UNHEALED WOUNDS, HYPOALBUMINAEAMIA AND CACHEXIA IN A BURNED CHILD - WHERE IS THE LIMIT?*

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SUMMARY. In our case report we attempt to reconstruct the case of a 10-yr-old boy in Iraq with severe burns who survived almost six months without any adequate treatment, i.e. adequate treatment according to our therapeutic standards. We also highlight the patient’s predominant symptoms, such as open wounds, hypoalbuminaemia, and cachexia.

Introduction

The skin is a complex multistructural and multifunctional organ and the first barrier to the outside world. The major function of the epidermis is to keep invading organisms “out” and water “in”. The dermis adds strength to the skin, since it is made of collagen and other extracellular matrix proteins. It contains a vascular and neural plexus. The vascular plexus is vital for temperature control and the neural plexus gives the skin the ability to sense the environment. The dermis also contains the skin adnexa (hair follicles and oil and sweat glands), which are lined by epithelia cells. These adnexa are essential for the healing of superficial burns.

The burn wound is a major source of inflammatory mediators which lead to hypermetabolism, muscle wasting, and, potentially, dysfunction of multiple organ systems. The best way to treat these systemic problems is to eliminate the source of the inflammatory mediators by removing the eschar and covering the wound. When the burn wound is large enough the mediators spill over into the systemic circulation and cause total body oedema and overwhelm any other body defence.1

Until 1970, when Zora Janžekovič reintroduced the method of early tangential excision of the burn wound with immediate skin grafting, burn wounds were treated by permitting the eschar to separate.7 Early wound excision and grafting of full-thickness wounds changed the natural course of the burn injury. The removal of the necrotic tissue and biological wound closure minimized the incidence of invasive wound infection and sepsis in paediatric burn patients.4

Case report

In May 2003 two children from Iraq with burns were admitted to the Children’s Burns Unit in Graz. They belonged to a group of eleven paediatric war victims who received medical treatment in Austria. One of them was a 10-yr-old boy, whose father gave us his medical history.

While carrying home a leaky can containing fuel oil, the boy got his trousers drenched with this flammable solvent. He entered a room where there was an open fire and his trousers immediately ignited. The boy sustained flame burns to his legs and a small area of the abdomen. This happened on 1 January 2003. The previously healthy youngster sustained approximately 25% total body surface area (TBSA) full-thickness burns. He was taken to the local hospital, where he spent two months. The therapeutic details were not known to the boy’s father. Surgical wound treatment was not performed. After two months the child was transferred to the central hospital in Baghdad on his parents’ request. Owing to the state of emergency at that time in Iraq, this paediatric patient was discharged home two weeks later, without having had surgery. This was mid-March 2003. The boy received no medical treatment

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during the following two months.

On the evening of 15 May 2003 we admitted the patient for further treatment. The next morning (16 May) the initial evaluation took place in the operating room. On examination the boy showed to be in a very poor condition. He was confined to bed, apathic, and hypothermic (body temperature <36 °C). He was also extremely underweight (23 kg, including oedema weighing approximately 2 kg), with massive general oedema, most prominently in the legs and feet; his body mass index (BMI) was 11.5 kg/m². The pulse rate was 88 beats per min and regular, the respiration rate 20 breaths per min, and oxygen saturation with pulse oximetry 98% on room air. Blood pressure was 89/69 mm Hg.

The patient presented a soft, non-tender, distended abdomen with hepatomegaly and no palpable mass. The burned skin on the legs and abdomen showed not only massive oedema but also remarkably congestive veins. He had open, foul-smelling, infected wounds on both lower extremities (20% TBSA), and over the os sacrum there was a deep, large decubital ulcer. As a result of his long-lasting immobilization he had suffered contractures in nearly all his joints, and every move or exercise was very painful for him.

On the same day a soft naso-gastric tube and central venous catheter were inserted.

The initial bacteriological findings showed sterile blood and urine cultures, and heavy growth of methicillin-resistant Staphylococcus aureus, Klebsiella pneumoniae, Pseudomonas aeruginosa, Enterococcus and Proteus spp. in wound swabs.

On 17 May, under general anaesthesia, all granulation tissue was excised and decubitus ulcers were surgically debridged. The wounds were temporarily covered with Biobrane™ (Bertec Pharmaceuticals Inc., Morgantown, USA) and Acticoat™ wound dressing (Smith & Nephew Inc.). On the decubitus ulcers we installed a vacuum-assisted closure (VAC) device. Pre-, intra-, and post-operatively the patient received several packed red blood cell units, platelet units, and fresh frozen plasma units. Antibiotics were given for peri-operative prophylaxis only.

From the beginning, enteral tube feeding was fairly well tolerated, but supplementary parenteral nutrition was necessary to ensure delivery of all required nutrients. Initially the patient tended to develop hyperglycaemia and hyperlipaemia and the calorie intake could therefore be increased only slowly and gradually. In the first two weeks it was necessary to use additional heat sources to keep the patient’s body temperature normal, even though he was in a warm environment. Wound dressings were changed regularly, and the physiotherapist practised active and passive ranging and strengthening on a daily basis.

