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CASE REPORT

SOFT-TISSUE AND BONE LOCAL REACTION TO ORTHOPAEDIC IMPLANT - CASE PRESENTATION

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Abstract. Orthopaedic implants are used at a large scale both for trauma and elective surgery. The components of the implant undergo corrosion and produce metal debris, which can trigger an immune response, depending on the amount and duration of the exposure, which can interfere with the normal healing mechanism, generating local complications. We present the case of a patient who suffered a distal femoral fracture, initially operated with a Dynamic Condylar Screw. Five months after the operation, the fracture did not heal, with an obvious persistent fracture line and malalignment of the fragments, so revision surgery was indicated. Intra-operative evaluation revealed local reactive lesions; and the histological exam showed chronic inflammation with many histiocytes, loaded with metal pigment, multinucleate giant cells and bone lamella evolving into sequestra, therefore confirming that the local reaction to implant contributed to the healing disturbance.

Keywords: orthopaedic implants, tissue reaction, implant reaction

Introduction

Orthopaedic implants are used at a large scale for fixing fractures and for replacement of arthritic joints. The main alloys used in production of these implants are stainless steel, titanium and cobalt, but the components may also contain polymers and ceramics.

Two mechanisms are basically responsible for tissular reaction to implant: the first is related to cell-mediated hypersensitivity to one of the materials of the implant- T lymphocytes form immunological memory after contact with metal debris generated by corrosion or wear and after a second contact with the same antigen, an inflammatory response is triggered. Continuous exposure creates a chronic inflammatory state with local clinical symptomatology and microscopic lesions. The second mechanism involves the local immune mechanisms, by activating B and T lymphocytes, thus generating a lymphoplasmacytic chronic inflammation. Both these mechanisms finally produce their effect through chronic inflammation, with differences regarding the cellularity, but they are most frequently associated. [1,2] Furthermore, chronic inflammation and metal debris inhibit osteoblasts and activates osteoclasts, which induce osteolysis and bone resorption, producing aseptic loosening of the implant up to failure. [3]

That is why patients must be thoroughly and individually analyzed for finding any abnormal elements that could act like a catalyzer for the local inflammation. Initial stability of the implant and local characteristics of the bone must be evaluated, and care must be taken for minimizing the surgical aggression, so as not to enhance the initial post-traumatic inflammation. Stability is very important, since an unstable or a loosen implant, with micro movements wears faster, resulting in inflammation and metal corrosion which can lead to accelerated hypersensitivity reactions and bone osteolysis. Osteolysis further decreases the stability of the implant, so a vicious circle will finally lead to fixation failure.

Clinical case

The patient was admitted in our hospital (Clinical Emergency Hospital Bucharest, Orthopaedic and Trauma Clinic) five months after a traffic accident, with left acetabular fracture and right distal femoral fracture.

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The patient was initially operated in another hospital for the femoral fracture, while orthpaedic treatment was performed for the acetabular fracture. At admittance in our hospital, clinical evaluation of the lower right limb revealed: post-op scar on the external aspect of the thigh and knee; severe pain with knee stiffness severely limiting walking, muscular atrophy of the thigh and shank; the pain was almost continuous at the level of distal right thigh and knee, without any response to local or general anti-inflammatory or analgesic drugs.

X-Ray exam showed a comminuted supra-condylar fracture of the right femur, operated with DCS (Dynamic Condylar Screw), with persistent fracture line and no tendency to healing. Analyzing the position of the DCS, (Figure1a) there is an abnormal position of the distal part of the implant (Figure 1b- yellow line), which does not follow the condylar line (Figure 1b- red line); this aspect disappeared in external rotation (Figure 1c), in which the condylar line follows the plate line, demonstrating that there was a malrotation of the distal fragment. At the same time, the axis of the diaphysis does not continue the axis of the distal fragment (Figure 1c), so the displacement of the distal fragment is valgus and internal rotation.

