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OSTEOLYTIC FORM OF TRAUMATIC INFLAMMATION OF THE BONE

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Observations of nine patients who clinically developed asymptomatically procressing traumatic inflammation of the bone. Its main symptom was advancing osteolysis.

In the previous work (2), we called attention to what role a bad internal stabilization of a fracture plays in the traumatic inflammation of the bone (pzk). We also ascertained that the course of the disease and its after-effects to a great degree depend on how the fracture is joined.

However, the method of joining the fracture is not the only factor forming the clinical picture of pzk. Next to internal stabilization which does not insure complete immobilization of fragments of fractures, the extent of soft-tissue destruction, blood supply restriction, location of the break, virulence of the germs, extent of the infection, immunobiological outbreaks, as well as antibiological treatment lacking potency, all play a vital role in the pathogenesis of pzk (1,2,4,5,6,7,11).

It can be assumed that the mutual, often accumulate, effects of the afore-mentioned factors to some extent qualify the probability of the development of an infectious complication in traumatological surgery, likewise forming a clinical picture of pzk between progressing very wildly, and festering forms in a manner at first chronic.

Following the outbreak of osseous adhesion occuring during the treatment of long bone fractures, we returned attention to the originally chronic, traumatic inflammation of the bone progressing practically without clinical symptoms. These observations are the object of the present report.

Discussion of Clinical Material.

We diagnosed the osteolytic form of traumatic bone inflammation in nine patients. After surgical intervention, only two patients suffered infection of soft-tissues, which we overcame using general and local antibiotics. There were no complications in the post-operative recovery of the remaining seven patients. The post-operative wound healed immediately. None of them had a fever immediately after the operation. Detailed data is contained in Table 1.

Table 1

Case No.	Personal data	Post-operative progress	Clinical symptoms OB*	Midoperative bacteriological exam	Probable cause	Second surical intervention of the fracture
1	K.W. 1. 31 multi-fragment fracture of the thigh	without complications	without symptoms 40/70	staphylococcus coagulation (+)	wilful premature burden of limb, oblique fragments	4 months

(continued on next page)

*[translator's note: Unable to expand this abbreviation]

Table 1 (continued)

Case No.	Personal data	Post-operative progress	Clinical symptoms OB	Midoperative bacteriological exam	Probable cause	Second surgical intervention of the fracture
2	M.Z. 1. 28 multi-fragment fracture of the thigh	without complications	3/7	staphylococcus coagulation (+)	middle fragment, screws too near fracture fissura	5 months
3	C.Z. 1. 28 multi-fragment fracture of the thigh	soft-tissue infection, effectively controlled by drainage	88/110 muscular atrophy, spurious joint	stercoraceous streptococcus, colon bacillus	oblique fragment, soft-tissue infection, wilful burdening	9 months
4	Sz. J 1. 10 multi-fragment fracture of the thigh	without complications	4/14	staphylococcus coagulation (+)	oblique fragment, wilful premature burdening of limb	12 weeks
5	U.A. 1. 19 multi-fragment fracture of the thigh	without complications	28/47	staphylococcus coagulation (+)	oblique fragment	6 months
6	L.J. 1. 9 multi-fragment fracture of the thigh	without complications	35/80	no data	oblique fragment	no data
7	W.H. 1. 38 multi-fragment fracture of the shoulder	temperature rise thru 2 weeks	spurious joint 35/60	staphylococcus coagulation (+)	oblique fragment poor stabilization, soft-tissue infection	3 months

(continued on next page)

Table 1 (continued)

Case No.	Personal data	Post-operative progress	Clinical symptoms OB	Midoperative bacteriological exam	Probable cause	Second surgical intervention of the fracture
8	M.Z. l. 42 multi-fragment fracture of the thigh	without complications	70/110 muscular atrophy	staphylococcus coagulation	premature burdening of the limb	3 months
9	M.Z. l. 49 multi-fragment fracture of the thigh	without complications	60/88 muscular atrophy	staphylococcus coagulation	premature burdening of the limb	3 months

It should be emphasized that all patients had similar type fractures. The presence of at least one oblique fragment was discovered which was most often in the shape of a butterfly wing. With one exception, the fractures were localized within the thigh (Fig. 1). The majority of the patients started arbitrarily and prematurely to put weight on the limb. There were no complaints at the check-up. None of them had a fever. Progressing osteolysis in the vicinity of the fracture (Fig. 2,3), practically without periosteal reaction, was the only symptom of the concealed disease in the patient which was proveable on consecutive X-ray pictures. We only detected muscular atrophy during the physical examination. Experimentally, we ascertained an increase of the sedimentation of blood cells. We observed, in the area encompassed by changes, the presence of granular tissue of the circumjacent joint and sometimes purulent centers in the immediate proximity of the bone with all patients during the second operation. The periosteum surrounding the uneven porous bone was usually swollen. As a rule, osteolytic loss was accomplished by fibering tissue granulation, tiny sequestrums and sometimes purulent marrow. Inflammatory changes of the bone were very often considerably more extensive than evident from an X-ray picture. The walls of

the loss barely bled.