As soon as the catabolic state began to slow down and the donor sites were ready to be harvested, the next surgical treatment was scheduled for 27 May. The right leg was excised down to the viable tissue. Split-thickness grafts were harvested from the scalp, meshed 4:1, and secured in place with meshed Omiderm™ and silver sulphadiazine dressing. The take rate after one week was 80% and the donor site also healed uneventfully.

One week later the right leg was excised in the same way and grafted with split-thickness 2:1 mesh grafts. The donor site was the patient’s back. The take rate after one week was 90%. The decubital ulcer was surgically debrided and grafted with split-thickness 2:1 mesh graft during the same surgical session. The skin graft was held in place on this difficult wound using a VAC dressing for five days. No further surgical procedures were necessary.

Multifaceted management focused on the treatment of pre-existing joint contractures and general muscle weakness as well as scar management. Rehabilitation therapists helped the patient with continued passive and active ranging, scar massage, and play activity to retrain him for the activities of daily life. Compression garments and thermoplastic splints were adjusted.

Thanks to the focused efforts of an experienced burn team the patient was discharged home in a satisfactory condition two months after admission. His wounds and donor sites were completely healed and he could walk again on his own, with negligible extension deficits in the knees and hips. The boy had gained almost 5 kg in weight and was in good spirits.

Table I shows the most pathological laboratory findings on the patient’s admission (normal reference range in brackets) and the wound treatment performed.

<table>
<thead>
<tr>
<th>Table I - Laboratory findings and treatment given</th>
<th>16 May 2003</th>
<th>17 May</th>
<th>27 May</th>
<th>3 June</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin (11.5-15.5 g/dl)</td>
<td>8.1</td>
<td>9.7</td>
<td>9.9</td>
<td>10.9</td>
</tr>
<tr>
<td>Thrombocytes (140-440 x 10⁹)</td>
<td>17</td>
<td>83</td>
<td>534</td>
<td>940</td>
</tr>
<tr>
<td>Serum sodium (135-145 mmol/l)</td>
<td>125</td>
<td>145</td>
<td>136</td>
<td>137</td>
</tr>
<tr>
<td>Protein (5.8-8.0 g/dl)</td>
<td>2.4</td>
<td>4.7</td>
<td>6.2</td>
<td>6.7</td>
</tr>
<tr>
<td>Albumin (3.5-5.5 g/dl)</td>
<td>0.6</td>
<td>3.0</td>
<td>3.1</td>
<td>2.8</td>
</tr>
<tr>
<td>C-reactive protein (3-8 mg/dl)</td>
<td>70</td>
<td>126</td>
<td>83</td>
<td>112</td>
</tr>
<tr>
<td>Cholinesterase (4500-12000 U/l)</td>
<td>1016</td>
<td>2737</td>
<td>2161</td>
<td>2610</td>
</tr>
<tr>
<td>Fibrinogen (180-400 mg/dl)</td>
<td>129</td>
<td>159</td>
<td>553</td>
<td>503</td>
</tr>
<tr>
<td>Body mass index (20-25 kg/m²)</td>
<td>11.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wound excision</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>First wound grafting</td>
<td>-</td>
<td>-</td>
<td>Performed</td>
<td></td>
</tr>
<tr>
<td>Second wound grafting</td>
<td>-</td>
<td>-</td>
<td>Perfomed</td>
<td></td>
</tr>
</tbody>
</table>
Discussion

This is a report on a kind of burns victim that in normal times we would not see here in Austria. We were astounded that a 10-yr-old boy with 25% full-thickness burns could have survived for 4.5 months with almost no treatment. Admittedly, the patient’s condition on admission was disastrous.

It is recorded in the literature\textsuperscript{10} that the chances of survival after burn injury have increased steadily during the past 50 years. At the end of World War II, only 50% of patients survived burns involving 40% TBSA, while today over 50% of all patients with burns in 80% TBSA survive and the survival rate may even be higher for adolescents and young adults. Survival in paediatric patients has improved to such an extent that the survival rate in children with burns involving 100% total body surface area is 50%.\textsuperscript{9}

This remarkable success can be attributed to a number of therapeutic developments, including vigorous fluid resuscitation, early burn wound excision, advances in critical care and nutrition, powerful topical and systemic antibiotics, and the evolution of specialized, multidisciplinary burn centres.

