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Laser-Needle Therapy for Spontaneous Osteonecrosis of the Knee

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Abstract

Objective: This case report describes the treatment of a 63-year-old patient with spontaneous osteonecrosis of the knee (SONK). ***Background Data:*** SONK usually appears in the elderly patient without the typical risk factors for osteonecrosis. It is characterized by acute and sudden pain, mostly occurring at the medial side of the knee joint. Symptoms usually worsen with physical activity and improve with rest. Besides physical therapy, limited weight-bearing and the use of analgesics and nonsteroidal anti-inflammatory drugs, we propose low-level laser therapy (LLLT) as a conservative treatment option. ***Methods:*** LLLT was carried out using laser needles emitting radiation with wavelengths of 685 and 885 nm, and a power density of 17.8 W/cm^2. Therapy sessions lasted 60 min and were performed daily over a period of 3 mo. The total irradiation dose emitted by 8 laser needles in 60 min of treatment was 1008 J. ***Results:*** Magnetic resonance imaging revealed distinct restitution of the spongiosa edema 5 wk after treatment onset, and the final check-up at 35 wk demonstrated complete restoration of integrity. ***Conclusion:*** The present case report provides the first indication that laser-needle therapy may be a promising tool for complementary and alternative therapeutic intervention for those with SONK.

Introduction

Osteonecrosis of the knee was first described by Ahlbäck and colleagues in 1968, and has been classified into two distinct types: (1) spontaneous or idiopathic osteonecrosis, and (2) secondary osteonecrosis associated with various risk factors such as steroid therapy, renal transplantation, systemic lupus erythematosus (SLE), alcohol abuse, caisson decompression sickness, Gaucher's disease, and hemoglobinopathies.[1,2] Spontaneous osteonecrosis of the knee (SONK) usually appears in the elderly patient over 55 years of age without the typical risk factors for osteonecrosis, with an age-related prevalence between 3.4% and 9.4%.[3] Women are three times more often affected than men. SONK is characterized by acute and sudden pain, mostly occurring at the medial side of the knee joint. Symptoms usually worsen with physical activity and improve with rest. Also, nocturnal pain is frequently observed, and clinical examination shows local hypersensitivity to pressure.[4] Even though the precise etiology still remains unclear, two major theories have been proposed.[5] The traumatic theory suggests that repeated microtraumata in porotic bone cause stress fractures and successive necrosis.[6] According to the vascular theory, occlusion of the blood supply at the arterial and venous side may lead to decreased bone microcirculation with subsequent edema formation. Edema increases bone marrow pressure, further diminishing the blood supply and resulting in osseus ischemia and necrosis.[4] Furthermore, elevated bone marrow pressure due to increased fat cell size and fat microemboli has been suggested to impair intraosseus microcirculation.[7] Established treatment options comprise physical therapy, limited weight-bearing, and the use of analgesics and nonsteroidal anti-inflammatory drugs. These conservative approaches are recommended, usually in the early stages of disease. But even in such cases, progression can not always be successfully hindered, and patients with severe necrotic changes may require surgical intervention (e.g., high tibial osteotomy or total knee replacement).[2,8–10] In this context and in consideration of the basic research on the biostimulatory effects of low-level laser therapy (LLLT) on microcirculation and vascularization as well as on osteogenesis, and its clinical effectiveness in bone and joint diseases such as osteoarthritis and rheumatoid arthritis, we propose LLLT as a promising therapeutic option for patients with

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SONK.[11–16] However, clinical data on its effectiveness are currently lacking. For the first time, we describe the treatment of a 63-year-old patient with SONK using low-level laser irradiation.

Case Report

A 63-year-old man presented to sports medicine consultation with pain in the right medial femur radiating to the medial joint cavity. The complaints had first developed a year before, were aggravated by exercise, ceased spontaneously, then recurred during exercise on the treadmill 3 wk earlier. Up to that point the patient had worked out 1–2 h daily. In recent months, however, exercising was possible only with limitations. This otherwise healthy patient denied preceding trauma and had no history of diabetes mellitus or other metabolic disorders.

