CRUSHING SYNDROME IN LOWER LIMBS TRAUMA

ABSTRACT

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CONTENT:

I. TEORETICAL STUDY

I.1 Introduction

I.2 Definition of crushing and crushing syndrome; etiology, pathogenesis

I.3 Historical research; the level of actual knowledge

I.4 Crush and crushing syndrome physiopathology

I.5 Local clinical frame and diagnostic elements in crushing trauma

I.6 Complications of crushing

I.7 Treatment principles in mangled extremities

II. MOTIVATION OF STUDY AND WORK HYPOTHESIS

III. STUDY OBJECTIVES

IV. MATERIAL AND METHOD

V. RESULTS OF THE RESEARCH

VI. GENERAL DISCUSSIONS

VII. CONCLUSIONS

VIII. BIBLIOGRAPHY

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The social and economical characteristics of the 21-th century generated a huge change of pathology, including the traumatic one, characterized by increase of energy of trauma agents, resulting in more complex trauma, affecting active population, generating morbidity and important mortality and important social costs. High energy trauma are more frequent, either by direct initial injury of multiple organs and systems (as in polytrauma), either by affecting all the structures of a single segment of one limb, with a severe systemic answer, leading to death of the patient.
There are some special types of trauma when there is no initial multi-organ injury, and still, if the treatment is not early and correct, the patient dies due to final multi-organ failure, and crushing trauma is an example of such a trauma. In this case, the initial injuries are strictly local, but systemic impact soon appears; it is called “Crushing syndrome” and, once appeared, it jeopardizes the patient’s life, demonstrating thus that the human body is one unique structure and its’ defense possibilities are limited.

The problem is even more important since nowadays, crushing may produce sometimes a considerable number of casualties—these situations are called “mass disasters”, represented by natural disasters and terrorist acts; “Disaster medicine” appeared as a response to these situations, in order to minimize their effects on the general population.

Working in an Emergency Hospital, the author choose this subject for the actual research due to these epidemiological aspects, together with its’ complex physiopathology and therapeutic rules, with the specific reason of underlining the necessity of a complex treatment within a multidisciplinary approach.

The first part of the paper analyses the anatomical, physiological and therapeutic elements of the mangled extremity, starting with the clear difference between two terms: CRUSHING represents the mechanism through which a high energy traumatic agent injures the affected limb opposite to a tough surface, the muscles are ruptured, the muscular fibers are completely destroyed and this is called TRAUMATIC RHABDOMYOLISIS. Due to this process, a lot of abnormal metabolic products appear and intoxicate the human body, producing irreversible phenomena responsible for Acute Renal Failure, Multi-Organ System Failure and death.

So we are to discuss about two different pathologies, which must be differentiated in order to treat them properly:

- The crushing mechanism reflects the traumatic MECHANISM which involves three elements:
  1. high energy trauma agent
  2. The striated muscle, which is crushed BETWEEN the traumatic force and the tough surface, and
  3. The underlying surface which acts like a counter-resistance. This pathogenic mechanism is the one which produces the local traumatic effects (traumatic rhabdomyolisis), while
- The “Crushing Syndrome” reflects the systemic effects due to the resobtion of the toxic products produced through traumatic crushing rhabdomyolisis
The first description of the “Crushing Syndrome” was performed in 1941, when E. G. L. Bywaters and Desmond Beall wrote “Crush injuries with impairment of renal function”, published in British Medical Journal; they describe four patients with lower limbs crushed for several hours under ruins; the outcome of these patients was the same: AFTER A FEW DAYS, THE PATIENTS DIED OF Acute Renal Failure, and the autopsies revealed muscular necrosis and considerable renal injuries, with proximal tubular necrosis and the distal tubular obstruction by brown crystals.

During the following years, research was performed in order to clarify the pato-physiology of mangled extremity, which is also reflected in the first part of this research paper.