Soon after a burn, changes are observed in the concentration of the plasma proteins. These protein changes affect plasma’s colloid osmotic pressure, which depends on both the concentration and the molecular size of the constituent protein molecules. Solutions of proteins with relatively low molecular weight (albumin, 70,000 daltons) have much greater colloid osmotic pressure per gram of protein than macroglobulins, with a molecular weight nearer one million daltons. Although the albumin concentration may fall to 50% of the normal level during early fluid therapy and persistently stay at that level for weeks after a burn, there is rarely a corresponding fall in the colloid osmotic pressure of plasma. This is due to the prompt appearance of acute-phase reactant proteins, many of which have molecular weights lower than that of albumin and thus have a relatively greater osmotic effect. The decrease in albumin concentration develops and remains, owing to an increased rate of catabolism, losses of exudates from the burn wound, and failure of anabolism to compensate for those losses.\textsuperscript{10}

An aggressive, earlier, and more frequent use of definitive surgical therapy for deep burns has become the norm. The natural tendency of full-thickness burn wounds is to promote an inflammatory response at the junction of the eschar and the underlying viable tissue. At this interface, bacterial proliferation in the eschar attracts polymorphonuclear leucocytes (neutrophils), which release large quantities of proteolytic enzymes and inflammatory mediators. Subsequent enzymatic action results in separation of the eschar from the new granulating interface. If the burn wound is large, the inflammatory response at the burn site becomes generalized. Mediators such as prostanoids, thromboxane, histamine, cytokines, and TNF are all produced and released from the burn site. The hypermetabolic response, with protein catabolism, increased metabolic rate, increased susceptibility to infection, marked weight loss, and poor wound healing, continues until the outpouring of mediators abates.\textsuperscript{11-13}

A burn patient’s metabolic rate can be twice that of a healthy person and cause tremendous wasting of lean body mass within a few weeks of injury. Failure to satisfy these increased energy and protein requirements results in impaired wound healing, cellular dysfunction, decreased resistance to infection, and ultimately death. Burn patients who lose 20% weight or more are in danger of a fatal degree of inanition and of succumbing to respiratory failure, pneumonia, and systemic infection. The hypermetabolism seen after a burn injury is a consequence of hormonal changes. A burn trauma produces a major increase in the catabolic hormones epinephrine, cortisol, and glucagons. The result is greatly accelerated gluconeogenesis, glycogenolysis, and muscle proteolysis. The clinical consequence of these hormonal changes is a markedly increased metabolic rate with skeletal muscle serving as a preferred fuel.\textsuperscript{13-15}

The heightened susceptibility of burn patients to infection has been noted for over 30 years. Since regulation of acute phase response is stimulated by many of the inflammatory cytokines produced by local macrophages and monocytes at the injury site, local induction may well match or exceed systemic induction in its importance to the host. A burn patient’s immunological status has a measurable impact on outcome in terms of survival, major morbidity, and death. The greatest difficulty in attempting to decipher the body’s response to injury is the complex interrelationship of the cytokine cascade, the arachidonic acid cascade, and the neuroendocrine axis.\textsuperscript{16,17}

Most mesenchymal wounds, which are caused by horizontal damage, heal through the formation of granulation tissue. Granulation tissue is a specialized young connective tissue, so called because of its bright red granular surface created by multiple, raised capillary loops, which form a structural axis surrounded by cells and extracellular matrix. It is basically a highly cellular, vascular, and fairly well-organized anatomical, physiological, and functional structure, used primarily to “clean” damaged areas (via macrophages), to “fill” existing gaps (via collagen), and to “feed” growing active new mesenchymal tissue (via neovessels). It is a very dynamic cellular and extracellular matrix which in time will progressively be replaced by a permanent, distinct, mature connective tissue called a scar. Adverse wound conditions will increase the possibility of abnormal healing.\textsuperscript{18}

The burn wound is considered to be a major source of inflammatory mediators, which play an important role
in initiating and maintaining the post-burn inflammatory response. The consequent reactions are associated with increased morbidity and mortality. Owing to the increased catabolism rate, losses of exudates from the burn wound, and the failure of anabolism to compensate for such losses, a decrease develops in albumin concentration and remains. In our patient the incredible albumin level was 0.6 g/dl.

The metabolic rate of burn patients can be twice that of a normal person, causing tremendous wasting of lean body mass within a few weeks of injury - with skeletal muscle serving as a preferred fuel. In our patient the unimaginable BMI was 11.5 kg/m².

**Conclusion**

Treating full-thickness burns by waiting for spontaneous eschar separation is not only a prolonged process associated with much pain and suffering but also a cause of severe metabolic derangements. As long as the body is exposed to foreign antigens and organisms, it will continue to “try to protect” the organism with an inflammatory response. If large amounts of virulent bacteria are present, the host will be overwhelmed and the infection will be destructive. However, in our patient the granulation tissues established a kind of a “stable equilibrium”. Owing to the local defence system, in this case the bacteria colonizing the granulation tissue were successfully prevented from invading the wound, subjugating the organism, and generating a fatal sepsis. Although the boy was healthy and of normal weight prior to the injury and although it was a chronic process to which the body might to a certain extent have adapted, his condition on admission was disastrous. The best illustration of the situation was the computer message after the numbers for BMI calculation had been entered - the message read: “Check your numbers”. And that was exactly what we felt like when we examined the boy for the very first time in our operating room.

**RÉSUMÉ.** Dans cette relation nous décrivons le cas d’un enfant iraquien âgé de 10 ans atteint de brûlures graves qui est réussi à survivre sans recevoir les soins appropriés, c’est-à-dire les soins appropriés selon nos normes thérapeutiques. Nous soulignons en outre les symptômes principaux du patient, comme par exemple les lésions ouvertes, l’hypoalbuminémie et la cachexie.

**BIBLIOGRAPHY**