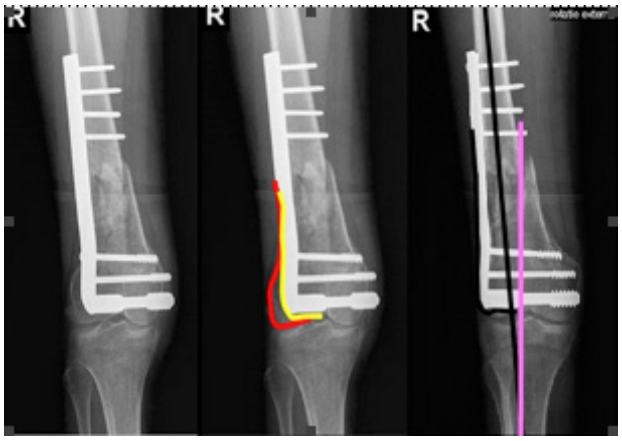


Fig.1. a) Left - comminuted supracondylar fracture of the right femur, operated with DCS; b) Center -Yellow line shows the contour of the DCS plate, which does not follow the condylar line depicted in red; c) Right - The right thigh is in external rotation. Black line shows the axis of the femur, which is different from the axis of the distal fragment (purple line). Black line that follows the DCS plate margin also follows the margin of the condyle, the distal fragment is in internal rotation.

A CT scan was performed and analyzed in order to evaluate the relative positions of the diaphysis and distal metaphysis; in Figure 2b the condylar fragment is figured in red, so as its sagittal axis; in Figure 2b, the same landmarks for the diaphysis are represented in black. Comparing these two figures, the internal rotation is obvious, with 15 degrees. Due to the fact that this malposition has considerable effect upon the local functional anatomy, revision surgery was indicated.

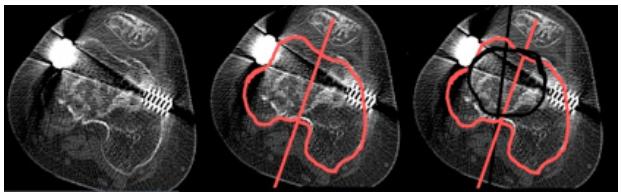
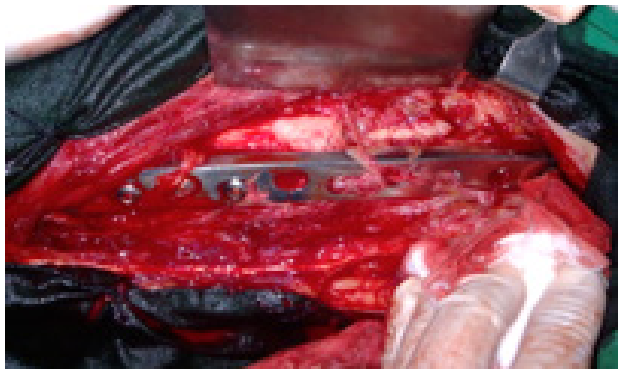


Fig.2. a) Left - CT coronal plane through distal screw of the plate; b) Center - Condylar fragment margins are depicted with red, red line represents the sagittal axis of this fragment; c) Right - black circle shows the diaphysis of the femur, black line shows the axis of the diaphysis. The angle between the axes of the two fragments is 15 degrees and represents the internal rotation of the distal fragment.

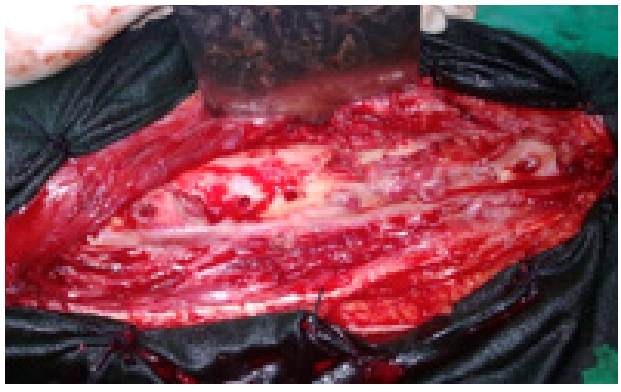
Intra-operative aspect (Figure 3) before implant removal reveals:

- fibrous membrane around the implant
- reactive tissues in the non-occupied wholes of the plate
- free space between the main fragments, with abnormal movement.