Following trauma, the energy which is absorbed by the tissues is the difference between the traumatic energy and the resistance of the tissue. If there is also a counter-resistance, the energy which is absorbed represents the difference between the traumatic energy and the tisular resistance PLUS the counter-resistance; due to the fact that the tissular resistance is small compared to that of the traumatic agent and the counter-resistance is also important, the absorbed energy in these cases is considerable higher.

There are two types of crushing:

1. The traumatic agent lasts for a short time, producing the local injuries, and after that, the affected limb is taken off from the traumatic energy. An example is represented by road traffic accidents, when a car crushes the leg.

2. The traumatic agent persists for a longer time (crushing by the ruins); the muscles are injured until decompressions. In this situation, there is always an ischemia due to compression, which is either temporarily, and Reperfusion Syndrome appears, or definitive, and then the Acute Peripheral Ischemia syndrome appear.

The local pato-physiology of the mangled extremity is:

a. Microscopically aspects the traumatic agent produces a total mechanical disruption of the muscular fibers, which no longer look like normal cells and no longer have normal components; their membrane no longer exists and all the intra-cellular components are bulked in the inter-cellular space...

After long-term crushing, the muscular injuries take several steps, like in Ischemia-reperfusion injuries: anaerobe metabolism is initiated, mitochondrial oxidative phosphorilation is inhibited, the mitochondrial and cellular ATP decreases, and the mechanisms ATP-dependant are affected, especially the membrane pumps. The normal intra- and extra cellular components are mixed, the sodium is pushed into the cells, it links water...
and intra-cellular edema appears as the first cellular disturbance within the reperfusion syndrome.

Once the cellular membrane looses (mechanical or chemical) its’ normal properties, including that of keeping apart the two spaces (intra- and extra-cellular), these two spaces are free to perform abnormal exchanges, and the consequences are:

- The membrane potential changes,
- the membrane pumps act reversely- potassium goes out and sodium goes in the cells,
- ATP intracellular level decreases, together with other energetically active products
- membrane lipids peroxidation occurs and the balance between TxA2- Lecutriens is broken
- Potassium, myoglobin and muscular enzymes go out form the cell
- Sodium and water go into the cells (if the cells still exist, like in long-time-crushing), and produce edema

The extracellular migration of all the above mentioned components is followed by:
- hyperkalemia, which affects the cardiac activity,
- serum myoglobin appears ( normally it is absent); myoglobin is a globular protein which contains HEM and it represents the main oxygen-transporting pigment of the muscle; unfortunately, it deposits itself when hypovolemia and acidosis occur and have renal toxicity by a dual mechanism: MECHANIC ( the casts obturate the tubules ) and BIOLOGICAL ( direct necrosis of the tubular cells)
- increasing levels of serum CK and LDH, so called muscular enzymes

b. Systemic pathological phenomena induced by the high energy traumatic agent, the so called “ Systemic Post-Aggressive Reaction” , which represents the response of the organism to trauma, due to the activation of the systems ruling the interaction with the environment. The phases of this reaction are: the initiating phase (the alarm phase, the immediate imbalance phase), which acts on the neuro-endocrinological mechanisms, producing the reactive phase, with catabolic processes, and then, depending on the efficacy of the treatment, there are two possibilities: either the outcome is favorable, and the healing phase takes place, either the Multiple System and Organ Failure occurs, followed by shock and death.
So, the local phenomena are responsible for the most severe systemic complications of crushing trauma:

- the thrombotic risk, due to: the high quantity of thromboplastine released from the tissue, the injuries of the endotelium and the disturbances of the microcirculation
- the toxic risk, due to the products of the anaerobe metabolism and to the myoglobin, and
- the septic risk, since the necrotic tissue is a very good host for the bacteria, and the immune depression can enhance this risk

The clinical aspect is not characteristic for the crushing mechanism, since many types of injuries can result from such a mechanism; the clinical aspect is thus characteristic to these injuries, creating different types of syndromes, depending on the structures which are disrupted by trauma.

