Metabolic and Endocrine Disorders Associated With Pseudarthrosis

Presentation of a Clinical Case

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Bone fatigue is a considerable risk factor causing fractures in high-performance athletes, as a result of many extrinsic and intrinsic factors. This article describes a 13-year-old girl, a professional skater with a fracture of the femur and atrophic pseudarthrosis 10 months after initial surgical treatment. A metabolic disturbance was found at the biological medical consultation; this was managed holistically, and the patient's fracture healed after 2 months of antihomotoxic and integrative treatment.

The use of unsuitable equipment, very intensive training schedules, and inappropriate diets are among the external risk factors that predispose towards bone pathology in athletes. Age; mechanical biophysical factors arising from the bone-muscle relationship, which alter physiological alignment; bone density; and metabolic or hormonal imbalances are intrinsic causes of stress fractures and pseudarthrosis. Prepubertal girls and women, as a result of the physiological changes inherent to their sexual development and monthly hormonal fluctuation, are a population especially at risk.[1]

In 1986, the US Food and Drug Administration defined pseudarthrosis as nonhealing of a fracture 9 months after injury. However, depending on the bone and the site of the injury, this period may vary. In fractures of the long bones in the middle third of the femur, a waiting period of 6 months is allowed, whereas neck fractures should heal within 3 months after the trauma.[2]

Although the exact cause of pseudarthrosis is not clear, it is believed that local factors (e.g., infection and poor vascularization) and systemic factors (e.g., nutritional state and hormonal balance) contribute to nonhealing of fractures. Although there are opposing opinions, there is considerable bibliographic evidence implicating nonsteroidal anti-inflammatory drugs and corticoids as important factors in fractures that are not healing.[3]

Pseudarthrosis can be hypertrophic or hypervascularized and atrophic or avascular.

Figure 1: Fracture

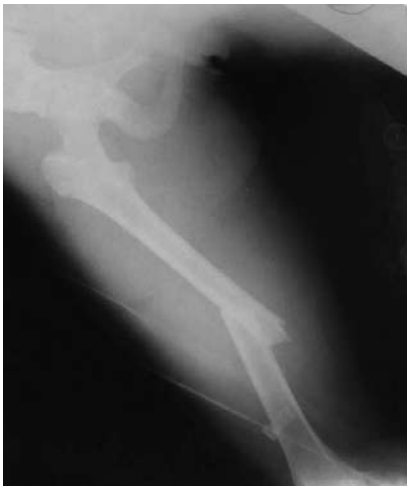


Figure 2: Intramedullary pin (June 26, 2004)

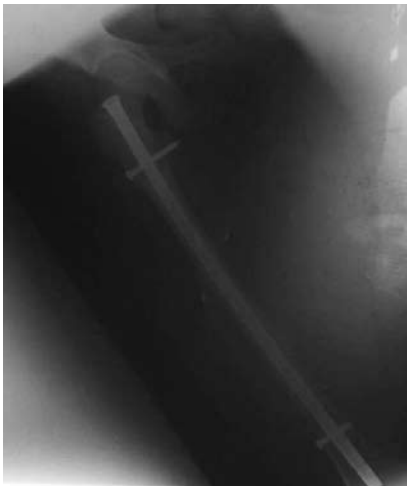
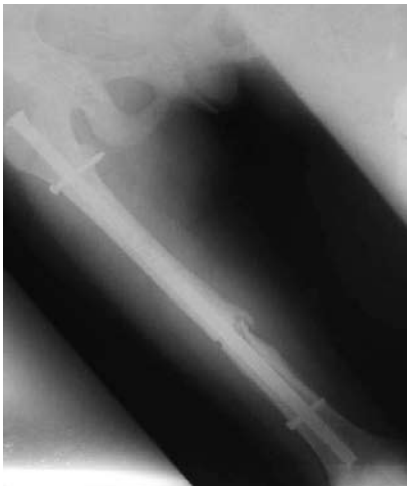