The implant was removed (Figure 3b); the fibrous membrane became obvious, and also the reactive tissue, which was removed and analyzed histologically: fibroconnective membrane which surrounds bone lamella with different lesions, from dystrophic lesions to bone sequestra and granulomatous reaction with giant cells and histiocytes with metal pigment in their cytoplasm.



(a)



(b)

Fig.3. Local aspect of the peri-implant tissues, fibrous membrane and reactive tissue around the implant and free space between fragments (a); local aspect after the implant was removed, the free holes of the plate have been filled with fibrous tissue (b)

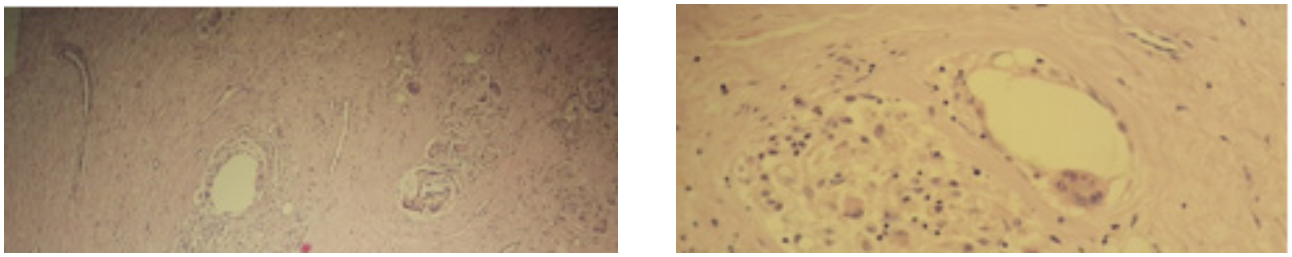


Fig.4. Histiocytes collections with granulations in their cytoplasm. Mononuclear inflammatory reaction (lymphocytes). H-E, X10 (a) and granuloma reaction with multinucleated giant cell, HE, X40 (b).

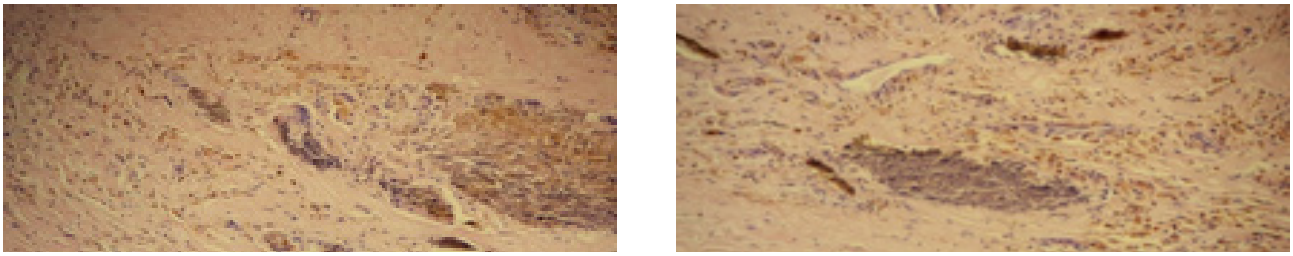


Fig.5. Histiocytic collections and isolated histiocytes loaded with brown and dark brown pigment resulted from the phagocytosis of the implant debris (a) HE, X10; Different magnification (b) HE, X40

Fig.6. Bone sequestra in resorption (purple) with multinucleated giant cells and histiocytes in center. HE, X10 (a) and different magniffication HE, X40 (b)

The result of this analysis confirmed the chronic inflammatory reaction to the implant, which was supposed to have contributed to the non-union of the fracture.

The treatment consisted of: debridement with excision of the fibrous tissue and avascular structure, cleaning of the free parts of the fragments, so as to ensure the fracture site with vascularized tissues, proper reduction, so as to assure the normal angles and rotation of the distal fragment and stabilization with angular stability plate after grafting with cancellous autograft taken from proximal tibial metaphysis. The general treatement consisted in antibio-prophylaxis and anti-thrombotic; active movements of the knee were started after acute pain remission and full weight bearing was allowed three months after revision surgery

The clinical and radiological outcome of the patient was favorable, with bone healing and knee recovery.