The main CLINICAL SYNDROMES following crushing, specific to the DIRECT action of the any traumatic agent, are:

- Contusion (muscular injuries, no bone injury)
- Fractures (closed or open)
- Syndromes produced by vascular injuries; arterial (ischemic or hemorrhagic) or venous (usually hemorrhage).
- Combination of some of the above mentioned

The clinical examination must begin with that of the skin, which can present lacerations, bruisers, longitudinal superficial injuries, thinning of the subcutaneous layer, wounds, blisters, or even skin necrosis areas. Usually, below the necrosis, a DEGLOVING area can be found, due to the phenomenon of traumatic liqutation of the subcutaneous layer and to a muscular haematoma.

This research paper underlines the importance of the following elements: **fluctuence, degloving and muscular tension**, since there are two types of syndromes which can be generated by a crushing mechanism, from the point of view of the Intra-Compartmental Pressure (depending on the status of the unextensible structures), which are: **with increased** Intra-Compartmental Pressure (generating the Compartment Syndrome, with the imbalance between the increasing volume of the content compared to the fix volume of the container) and without increased Intra-Compartmental Pressure.

When Compartment Syndrome appears in a mangled extremity, it has no particular characteristics, but needs the same diagnostic and therapeutic algorithm as a usual one,
especially decompresive fasciotomy during the status phase, otherwise irreversible injuries occur.

The chapter referring to the clinical aspect of crushing trauma underlines the fact that it is unspecific and extremely diverse, without any specificity regarding the mechanism. A mangled extremity can have all the structures affected and the injuries can be unique or associated, depending on the number of these affected structures.

There is a very clear confection between Compartment Syndrome and the crushing mechanism: the crushing mechanism CAN generate a Compartment Syndrome when the fascia and other unextendable structures are intact, but NOT ALL the crushings are followed by a Compartment Syndrome.

The Diagnosis of the Crushing Syndrome is based on identifying the systemic effects of toxicity of the local products of rhabdomyolysis:

- **Crushing injury to a large mass of skeletal muscle.**
- There are sensory and motor disturbances in the compressed limbs, which subsequently become tense and swollen.
- **Myoglobinuria and/or haematuria.**
- **Peak creatine kinase (CK) > 1000 U/L.**
- Renal problems are common with one of the following characteristics; oliguria (urine output <400 ml/24 h), elevated levels of blood urea, serum creatinine, uric acid, K, PO4, or decreased Ca.

The most valuable paraclinical investigations (despite the routine ones and the measurement of the Intra-Compartmental Pressure) are the serum values of CK and LDH, which are the most specific ones for muscular injuries. They have not only a diagnostic value, but also a monitoring one and a prognostic one for the local and general status of the patient with traumatic rhabdomyolysis.

The chapter referring to the complications of these traume describe both the local and the general ones. The most significant local complications are: Acute Peripheral Ischaemia (API), the Compartment Syndrome (CS), the thrombotic complications (arterial and venous-Deep Vein Thrombosis DVT) and the septic complications.

The general complications described in this paper are: the Crushing Syndrome, the fat embolism, the thrombo-embolism and the septic systemic complications: septicaemia and secondary septic disseminations.
The most severe systemic complication is Multiple System organ Failure (MSOF, MDOS) which represents a progressive insufficiency of two or more organs or systems, which cannot maintain their homeostasis without a vigorous external support, corresponding to the cellular translation of shock. MSOF does not represent a separate disease, but a complication of many types of injuries (mechanical, chemical, septic, etc) which quite frequent evolves to death.

The treatment of a patient with a mangled extremity is very complex, local and general, and it needs a multidisciplinary team; within the general treatment, antibiotic and anti-thrombotic treatments are mandatory, and the monitoring of a patient with a high energy trauma is very important and should be performed both clinical and paraclinical (CK, LDH, hemogram, renal tests especially)

As of the local treatment, EMERGENCY SURGERY is MANDATORY in mangled extremities, due to the rapid spreading of the general phenomena induced by the local disturbances, especially those due to muscular necrosis. So, the first step is SURGERY in order to excise all the damaged muscles, perform hemostasis and cover the fracture site with viable tissue.