Figure 3: Pseudarthrosis at follow-up



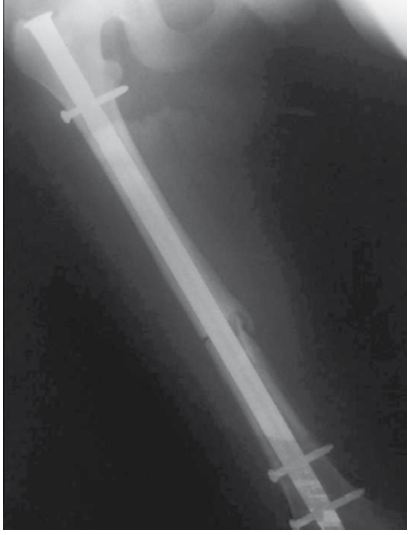


Figure 4: Second operative procedure

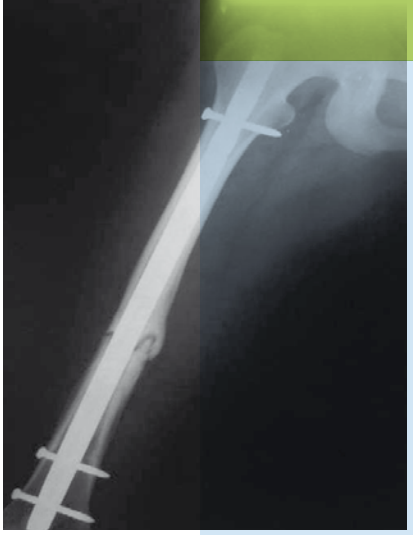


Figure 5: Postoperative view 5 months after the second operative procedure

Clinical case

The patient is a 13-year-old sportsperson who, on June 25, 2004, experienced a displaced fracture in the middle third of the right femur (Figure 1), which required surgical treatment with an intramedullary pin (Figure 2).

A 5-month postoperative follow-up X-ray showed pseudarthrosis (Figure 3). Thus, from an orthopedic viewpoint of the mechanical instability and hypertrophic pseudarthrosis, a further intervention changing the pin for one of a larger diameter with double distal locking was performed on November 17, 2004 (Figure 4).

Five months after the second operative procedure, the fracture was classified as atrophic pseudarthrosis (Figure 5), and the treating orthopedic surgeon proposed a third intervention. The patient decided to consult a biological medicine specialist to obtain a second opinion.

The consultation on April 13, 2005, showed that the patient was in pain, with no support from the lower right limb, and had a high consumption of nonsteroidal anti-inflammatory drugs.

The results of the Meridian Stress Assessment (developed by Reinhold Voll) were pancreatic and splenic dysfunction (Table 1); therefore, clinical laboratory tests were performed to complete the investigation (Table 2). These test results showed a state of hypercortisolism with a normal basal insulin level (no postprandial insulin test result was available). The postprandial glucose response at 30 minutes was normal; however, at 1 hour, it was very low. The thyrotropin level was in the normal range, the free thyroxine level was normal, and the triiodothyronine level was not obtained. The parathyroid hormone level was normal; the result of bone densitometry showed osteopenia.

Organ	Right side	Left side
Lymphatic deg.	48	46
Lung	54	46
Large intestine	34	56
Central nervous system deg.	42	46
Circulation	46	48
Allergy deg.	42	42
Parenchyma deg.	34	42
Endocrine	46	46
Heart	52	46
Small intestine	44	58
Pancreas	**18**	
Spleen		**16**
Liver	44	46
Joint deg.	46	36
Stomach	54	52
Fibroid deg.	58	48
Skin deg.	66	58
Fat deg.	56	58
Gallbladder	70	52
Kidney	54	52
Bladder	48	56
Uterus/prostate	48	54

Table 1:
*Meridian Stress Assessment results**

Laboratory test	Patient value	Reference value
Urinary cortisol, µg/24 h	**60.86**	**5-55**
Basal blood glucose, mg/dL	79	70-105
Postprandial blood glucose at 30 min, mg/dL	125	> 110
Postprandial blood glucose at 1 h, mg/dL	**74**	**120-170**
Postprandial blood glucose at 2 h, mg/dL	94	70-120
Thyrotropin, µUI/mL	2.40	0.35-5.50
Free thyroxine, ng/dL	1.06	0.93-1.70
Parathyroid hormone, pg/mL	31.3	11.0-79.5
Basal insulin, µU/mL	**5.02**	**2.60 -24.90**

Table 2:
Clinical laboratory results

* Normal values, 40-60; Irritation, 61-80; Inflammation, 81-100; Weakness, 31-39; Degeneration, < 30

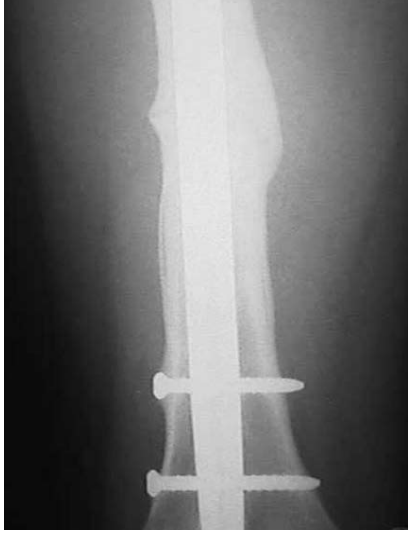


Figure 6: Consolidated fracture (June 23, 2005)

Treatment was started as follows:

- Osteoheel, 1 tablet 4 times per day
- Strumeel, 1 tablet 4 times per day
- Momordica compositum, 1 ampoule twice weekly, 10 doses
- Placenta compositum, 1 ampoule twice weekly, 10 doses
- Acidum citricum-Injeel, 1 ampoule twice weekly, 10 doses
- Lymphomyosot, 1 ampoule twice weekly, 10 doses

Nutritional changes reducing the intake of rapidly absorbed carbohydrates (refined sugars) and avoiding high-sodium processed foods (ready meals and fast food) were recommended.

