be partly mediated by an increase of growth hormone secretion. Other nutrients involved in the complex mechanism of wound healing include vitamins A, C, and E as well as trace elements (zinc, copper, selenium, manganese). The role of iron still has to be demonstrated but severe anemia has been shown to reduce wound healing.

3. Timing and route of feeding

Early nutritional support (within 72 h) is more beneficial than delayed feeding in patients with blunt or penetrating trauma because it leads to a threefold decrease in sepsis. Total enteral nutrition (TEN) is preferred to total parenteral nutrition (TPN) because it allows better utilization of nutrients, prevents gut mucosa atrophy, preserves gut flora, reduces stress response and maintains immunocompetence. Patients with an Abdominal Trauma Index > 15 and fed by TEN show reduced septic complications from intra-abdominal abscesses and pneumonias compared to those fed by TPN. In case of severe trauma (Injury Severity Score > 18 and Abdominal Trauma Index > 20) or less severe trauma with flail chest, spinal cord injury, severe pelvic fracture, major soft tissue injury or closed head injury, small bowel feeding is indicated. Gastric feeding may be poorly tolerated during the 3–4 first days after trauma because of gastroparesis. If a laparotomy is performed, a fine-bore jejunalostomy is preferred to a nasogastric tube.

However, small bowel feeding is contraindicated in case of full-blown shock, sepsis and incomplete resuscitation causing reduced splanchnic blood flow, which may then cause non-occlusive bowel necrosis. Indeed, the small bowel requires an increased blood flow to absorb nutrients. This cannot be achieved by the shocked patient.

Total parenteral nutrition should be given in cases of prolonged gastrointestinal dysfunction, e.g. massive small bowel resection, high-output fistulas, intolerance to enteral feeding and high risk of non-occlusive small bowel necrosis (shock resuscitation, high dose of alpha-agonists and intermittent hemodialysis with haemodynamic instability).

4. Energy needs and delivery

Patients with moderate trauma need about 25–30 non-protein kcal kg⁻¹ day⁻¹ (NPC) with 1.0–1.5 g kg⁻¹ protein and a NPC Nitrogen ratio of 80–120. All protein losses such as those due to chest or abdominal drainage, severe skin damages, proteinuria or intestinal secretion should be fully replaced. With resolving stress, the energy requirements remain the same but the protein needs decrease to 1.0–1.2 g kg⁻¹, leading to a NPC: Nitrogen ratio of 130–160. Immune-enhancing diets are not recommended for these patients.

In patients with severe trauma (Abdominal Trauma Index > 20 or Injury Severity Score > 18) or less severe trauma with complications, the calorie needs remain the same but an increased intake of protein may be beneficial. In this perspective, immune-enhancing diets (2.2–2.5 g protein kg⁻¹ day⁻¹), which are enriched in L-arginine, glutamine, ω-3 fatty acids and/or nucleotides may be useful since they induce fewer infections, less MOD, decreased use of antibiotics and shorter length of hospital stay than polymeric diets. These diets should be continued for 7–10 days only and changed to standard diets thereafter since, in the long term, they induce an energy deficit (Table 1). The advantage of fiber-enriched diets has not yet been demonstrated in trauma patients but they do not have any deleterious effects either. Intracolonic fermentation of fibers liberates energy-rich precursors, such as butyrate, for the colonocytes. In addition fibers-enriched diets mimic the physiologic feed profile and should therefore be used.

After shock resuscitation, enteral nutrition should be started at 15 ml h⁻¹ and advanced to 25 ml h⁻¹ eight to 12 h later. A daily increase of 25 ml h⁻¹ to goal depending upon tolerance is advised. In case of severe pelvic fractures with large retroperitoneal hematomas, an Abdominal Trauma Index > 40, multiple bowel injuries

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Route of feeding</th>
<th>Formula</th>
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<tbody>
<tr>
<td>Severe trauma or moderate</td>
<td>Enteral</td>
<td>Immune-enhancing for 7–10 days*, then high protein and fiber-enriched polymeric diet</td>
</tr>
<tr>
<td>trauma with complications</td>
<td>Parenteral</td>
<td>Standard “All-In-One” solution</td>
</tr>
<tr>
<td>Moderate trauma</td>
<td>Enteral</td>
<td>Fiber-enriched polymeric solution</td>
</tr>
<tr>
<td>Intolerance to enteral feeding</td>
<td>Parenteral</td>
<td>Fiber-enriched polymeric solution</td>
</tr>
<tr>
<td>Risk of non-obstructive</td>
<td>Parenteral</td>
<td>High in water, K, P.</td>
</tr>
<tr>
<td>bowel necrosis</td>
<td>Parenteral</td>
<td>Mg, Ca</td>
</tr>
</tbody>
</table>

Table 1. Routes and types of nutritional support according to the organs injured during trauma. (A recent publication by Heyland et al. in JAMA 2001 suggests that Immune-enhancing diet may increase the risk of mortality in critically ill patients.)
and spinal cord injuries, energy delivery may be difficult to advance because of reduced gut motility.

