Closed fractures associated with acute peripheral ischaemia – diagnostic and therapeutic problems

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Abstract: Acute Peripheral Ischaemia (API) is the most severe acute complication after both opened and closed fractures, as ischaemia compromises not only the vitality of the affected limb, but also the patient's life, the metabolic anaerobic changes following ischaemia having serious local and general consequences.
That explains why early diagnosis of API is very important for the prognosis of the traumatised limb. The main element used for the diagnosis of API is the peripheral pulse, since it's absence is the first alarm sign of ischaemia. After clinical suspicion, Doppler and arteriographic examinations complete the diagnosis of API.
There are certain situations when clinical diagnosis of ischaemia is difficult at the first examination and this study presents two of them: the first – when systolic blood pressure is lower than 60 mm Hg and the peripheral pulse is absent; the second – when API onset is after an interval (hours) following trauma.
In the first situation, clinical reevaluation of the peripheral pulse will be performed after haemodynamical stabilization, in order to determine the real vascular status of the limbs. In the second situation, there is a certain period of time, called "free time", between trauma and API onset; in these cases, arterial thrombosis following arterial contusion can be the cause of ischaemia.
In all the cases, surgery was performed immediately after API diagnosis, in order to identify and treat the complex injuries (bone and vascular).

Key words
acute peripheral ischaemia, closed fracture, reevaluation.

Introduction

Modern traumatology is characterized by higher energy trauma agents, like high-speed vehicles (generating more severe road traffic accidents), high velocity missiles, complex technical procedures using inflammable materials, generating severe work accidents. When interacting with the victims, all these produce severe injuries, because the energy of the trauma agent is almost interely transferred to the affected tissue (except a very small amount, lost due to friction), producing different amounts of cellular and tissular disruptions. When acting on a part of one limb, traumatic forces are transferred to its' structures, often producing a fracture, but high energy agents generate complex injuries: bone, muscular, vascular and nervous injury.

The proximity of the bones to the blood vessels of the limbs often generates vascular complications, which are so complex that a unique classification is difficult to be established, as many criteria are to be discussed: the type of the vessels affected (arteries, veins, or both), the size of the vessels (large, medium or small), the main effect of the injury (ischaemia or hemorrhage) the amount of the vascular wall and diameter affected, the local consequences (functional and structural damages of the tissues produced by the vascular injury), systemic impact of the vascular damage (by itself and by local metabolic changes).

Considered to be the most severe of all the vascular complications of trauma, arterial injuries have both local and general consequences due to **A**cute **P**eripheral **I**schaemia (API), which can be defined as the syndrome consisting of metabolic disturbances, followed by structural changes of the affected tissues, with clinical correspondence, due to the imbalance between the local need of blood and the amount of oxygenated blood supply, diminished after arterial injuries. [1]

It is known that acute ischaemia affects all the tissues, depending on their sensitivity to hypoxia (which is the main consequence of ischaemia), so the moment when the hypoxic injury of different tissues becomes clinically detectable depends on the metabolism of each tissue, and especially on the oxygen dependence of that metabolism (i.e., the more oxygen is involved in the metabolism of a certain tissue, the earlier that tissue becomes necrotic following acute ischaemia. [2]

The importance of acute ischaemia is not only local, but it soon becomes systemic, as necrotic tissues, especially the muscles, generates anaerobic metabolic products which produce liver and kidney failure. [3] That is why modern scoring systems of limb trauma refer not only to the fracture, but also to the vascular status following trauma, and one good example is the

well-known MESS Score, which evaluates the prognosis of an ischaemic limb, concerning both local and systemic factors: the age of the patient, signs of API (including the DURATA), signs of shock and the energy of traumatic agent. [4]

Mangled Extremity Severity Score (MEES):

Category	
A. Skeletal/soft – tissue injury	
Low energy (stab, simple fracture, low velocity gunshot wound)	1
Medium energy (open or multiple fractures, dislocation)	2
High energy (close range shotgun, high velocity gunshot, crush)	3
Very high energy (above end gross contamination, soft tissue avulsion)	4
B. Limb ischemic	
Pulse reduced or absent but perfusion normal	1
Pulseless, paresthesias, diminished capillary refill	2
Cool, paralyzed, insensate, numb	
C. Shock	
Systolic blood pressure always over 90 mm Hg	0
Hypotensive transiently	1
Persistent hipotension	2
D. Age (YEARS)	
<30	0
30-50	1
>50	2

Score doubles for ischemia > 6 hours

A total MEES score of 7 or greater is predictive of amputation

Literature shows that API can complicate most of the skeleton trauma, that it can occur simultaneously with the skeleton injury, or after a certain period of time, called "free interval", after open fractures (type III C in Gustillo-Andersen classification), and closed fractures. The fact that the open fracture needs immediate surgical debridement avoids the situation of not-diagnosing API, since the vessels are examined intra-operative. When the fracture is closed, it is very important for the trauma surgeon to perform a thorough clinical examination of the injured limb, especially after certain types of fracture, when he must "expect" arterial damage.