At the 2-month clinical follow-up, pain was absent, normal electrical measurements of the pancreas (44) and spleen (48) were noted, and radiography showed healing of the fracture (Figure 6); therefore, the intramedullary pin was removed (Figure 7). Laboratory findings at the end of treatment were normal.

Discussion

According to the Meridian Stress Assessment, this patient had an abnormality of the pancreas. Her low glucose level, using the result of the oral glucose tolerance test at 60 minutes, indicates hypoglycemia and a state of chronic hypercortisolism.

This state of transitory hypoglycemia leads to a functional imbalance of the hypothalamus-pituitary-adrenal cortex axis[4-6]; therefore, the response is an increase in β-adrenergic activity in the hypothalamus, with the release of the growth hormones somatotrophin and corticotropin and increased secretion of cortisol and epinephrine.[5,6]

The cortisol acts like a counterregulating hormone and induces the production of glucose, activating the gluconeogenesis pathway. If the hypoglycemia persists, the level of cortisol rises, conforming a state of chronic hypercortisolism.

The increased cortisol levels in this patient could be secondary to the hypoglycemia and stress produced by competitive exercise and the influence of interleukin 6 as a chronic inflammatory cytokine.[7]

Intense exercise by high-performance athletes suppresses the function of the T cells and natural killer cells and increases the release of cortisol and interleukin 6 proinflammatory factors.[7]

Cortisol causes a reduction in bone formation and an increase in resorption by various mechanisms (Figure 8).[8]

Cortisol antagonizes the action of 1,25-dihydroxyvitamin D_3 or calcitriol, which acts on the osteoblast by increasing the synthesis of tissue growth factor β (TGF-β) and raising the number of insulinlike growth factor receptors, whose anabolic effect regulates bone growth and tissue repair.[8-12] Vitamin D_3 increases the synthesis of osteocalcin and osteopontin by improving the mineralization of the collagen fibrils of the bone when they are depleted.[9-12]

The formation of hydroxyapatite alters with sodium/calcium interchange in the renal distal tubules, where phosphorus and magnesium are also lost. Each gram of sodium ion in urine corresponds to 26.3 mg of lost calcium; therefore, salty and fast food diets are not recommended.[13]

Ingesting oily seeds and extra virgin vegetable oils rich in polyunsaturated fatty acids and conjugated linoleic acid increases the absorption rate of calcium in the cells and reduces osteoclastogenesis.[14]

Acidification secondary to the ingestion of refined sugar and proteins with sulfur atoms (methionine and cysteine) alters the mineralization and metabolism of the bone.[9]

The concentration of protons in the plasma and in the extracellular fluid is about 40 nM, corresponding to a pH of 7.4; to stabilize and alkalize this, there are systems that include balancing phosphate with calcium and magnesium ions originating from the bone matrix at the expense of weakening the bone.[9]