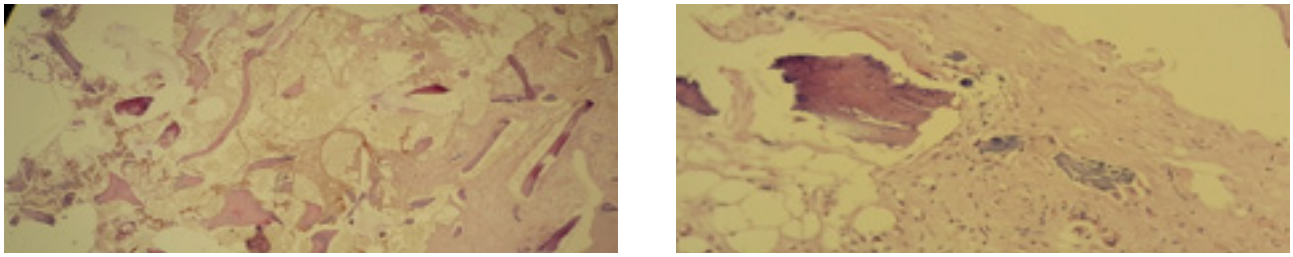


Fig.7. Bone lamellae in different degrees of involution. Pink: dystrophic lesions, Purple: bone sequestra. HE, 10X (a) and HE, X40 (b)

Discussions - was the foreign body reaction the cause of non-union?

Although the malposition was obvious, it could hardly be considered the only cause of non-union, let alone the fact that the inflammatory response was histologically proven, thus increasing the possibility that a direct correlation between poor bone healing and implant reaction had existed.

The macroscopic and histologic findings of tissue around the implant reveal a tissular reaction with metal pigment staining, lymphoplasmacytic infiltration and bone lamellae in different evolution state, evolving to sequestrum. The reaction between the metal corrosion and debris on one hand and the host system on the other could have been a catalyzing factor for the nonunion of the fracture.

Although the whole mechanism is not understood, it is clear that osteolysis appears due to a complex host response triggered do to implant debris and corrosion products exposure. It is a multi-factorial process, depending on implant composition, surgical technique and patient factors. [3] Improper surgical technique can lead to poor reduction of the fracture, affecting the implant stability which leads to faster metal wear, and activation of an immune response, which in sensitized individuals can trigger accelerated bone resorption and further implant loosening. [6]

Normal bone turnover involves a balance between bone formation and bone resorption. [5] The osteoblasts, which are the promoters of bone formation, are disrupted by metal cytotoxicity induced by the debris. Osteoblasts undergo a time and dose dependent reduction in proliferation; also appear modifications in shape and size; thus the osteoblasts become able to release proinflammatory cytokines which activate the precursors of osteoclasts. Osteoclasts are highly specialized multinucleated cells that are responsible for lacunar bone resorption. [3,4]

Also phagocytosis of metal debris by macrophages stimulates the production of macrophage-colony stimulating factor, proinflammatory cytokines, and receptor activator of nuclear factor kappa B (RANK). which is expressed in osteoclast precursors and RANK ligand (RANKL), expressed by osteoblasts and several other cells.[3] Thus proresorptive conditions are created with metal debris stimulating macrophages and osteoblasts to produce chemical mediators that attract and activate osteoclasts and inhibit osteoblastic activity. All this leads to bone resorption around the implant, loosening of the implant, abnormal stability and implant failure.[3]

Another factor that could have led to this situation was the material in the first implant; stainless steel. There is a greater incidence of hypersensibility to stainless steel implants then there is to titanium implants. During the reintervention there was used a titanium implant.

Conclusions

All implants interact with tissues sourounding them; this reaction is influenced by the tipe of implant used, surgical technique, and the host imunologic sistem. It is currently unclear whether implant failure modifies the tissues surrounding the implant or if there are microscopically quantifiable alterations of the tissue before the implant fails, probably both situations occur simultaneously.

Every patient must be evaluated thoroughly, even if it is used a bio-compatible implant that has been used in other patients with success; the host immune system of every one is different and could have an exaggerated response to that metal alloy.

With an upscale of arthroplasties and post-traumatic orthopaedic interventions further research in bio-compatible materials is essential for the development of safer, more reliable implants.

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