As the correct treatment of arterial injuries, concerning the moment and the type of intervention, is vital not only for the limb, but also for the patient's life, early diagnosis of API is absolutely necessary, and this is performed primarily by clinical examination, which shows disappearance (or decrease, compared to the contralateral limb) of the peripheral pulse. After that, Doppler and arteriographic examinations shows precisely the place and gives informations about the type of injury. [5], [6], [7]

Since peripheral pulse is the major indicator for the arterial status of the limb, it is also the most important factor used in clinical diagnosis of API. It is palpable only if systolic blood pressure exceeds 60 mm Hg, when peripheral pulse will be palpable at the other limbs except the ischaemic one. If systolic blood pressure is under 60 mm Hg, peripheral pulse will not be palpable at any of the limbs, so API diagnosis is impossible until haemodynamic stabilization.

The purpose of the study is not to describe the treatment of closed fractures associated with arterial injuries, but to present two major problems concerning diagnosis of API complicating closed fractures, when API is not detected at the first clinical examination: the situations when systolic blood pressure is under 60 mm Hg (acute post-traumatic hypotension) and peripheral pulse is not palpable as well as those when there is a "free interval" between trauma and API onset.

Material and Method

Fifty-four patients were operated in Orthopedics and Trauma Clinic of Emergency Hospital Bucharest between 1.01.1998 and 1.01.2002 for API (complicating closed fractures) which could not be diagnosed by primary examination, but some time after the first examination. None of the patients had any arterial pathology before trauma (revealed by the patients, by their families or accordding with the clinical findings.)

The patients were studied concerning the type of the fracture, the reason for delayed diagnosis of API, the moment of API diagnosis and the arterial injury.

Using the AO classification, the types of the fractures were: twenty femural shaft fractures, sixteen fractures of distal femur, ten tibial fractures, and eight humeral fractures.

In all fifty-four cases, API was not detected at the first examination, but after some time, for two main reasons, so the patients were divided into the following two groups:

Group A – thirty-six cases, mean age 42,6 yrs (twenty-six to sixty-six yrs), with systolic blood pressure less than 60 mm Hg at first examination. The patients in this group did not have palpable pulse at any of the peripheral arteries when they were first examined. After haemodynamic stabilization, peripheral pulse became palpable except on the distal part of the affected limb, and the mean time between first examination and the moment when API was diagnosed was sixteen minutes.

Group B – eighteen cases, mean age thirty-two yrs (fourteen to fifty-six yrs), with bilateral peripheral pulse

palpable on the first examination at upper and lower limbs; after a certain period of time – mean value fourteen hrs (twelve to eighteen hrs) – API was clinically diagnosed at one of the limbs, as peripheral pulse was no longer detectable and the signs of ischaemia also appeared.

In all these cases, when API was diagnosed, arteriography was performed, and afterwards surgical treatment confirmed the arterial injury, described it precisely and repaired the associated injuries. Surgery consisted of the following standard procedures: bone stabilization, venous drainage restoration for humeral and femural veins, major arterial axis restoration, associated injuries of muscles, nerves, tendons treatment, and decompressive fasciotomy, for reperffusion syndrome prophylaxis.

The patients from the two groups were studied for the type of the fracture, the moment of API diagnosis, and the arterial injury producing API.

Results

The fractures in group A were: twelve femural shaft fractures, ten distal femural fractures, eight tibial fractures and six humeral fractures.

The time between the first examination and the moment when API was first diagnosed had an average value of sixteen minutes. This time was less than fifteen minutes (above ten minutes) in twenty-four cases, when acute hypotension produced by trauma was corrected by oxygen, intravenous fluid and drug administration, without requiring any surgical procedure. The time was longer than fifteen minutes (not exceeding thirty-three minutes) in twelve cases when hypotension was produced by associated injuries, which needed surgical treatment: eight ruptured spleen, which needed splenectomy, three liver injuries, which needed hepatorhaphy, one mesenteric desinsertion, with haemoperitoneum, which required resection and haemostasis.