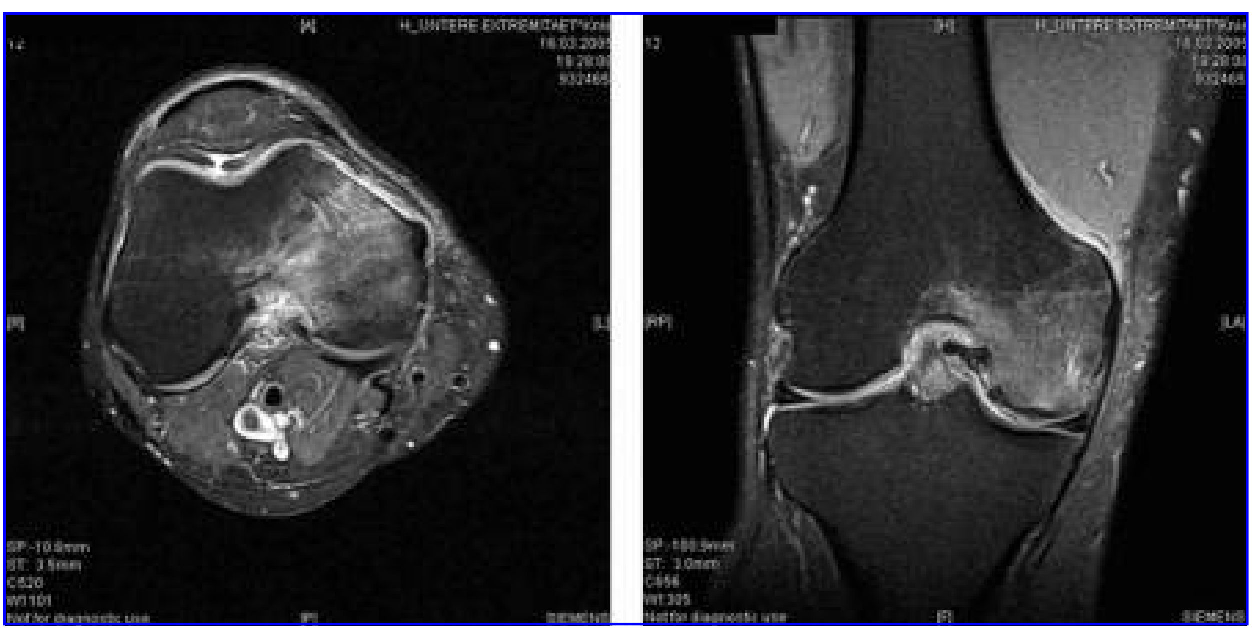
On clinical examination the medial distal femur of the knee joint was sensitive to pressure. The circumference measurement at the joint cavity revealed a left-to-right proportion of 36.5 cm to 35 cm. No other side discrepancies, swelling, or hyperthermia were apparent. All relevant functional tests of the knee were normal. With the exception of increased homocysteine values, all relevant laboratory values, including the rheumatoid factors, were unremarkable. Furthermore, predisposing factors associated with secondary osteonecrosis, such as long-term glucocorticoid therapy, renal transplantation, SLE, alcohol abuse, caisson decompression sickness, Gaucher's disease, and hemoglobinopathies could be excluded.

The magnetic resonance imaging (MRI) examination of the right knee joint performed 2 d later (on March 16, 2005) revealed Morbus Ahlbäck (spontaneous osteonecrosis of the knee, stage III) at the coronary fat-suppressed PD TSE sequence (Fig. 1). There was a linearly demarcated subcortical focus at the medial femur condyle with adjacent spongiosa edema (necrotic zone) reaching deep into the bone marrow. There was no osteochondritis dissecans. Furthermore, chondral irregularities were present at the medial femur condyle, with a lesion consisting of less than 50% of the normal cartilage thickness, in accordance with grade II chondropathy. The frontal view showed centrally and along the medial condyle a retropatellar cartilage lesion also in accordance with grade III chondropathy. Irritation at the lower portion of the medial retinaculum was also revealed.

We explained to our patient the various therapeutic alternatives, in particular the conservative options, and recommended no or only very minor surgical intervention. The patient decided on a conservative course of laser therapy. The therapy was conducted with the commercially available Laserneedle® System (Germany).

The device consists of eight laser needles, each attached to the end of an optical fiber. Laser diodes were used for the light source, and they emit red light at a wavelength of 685 nm and infrared light at a wavelength of 885 nm (bichromatic emission) in continuous-wave mode with an output power of 35 mW per laser needle. The fiber core diameter was 0.5 mm, resulting in a power density of 17.8 W/cm^2 per laser needle. The use of two wavelengths with different scattering properties has the advantage that the tissue light absorbance is more homogeneous, which is critical to achieve the optimal therapeutic effect. The laser needles were not inserted into the skin, but were taped to the skin along the distal part of the femur in the region of the medial condyle and joint cavity with the patient lying relaxed on his back. Therapy sessions lasted 60 min and were performed daily over a period of 3 mo. The total irradiation dose emitted by the eight laser needles in each 60-min treatment session was 1008 J.