Fig. 1. Case 1 (K.W.), Radiogram of the bone immediately after the accident.

Fig. 2. Case 1 (K.W.), Radiogram made one month after the union of the fracture.

Fig. 3. Case 1 (K.W.), Photo made 16 weeks after the fracture union. In comparison with the previous photo, the fissura of the fracture has succumbed to considerable widening, and the oblique fragments to partial osteolysis.

Recognizing that surgery is the primary treatment of this form of traumatic inflammation of the bone, we based our procedures on the principles published by Willenger, which are applied with little variation by other authors as well (7,8,12,13). During the second operation, we removed the joiners in cases where the correct adhesion was reached, in spite of the continuing infection process, or, when the joiners failed to stabilize both parts. For those patients with poor adhesion or whose osteolytic loss was too large, we left a plate to limit the twisting of screws remaining directly at or in close proximity to the loss. In each case, after using a reamer to clean the granulation and fibrous tissue from the walls of the loss and to

remove the slerotic layer of the bone, we packed it airtight with an autogenous spongy transplant. The surgical intervention was completed by establishing lavage drainage which was conducted according to accepted procedures in our Department (3).

We used plaster cast immobilization in those cases of lack of adhesion where there were also indications for the removal of the joiners. After local treatment, all patients experienced quick healing of the fistulas after removal of the drainage, also reconstruction of the transplant and consolidation of the adhesion quickly followed and the infectious process succumbed to cure (Fig. 4).

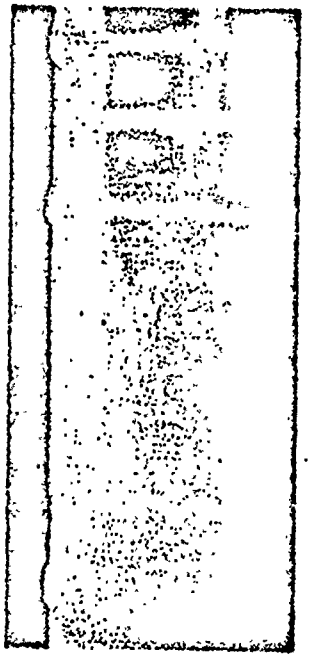

Fig. 4. Radiological image 8 weeks after the second surgical intervention. Complete reconstruction of the spongy transplantation without a radiological symptom of bone inflammation.

DISCUSSION

Detailed analysis of clinical material performed additionally with some of the patients *ex post* permitted a definate generalization. It can be accepted that the osteolytic form of traumatic inflammation of the bone has all the traits of a primary chronic infection of little virulence (10). It seems to us that the main cause of the development of this complication is the opportune tissue breeding ground

in the region of the fracture. Here we should note the presence of the following: small oblique bone fragments, premature wilful burdening of the limb, and incorrect stabilization of the sections in one case and fracture fissurae being too closely secured by screws in the other. All of these factors caused the occurrence of tissue with a poor blood supply which was located in the area of the bony injury. We discovered only two patients with lingering infection which manifested itself after the realization of osteosynthesis. The postoperative progress of the remaining patients was not complicated and there was no basis for the diagnosis of infectious complications. It should be noted that the localization of the changes concerned those bones in which the risk of development of inflammatory complications is not great (1,4,7). We consider as well that in conditions threatening complications in traumatological surgery, the use of antibiotics, based on the proposals of Küntscher and Willengger (6,14), resulted in the fact that in none of these cases was the full manifestation of symptoms of traumatic inflammation of the bone reached. However, in the case of the existence of a suitable tissue breeding ground, the use of antibiotics proved insufficient for the complete suppression of the inflammation, but in the end it was effective as the inflammation process proceeded practically asymptomatically in all cases.

It appears that there is a great clinical and morphological resemblance of the osteolytic form of traumatic inflammation to the form of blood-derived bone inflammation described in Popkirov (8,9) which was defined by the author as *"osteomyelitis antibiotica"*.

Second to and independent from those pathogenetic factors (tissue breeding-ground, virulence of the microbes, or the destruction of the immunity of an organism) which played a crucial part here, is the healing procedure which must intend to check the vicious circle of the cause and effects of the morbid process. It is only possible to bring this about by surgical intervention which changes the conditions of the tissue breeding-ground and makes the complete destruction of the infection possible (7,8).

We believe that the second operation should be as soon as possible because waiting too long in the event of progressive osteolysis leads to the origin of losses which are hart to make up for afterwards and also to the cessation of the stabilizing activity of the joiners. The possibility also exists of the stimulation of the infectious process and the full manifestation of the pzk symptoms. First of all, it seems a surgical plan should be established to supplement the loss by autogenous spongy transplantation and the employment of cleansing drainage. Metal joiners should be used only in those cases when the fracture has ceased stabilizing or when the extreme adhesion of the fracture has been reached despite the progressing inflammation process (8,12,13). Rather, we save the joiners for the time when there is no adhesion or the adhesion is poor, and they can still fulfil their primary role of stabilizing the fracture.

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