The arterial injuries in group A, suggested by arteriography and precisely described intra-operatory, were: arterial section – seventeen cases (complete – twelve cases, incomplete – five cases), injury of internal and medium layer, with intact external layer – eight cases, thrombosis – three cases, compression and spasm – eight cases, and incomplete section with subsequent thrombosis- five cases.

Group B – eighteen cases, mean age thirty-two yrs (fourteen to fifty-six yrs), with bilateral peripheral pulse palpable on the first examination at upper and lower limbs; after a certain period of time – mean value fourteen hrs (twelve to eighteen hrs) – API was clinically diagnosed at one of the limbs, as peripheral pulse was no longer detectable, and signs of ischaemia also appeared: pallor, pain and decreased cutaneous temperature in all the eighteen cases, and paresthaesia in two cases. The signs that alerted the patients, who required repeated orthopaedical examination (which detected the absence of peripheral pulse) were: the sensation of "cold fingers" of the fractured limb – eighteen cases, pain – six cases, paresthaesia – twelve cases. Since API was diagnosed in early stages, none of the neurological signs of irreversible ischaemia (paralysis or anesthesia) appeared.

The fractures in group B were: eight femural shaft fractures, six distal femural fractures, two tibial fractures and two humeral fractures.

The arterial injury in group B (detected intra-operatory) was arterial thrombosis in all eighteen cases. In two cases minimal lateral injuries of the artery (a longitudinal 2 mm injury in one case and a round lateral injury with 3 mm in diameter) with subsequent thrombosis were detected; in these cases, the haematoma surrounding the artery had a medium size and acted as a compressive haemostatic mechanism.

Discussion

Arterial injuries complicating fractures are situations quite often described in literature and seen in trauma services, since agents with high traumatic force transfer their energy to all the tissues in the involved region. As Acute Peripheral Ischaemia is a situation that needs immediate treatment, otherwise not only the affected part of the limb will be compromised, but sometimes the patient's life too, that is why early diagnosis of this condition is vital for the patient. [8]

The first diagnosis of API is a clinical one – the absence of the peripheral pulse is the major sign, detected by the physician, who has to evaluate the vascular status of the injured limb. There are certain situations when API cannot be diagnosed immediately after trauma, even if the arterial injury exists. Two of these situations are discussed in this study: the first refers to post-traumatic haemodynamic changes, which disturbs clinical evaluation of the vascular status. This evaluation concerns both the arterial and venous circulation – the arterial evaluation includes subjective elements, described by the patient, and objective elements – peripheral pulse, cutaneous temperature and color, capillary pulse, active movements. [9] If the subjective elements cannot be described by the patient due to his status, peripheral pulse is the only reliable element. As acute ischaemia is defined as an imbalance between the local needs and practical supply of oxygen determined by an arterial acute injury, peripheral pulse of the injured limb must be compared with the pulse of other limbs, in order to be sure that the pulse absence is not the consequence of a chronic disease, like arteritis.

If the peripheral pulse cannot be detected in any of the limbs due to acute post-traumatic hypotension,