5. Severe head injury

Patients with severe head injury suffer hypermetabolism, hypercatabolism, hyperglycemia, acute phase response and immune system alterations (Table 2).

Their energy expenditure equals 135–165% of the basal metabolic rate calculated by the Harris-Benedict equation. This very high energy expenditure is related to increased catecholamine levels and possibly to hyperactivity of the autonomic nervous system. It is highest during the first three days after injury and correlates inversely with the Glasgow Coma Score. Patients with decerebrate/decorticate activity show higher energy expenditure than those with flaccid weakness. On the other hand, brain dead patients are hypometabolic and present an energy expenditure of 70–80% of basal metabolic rate. Protein breakdown increases as a result of immobility, decreased nitrogen efficiency, steroid administration and decreased nutrient intakes. Protein breakdown leads to muscle wasting and lower levels of visceral proteins (prealbumin, albumin, transferrin). Low serum albumin concentrations are also a reflection of redistribution due to inflammation and to dilution from fluids. Urinary nitrogen loss is high (12–30 g day⁻¹) for at least 3 weeks after head injury but it is not yet clear whether this is a sign of hypercatabolism.

Hyperglycemia, frequent during the first 24 h after head injury and other traumas, aggravates the preexisting ischemia, probably by stimulating the anaerobic metabolism and thus increasing intracellular lactate and acidosis in brain tissue. The increase levels of intracellular lactate contribute to neuronal damage. Hyperglycemia is positively correlated to severity of head injury and neurological outcome and inversely correlated with Glasgow Coma Score. A continuous infusion of insulin is indicated with glucose metabolism Abdominal and torso trauma Head trauma

<table>
<thead>
<tr>
<th>Metabolic change</th>
<th>Abdominal and torso trauma</th>
<th>Head trauma</th>
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<tbody>
<tr>
<td>Hypermetabolism</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Protein breakdown</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Acute phase response</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Immune system alterations</td>
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Nutritional support should be initiated only when intracranial pressure and peripheral hemodynamics have been stabilized. As for other traumas, early nutrition is preferable to delayed nutrition because it improves outcome and decreases infections rate. The enteral route is preferred to the parenteral route for the reasons explained under “timing and route of feeding”. However, in contrast to other traumas, enteral feeding has little effect on infectious complications. If enteral nutrition cannot be initiated 48 h after admission, parenteral nutrition has to be considered. Gastrointestinal motility may also be reduced because of pheno-barbital-induced coma. Post-pyloric feeding is therefore preferable although most patients tolerate gastric feeding within 72–96 h of injury.

Regarding energy intakes, the physicians should take care not to overfeed these patients. Indeed, conversion of excess nutrients to glycogen or lipid requires energy and thus increases O₂ consumption. This in turn leads to an increased CO₂ production and dilation of brain arteries, which raises intracranial pressure. If CO₂ production is seen as a deleterious factor, then total energy intake should be decreased. In previous reports, a decrease of the carbohydrate to lipid ratio has been advised because glucose oxidation leads to 30% more CO₂ production than lipid oxidation. However, this is no longer relevant because a high lipid intake has been associated with immune dysfunctions.

6. Summary

Trauma is characterized by combination of cardiovascular, inflammatory and metabolic responses. During the cardiovascular phase, priority is given to resuscitation and maintenance of vital functions. Nutritional support is useful during the inflammatory and metabolic phase and improves the patient’s outcome. TEN versus TPN, as well as early TEN versus delayed TEN reduce the incidence of septic complications in trauma patients. In trauma patients, post-pyloric feeding may be preferred to gastric feeding for the first 3–4 days after trauma. Immune-enhancing enteral diets may be useful in severely injured subjects. In head-injured patients, specific attention should be given to control of intracranial pressure. For this purpose, overfeeding should be avoided.

Conflict of interest

There is no conflict of interest.

Further reading