According to the personal analysis that I have made of this clinical case,

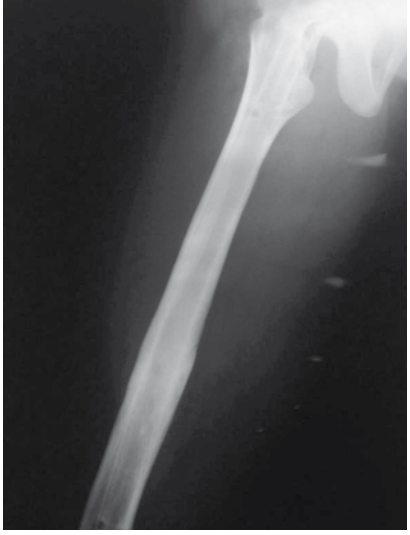
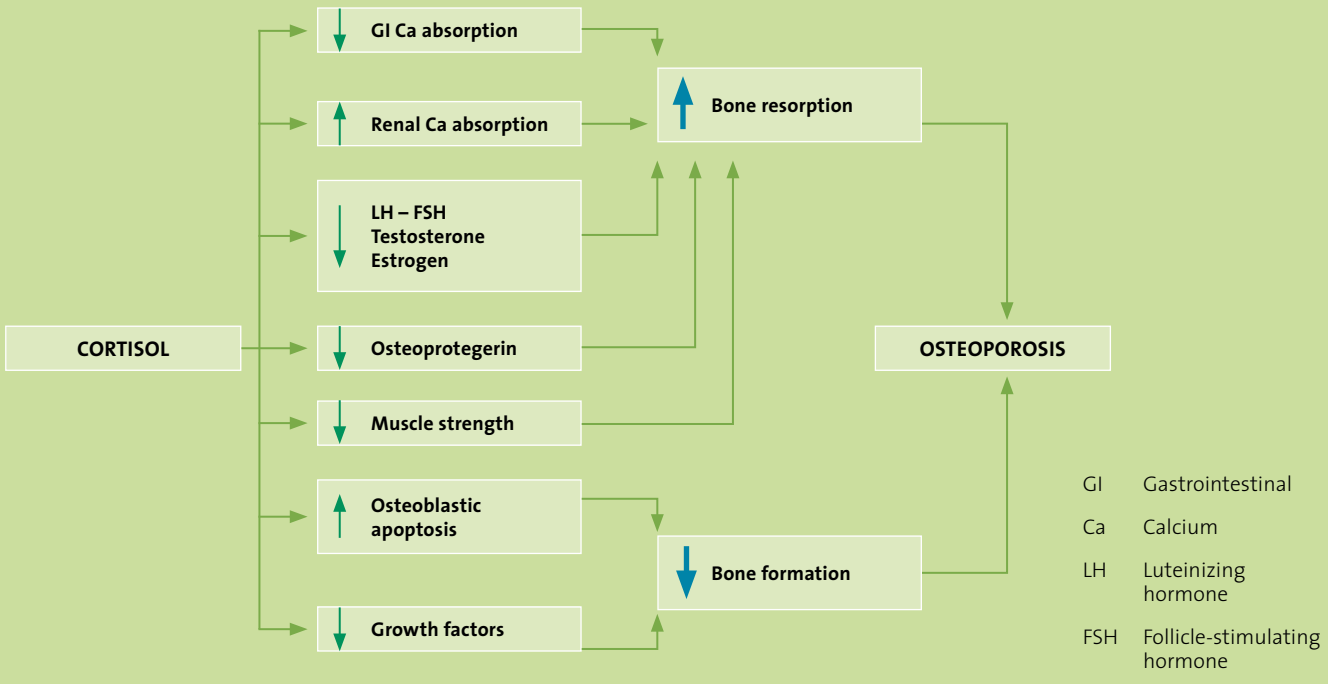


Figure 7: Fracture without intramedullary pin (November 28, 2006)

antihomotoxic medications could hypothetically have acted in the following manner in healing the fracture:

1. Antihomotoxic medications, which contain low doses of antigens, could have stimulated the production of TGF-β from the lymphocyte line T-helper cell 3. This TGF-β intervenes in the reconstruction of the bone matrix by inhibiting the activation of the osteoclasts and stimulating the action of the osteoblasts, promoting the healing of the tissue and the resolution of the inflammation.[15-18]
2. The bioregulatory effect of Momordica compositum in the pancreas in controlling hypoglycemia and secondary hypercortisolism could be the result of a possible improvement in the expression of glucotransporters in the cells and hypothetically might increase the secretion of amylin and preptin. These 2 polypeptides are cosecreted with insulin from the β cells of the pancreas; their function is to stimulate osteoblastic proliferation, reduce osteoblastic apoptosis, and inhibit osteoclastic activity.[19-25]
3. Possibly, Acidum citricum-Injeel, a Krebs cycle catalyst and calcium metabolism regulator that improves the absorption of vitamin D, could act in the renal tubule cells by stimulating the mitochondrial 1α-hydroxylase responsible for transforming 25-hydroxycholecalciferol (inactive) into 1,25-dihydroxycholecalciferol (active) or calcitriol.

Figure 8: Effects of cortisol on bone[8]



Conclusion

Antihomotoxic treatment drains the matrix (Lymphomyosot), regulates the endocrine function of the pancreas (Momordica compositum), regulates thyroid function (Strumeel), solves the problem of avascular atrophic pseudarthrosis (Placenta compositum), and re-establishes the metabolic balance of bone, the intrinsic calcium metabolism, and vitamin D absorption (Osteoheel and Acidum citricum-Injeel).

Pseudarthrosis is not an exclusively mechanical problem. It must be confronted integrally, from the profession or lifestyle to the metabolism of the organism, the diet, the neuroendocrine system, and immunological modulation. "The whole organism suffers with the fracture of a long bone."[26]

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