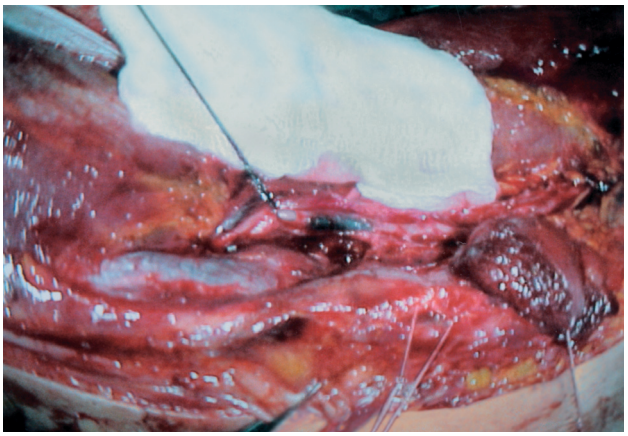


Fig. 1 C.V. 33 yrs
Thrombosis on femural artery after distal
femural fracture

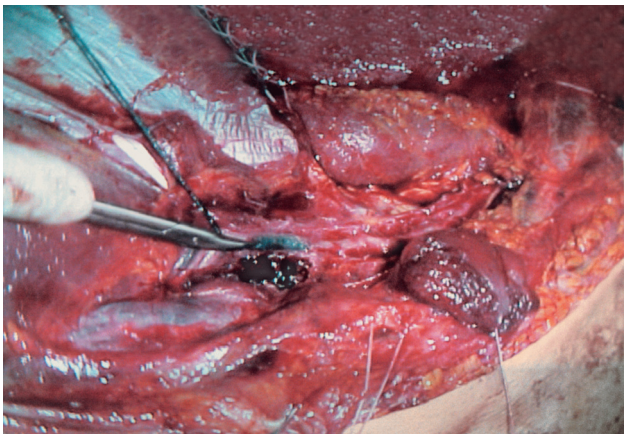


Fig. 2 C.V.-33 yrs
Thrombosis on femural artery after
distal femural fracture

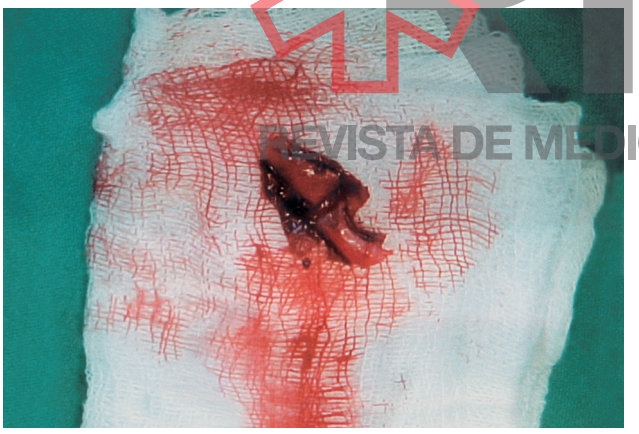


Fig. 3
Same case
Intact external layer, internal and
medium thrombosis injured,
intravascular thrombosis

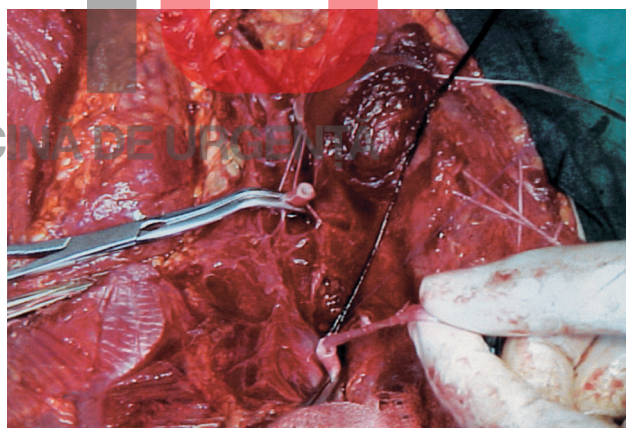


Fig. 4
Same case
resection of the injured artery,
arterioplasty with
inverted saphene graft