A



B

FIG. 1. These MRI images, made March 16, 2005, are a coronary fat-suppressed PD TSE sequence. (**A**) Axial and (**B**) frontal images, showing a linearly subcortical focus at the medial femur condyle with adjacent spongiosa edema (necrotic zone) reaching deep into the bone marrow.

The treatment parameters were deduced from our experimental data on human osteoblast cultures, which demonstrated a significant increase in osteoanabolic activity.[17] The treatment duration and frequency of sessions were chosen and adapted according to the patient's functional status and level of pain.

No additional treatment was administered during the LLLT therapy period. The patient quit jogging and kept fit only by weight training once a week, daily exercising on a cycling ergometer for 30 min, and playing golf occasionally.

We agreed with our patient to assess therapeutic progress regularly with MRI. The first check-up on April 25, 2005, revealed distinct regression of the spongiosa edema at the medial femur condyle, as well as a decrease in size of the subcortical focus (Fig. 2). The cartilage lesion grade II at the medial inner femur condyle did not at that point show any change. The signal intensity of the linearly demarcated subcortical focus lay under that of the joint cavity, implying no communication between the lesion and the joint cavity. The check-up revealed as secondary findings small synovial cysts in Hoffa's fat pad. Clinically marked pain reduction and virtually complete resolution of the patient's complaints had also taken place. The patient experienced pain only when briskly walking. Since he was satisfied with therapy results up to that point, we agreed to continue it.

The next check-up on June 16, 2005, 3 mo after treatment onset, revealed nearly complete restitution of the spongiosa edema (Fig. 3). A faintly visible subcortical demarcation continued to show on the images. The depth of the cartilage ulceration at the femoral condyle had receded to less than 50% of the total cartilage thickness, in accordance with grade II chondropathy. Our patient was clinically entirely pain-free, even when training vigorously on the bicycle.

During the follow-up period, the patient also did not take any medication. The final check-up on December 5, 2005, confirmed the findings of June 16: full recovery had taken place (Fig. 4). The initially diagnosed Morbus Ahlbäck could no longer be seen, and 35 wk after treatment onset the chondropathy at the medial femur condyle showed complete restitution. Any remaining pathology was at this point minor, in accordance with grade I chondropathy. The cartilage at the femoral condyle also showed marked improvement. There were slight alterations seen in the chondral surface, consisting only of a solitary, flat lesion, in accordance with grade I–II chondropathy.

Discussion

MRI has proven to be the most sensitive method to detect SONK at an early stage,[18] to enhance visualization of bone marrow, and to distinguish necrotic tissue from viable tissue with a high level of specificity.[2] This method is currently considered the gold standard and was therefore used for diagnosis and therapeutic evaluation in the present case.

Based on MRI findings, SONK can be categorized into four stages.[19] In stage I, bone marrow edema is present in the load-bearing zone of the femoral condyle. This initial stage is reversible. In stage II, early subchondral fracturing with flattening of the affected weight-bearing portion of the femoral condyle is observed. Further progression leads to osteochondral fracturing in stage III, and consequently to secondary osteoarthritis in stage IV.

A B

FIG. 2. These MRI images, made on April 25, 2005, are a coronary fat-suppressed PD TSE sequence. (**A**) Axial and (**B**) frontal images, demonstrating distinct regression of the spongiosa edema at the medial femur, as well as a decrease in size of the subcortical focus.