Surgery takes place in MORE STEPS, (usually called “second-look”) due to the unexpected evolution of these trauma and needs a thorough local and general monitoring of the patient. Modern surgery in mangled extremities have some mandatory steps: exploring, evaluating the injuries, especially the vitality (of muscles, fascia, vascular integrity), and scoring the injuries. In order to choose the type of surgery- amputation or conservative surgery, MESS-Mangled Extremity Severity Score was introduced.

- osteo-muscular injuries (1-4 points)
- shock signs (0-2 points)
- acute ischemia (0-3 points +2 points/6 h of ischemia)
- age (0-2 points)

Total number of points: BETWEEN 3-6 pts- conservative treatment is possible; ABOVE 7 pts- recommended treatment is amputation.

If conservative treatment is chosen, the following are mandatory: mechanical cleaning, hemostasis, the excision of all the dead tissue within the viability limits previously mentioned, a process called NECRECTOMY or DEBRIDEMENT, chemical cleaning with sterile antiseptic solutions, repeated after each step of the excision, treatment of the open fracture (cleaning of the fracture and stabilizing it, usually with an external fixator), meshing all the
remaining spaces to avoid the formation of cavities in which secretions could accumulate, evaluating the possibilities of covering the fractures, as any exposed bone is predisposed to necrosis (covering the fracture will be attempted using viable muscle fascicles without tensing them, otherwise they will necrose), evaluating the possibilities of covering the cutaneous defects WITHOUT tensing the edges of the wound, for the same reason.

If amputation is chosen, it will be done according to the same principles: in obviously healthy tissue, with the stump left open, respecting the rules necessary for the good fit of a prosthesis (rules which are much more flexible now due to technical progresses in the field of prosthetics), after the excision of all necrosed tissue; the premises for a functioning stump are the same as for a functioning limb: viable tissue, mechanical and chemical decontamination.

In the part devoted to the author’s original contribution, we have outlined the reasons for choosing this subject and the work hypotheses, the goals, the research material and method, having used validated statistical methods. As for presenting the results, a statistical study was conducted regarding the demographic characteristics of the studied group, the temporal characteristics (the moment of beginning correct treatment), injury location, clinical forms and incidence of the main complications.

To illustrate the elements earlier described, we analyzed cases characteristic for the clinical signs, the diagnosis and especially the treatment of crush trauma, underlining the therapeutic particularities of each case. The cases are illustrated with an iconography that exemplifies the theoretical data described earlier.

The outcome of the patients treated in the Orthopedics and Trauma Clinic of the Clinical Emergency Hospital of Bucharest reflects the validity of the proposed therapeutic algorithm, as well as the need to use a complex evaluation system with scores that should reflect the local status as well as the systemic impact of the crushing.

The author’s original contribution consists of presenting the local and general criteria that govern the treatment and that must be reference points in monitoring the patients.

For the purpose of summarizing the elements described in the general part as well as those of the author’s original contribution, the chapter dedicated to General Discussions reflects the basic ideas and also compares the data obtained by the author to the data already validated by medical literature, observing that the data obtained in the present research paper is compatible with that from international specialty literature, which makes it valid.
In conclusion, the analysis of the studied cases has shown the need to monitor patients with crush injuries according to the previously described physiopathological data, given the fact that, despite it being an initially localized lesion, the systemic consequences of crush trauma appear quickly (in the absence of quick and correct treatment) and are irreversible, endangering the life of the patient.

The therapeutic algorithm for crush trauma proposed in the present research paper aims to improve local treatment and decrease the systemic impact of this kind of trauma, especially when lesions of this type appear in events with multiple human victims.