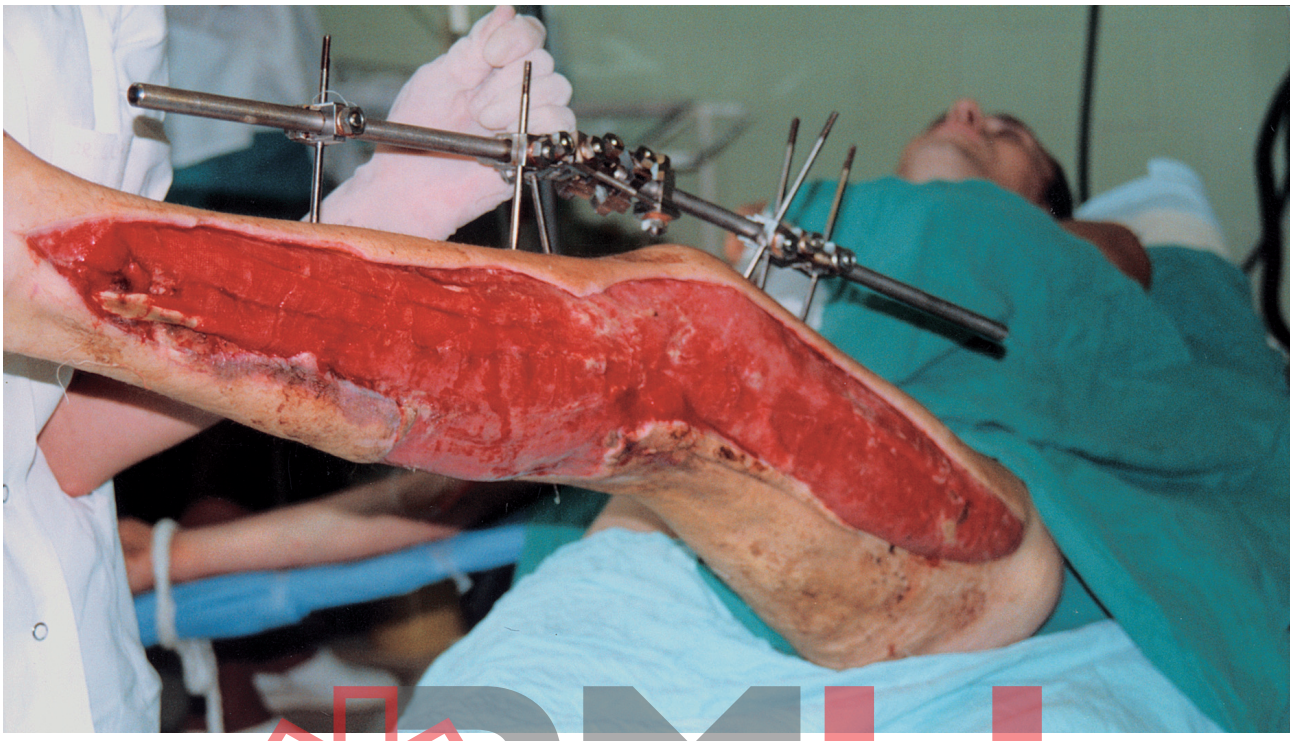
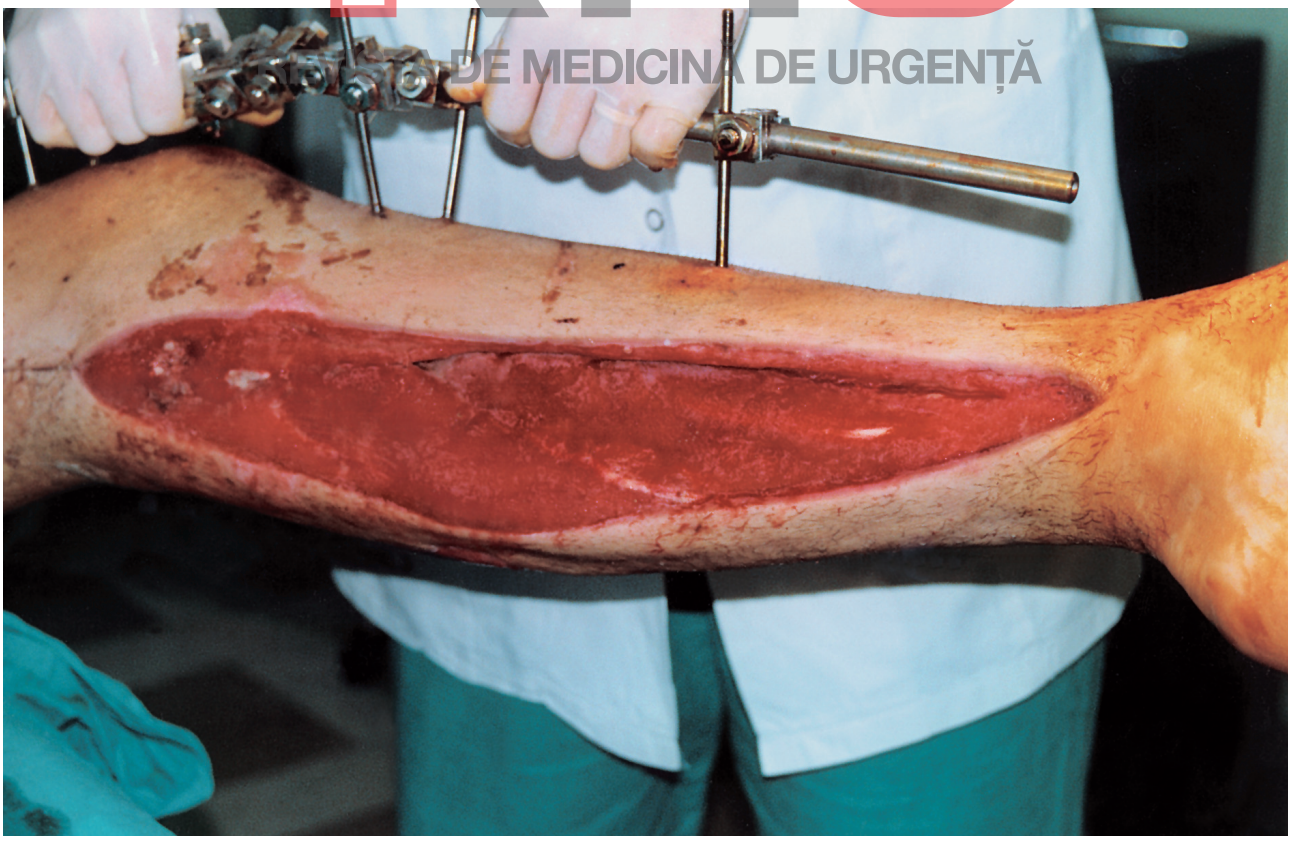