A B

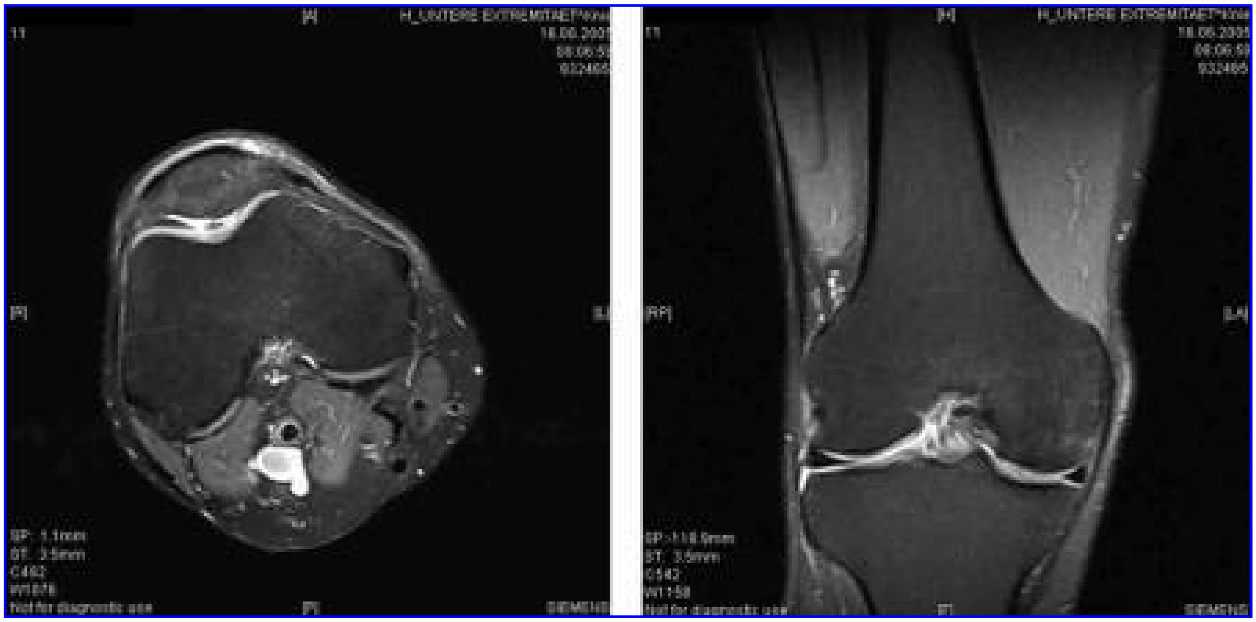


FIG. 3. These MRI images, made on June 16, 2005, are a coronary fat-suppressed PD TSE sequence. (**A**) Axial and (**B**) frontal images, showing almost complete restitution of the spongiosa edema.

Derived from the MRI data showing SONK stage III and in accordance with our patient's request, we decided to treat nonsurgically. With regard to the basic evidence of laser-induced biostimulation,[11–14] clinical research demonstrates the potential positive effects of LLLT in osteoarthritis and rheumatoid arthritis,[15,16] and with our own clinical experience we considered this treatment option to be appropriate in the present case. Laser-needle stimulation using the parameters described above demonstrated successive restitution over the course of the therapy. Distinct restitution of the spongiosa edema took place 5 wk after treatment onset, and the final check-up of December 5, 2005 revealed complete restoration of joint integrity. These findings may be explained by the above-mentioned evidence suggesting the biostimulatory effects of LLLT on biological processes. Laser-induced vascular relaxation and increased microcirculation have been demonstrated in animals[20,21] and in humans.[11,22] Data from the study of Bayat et al. in-

A B

FIG. 4. These MRI images, made on December 5, 2005, are a coronary fat-suppressed PD TSE sequence. (**A**) Axial and (**B**) frontal images, demonstrating complete restoration of joint integrity.

dicated that laser irradiation of random skin flaps without recognizable blood vessels in rats reduced vasospasm, produced vasodilation, and significantly reduced necrosis.[23] In addition, recent research has identified laser-induced regenerative processes in surgically-damaged rat tibia. Garavello et al. demonstrated that laser therapy applied transcutaneously accelerated the deposition of bone matrix, and histological characteristics showing active recovery of the injured tissue.[12] Using biochemical and radioactive labeling methods, Yaakobi et al. found a significant increase in osteoblastic activity at the injured site, as reflected by increased alkaline phosphatase activity.[13] The laser treatment also evoked a twofold increase in the rate of bone repair, as evidenced by the rate and extent of calcification. Likewise, morphometrical and histological analyses by Garavello-Freitas et al. showed that daily laser irradiation stimulated the growth of the trabecular area, and improved parallel organization of bone matrix collagen fibers within the first week, probably related to the activation of osteoblasts to produce bone matrix.[14] In the second week, the effects of laser irradiation changed from stimulatory action on bone growth to an inhibitory action, indicating increased activity of osteoclasts to promote bone resorption and remodeling. The authors therefore concluded that a two-stage mechanism might be involved in the interaction of the laser and the bone repair process. Since an adequate blood supply or neovascularization is crucial for regenerative and proliferative processes, it was suggested that laser irradiation also promotes angioneogenesis. This assumption may be verified by light microscopic examination of histological sections that reveal new formation of blood vessels over the course of low-level laser stimulation.[12]

Conclusion

The present case report provides the first indication that laser-needle stimulation with the parameters described here may be a promising tool for complementary and alternative therapeutic intervention for spontaneous osteonecrosis of the knee. It is likely that laser-needle therapy stimulated neovascularization and osteogenesis. Further research in the form of randomized, controlled trials should be done to assess the clinical effectiveness of laser-needle therapy and compare it to current standard treatments for SONK.

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