Fig. 5 Same case – External fasciotomy and external fixator

Fig. 6 Internal fasciotomy and external fixator – same case



it is impossible to diagnose API in one of the limbs until haemodynamic stabilization, which leads to the presence of the peripheral puls in all the limbs except the ischaemic one.

That means that when the patient's blood pressure is critically low, and the peripheral pulse is absent in all the limbs, clinical examination of the injured limb must be repeated until the patient is stable and the pulse appears, in order to ensure that arterial damage has not occured near the fracture. If the pulse is symmetrical at the upper and lower limbs, it means that there is no disturbance of the peripheral arterial flow in that moment.

If the pulse is absent in one limb, that limb is ischaemic and we have to find out whether the ischaemia is due to recent trauma or preceded trauma. If that limb has a fracture and the clinical examination suggests that the arterial injury level is at the site of the fracture, the probability of direct fracture – arterial damage corelation is very high and it must be considered at once. An arteriographic examination is recommended and if, hazardously, this is not possible, immediate surgery must be performed [11], [12], [13]. The possibility of ischaemia existing before trauma is very low, and this can be suggested by an obvious difference at the inspection of the ischaemic limb, characteristic for chronic injuries (atrophy of the muscles, skin, hairness, nails, or even signs of gangrene). This situation is quite rare, but it must be considered and if the patient cannot provide any useful information for a differential diagnosis, arteriographic examination may complete the clinical findings. Nethertheless, whenever the suspicion of acute injury exists, even on an arteritic background, surgical evaluation will clear up the diagnosis.

The second situation this study refers to is represented by the cases when there is a certain period usually called "free interval" between the trauma and the API onset (a few hours).

In our study, the arterial injury in the cases with a late onset of API was the arterial thrombosis. Literature presents post-traumatic arterial thrombosis as a very serious complication, as it occurs late after trauma, the artery looking intact, but the mechanism is a microscopic one: the initial trauma is only a contusion which does not disrupt the arterial wall, but produces thrombosis or disruption of vasa vasorum – generateing ischaemia of the arterial wall. The most sensitive structure to ischaemia (of the entire arterial wall) is the internal layer (the intima). Subsequently to the initial trauma, the intimal layer is injured both directly (mechanical) and indirectly, through ischaemia of the wall. The injury of the intima damages the connections between the endothelial cells, so the endothelium looses its continuity from place to place, exposing the subendothelial collagen fibers to the blood flow, which generates conditions for blood cells adherence, leeding to thrombosis. [14], [15], [16]

In these cases, ischaemia is frequently diagnosed after "alarm signs" are reported by the patient to the physician, who performs a new examination, suspecting an ischaemia. The earlier the ischaemia is diagnosed, the better the prognosis of the patient will be. A critical matter is represented by the situation when the patient is not conscious and he cannot alarm the physician. In this situation, the only possibility of an early diagnosis of API is the repeated vascular evaluation, especially when the fracture has a certain location and features. We can use the term "dangerous environments" for the locations most likely to determine arterial complications of the fracture. In such locations, the proximity of bone to arterial vessels make them sensitive to fracture displacement and post-traumatic injury.

Conclusions

We underline the importance of suspecting acute post-traumatic ischaemia in a fractured limb and the necessity of confirming or excluding it firmly, by clinical and paraclinical examinations [17].

Certain locations can be considered "dangerous environments" and fractures occuring in these locations will be considered as potential arterial-generating injuries and must be carefully monitored. The key word in these cases is reevaluation.

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