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Selection of treatment modalities in children with chronic osteomyelitis

Kronik osteomiyelitli çocuklarda cerrahi tedavi yöntemlerinin seçimi

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Amaç: Kronik osteomiyelit tanısıyla tedavi edilen çocuk hastalarda klinik ve izlem bulguları ve tedavi yöntemleri değerlendirildi.

Çalışma planı: Çalışmaya kronik osteomiyelit nedeniyle tedavi edilen 22 çocuk hasta (14 erkek, 8 kız; ort. yaş 8±7) alındı. Enfeksiyon bölgeleri 11 hastada femur, sekiz hastada tibia, bir hastada ulna, iki hastada radiustu. On altı hastada geçirilmiş travma öyküsü, 14 hastada patolojik kırık, bunların da dokuzunda segmenter kemik kaybı vardı. Tüm olgulara en az bir kez debridman uygulandı ve en az altı hafta olmak üzere antibiyotik tedavisi verildi. Gerektiğinde antibiyogram sonuçlarına göre ilaçlar değiştirildi. İmmobilizasyon alçıyla sağlandı; ancak, kemik instabilitesi varlığında eksternal fiksasyon yapıldı. Ortalama takip süresi 54 ay idi.

Sonuçlar: On üç hastada debridman işlemleri, altı haftalık antibiyotik tedavisi, ek greftleme (5 hasta) ve immobilizasyon ile klinik iyileşme sağlandı. Segmenter kemik kaybı olan dokuz hastanın yedisinde kemik rekonstrüksiyonu işlemlerine gerek duyuldu. Ulnar osteomiyeliti olan bir olguda kendiliğinden cisim rejenerasyonu gözlendi.

Çıkarımlar: Çocuk kronik osteomiyelitlerinin yarısından çoğu debridman ve antibiyotik tedavisiyle tedavi edilebilir. Bununla birlikte, debridman sonrası segmenter kemik kaybı görülen hastalarda kemik rekonstrüksiyonu işlemleri gerekir.

Anahtar sözcükler: Çocuk; debridman/yöntem; osteomiyelit/etyoloji/mikrobiyoloji/tedavi.

Objectives: We evaluated clinical and follow-up findings and treatment methods of pediatric patients with chronic osteomyelitis.

Methods: The study included 22 children (14 boys, 8 girls; mean age 8±7 years) who were treated for chronic osteomyelitis. Infection sites were the femur, tibia, ulna, and radius in 11, 8, 1, and 2 patients, respectively. Sixteen patients had a history of trauma. Fourteen patients had fractures, nine of which were associated with segmentary bone defects. All the patients underwent at least one debridement and received antibiotic treatment for at least six weeks. When necessary, medications were modified according to the antibiogram results. Cast immobilization was applied, but external fixation was used when bone instability existed. The mean follow-up period was 54 months.

Results: Clinical improvement was achieved in 13 patients following surgical debridement procedures, antibiotic treatment for six weeks, secondary grafting (5 patients), and cast immobilization. Of nine patients with segmentary bone losses, seven patients needed bone reconstruction procedures. Spontaneous shaft regeneration was observed in one patient with ulnar osteomyelitis.

Conclusion: The results of surgical debridement and antibiotic treatment are satisfactory in more than half of the pediatric patients with chronic osteomyelitis. However, those developing segmentary bone defects after surgical debridement require bone reconstruction procedures.

Key words: Child; debridement/methods; osteomyelitis/etiology/microbiology/therapy.

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The treatment of osteomyelitis (OM) has been evolving since late 1940s with the achievement of penicillin production for clinical usage. After the revolution in infection treatment, OM is now a disease of high morbidity rather than high mortality.[1,2] With the greater awareness of the osteoarticular infections, improved diagnostic tests and increased clinical usage of antibiotics (AB), chronic OM became an uncommon problem in children. By the time, trauma and operative procedures for bone are the rising causes of OM, in comparison to hematogenous spreading. [3] Socioeconomic, biologic and hereditary factors are effective on the initiation of the disease.[1,2,4] Minor trauma usually exists in the history. After the permanent adhesion of the microorganism to the bone, inflammation, bone infarction, cortical destruction, and sub-periosteal pus formation are the steps of the onset of the disease in consequence. If the duration is more than a month or if there is recurrence after the first remission, it is possible to name the case as chronic osteomyelitis.[5-8] The orthopaedic surgeon should have some objective evaluation findings to guide him, before planning the treatment of a case with chronic osteomyelitis.

Clinical findings, follow up records and treatment methods of the pediatric patients with chronic osteomyelitis were evaluated in this study based on the review of the literature and 12 years of experience.

Patients and methods

Twenty-two children (14 male, 8 female, mean age 8±7) with chronic osteomyelitis, treated between December 1990 and December 2002 were included in this study. The mean duration time of the complaints was 5±4 months at the first admittance. Infection sites were femur (n=11), tibia (n=8), ulna (n=1), and radius (n=2). The mean follow up was 54 months. The infection took place on diaphysis in 13 patients and metaphysis in nine patients.

Sixteen of the patients had trauma history. Nine of them had minor ones as falling, blunt hit by a soccer ball, hit by a kick; seven of the patients had trauma history resulting a fracture. Three of them with femoral fractures (ages were 15,15, and 13 and all were male) had been operated on, in other hospitals and infection developed as postoperative complications. Remaining four patients had radius (2), ulna (1) and tibia (1) fractures. They were from rural area and had been immobilized in a kind of traditional bandage by local unlicenced bone-setters. After the removal of it, redness, swelling, and pain were noticed.

Six patients had no trauma history. Two of them were boys and they were both one year old. One of them had a sequester of 3 centimeters (cm) and a pathological fracture on his left femur. The other had the infection on his left tibia for two months, relatively in slow progress; with no sequester formation. The third patient was a 6-year-old girl, who had infection on her left tibia for 5 months and a sequester of 7 cm. The fourth patient was a 13-year-old girl, had been suffering from femoral chronic OM for 3 years.

Clinical examination, complete blood count, erythrocyte sedimantation rate (ESR), C-reactive protein (CRP) measurement and radiographic evaluation were done for each of the patients at the first admittance. All of them had x-ray findings supporting the diagnosis of osteomyelitis and elevated values of ESR, and CRP. All but three had draining sinuses. The patients went under one or several debridement operations. All of the infected soft tissues were resected and the bone was curettaged down to the healthy structure with good blood supply during the procedure. We didn't refrain from radical debridement concerning the soft tissue coverage. Segmentary defects occured in nine patients after the procedures. Tissue samples were taken from bones and environmental soft tissue, for histopathologic evaluation and cultures and antibiogram investigation. Empiric antibiotic (AB) treatment was started immediately after the surgical debridement. First generation sephalosphorines were the first choice before, but, we have been preferring sulbactam-ampicilline as the first drug to use since 1999 because of the resistant strains of the bacteria. AB treatment was modified depending on antibiograms when needed and carried on, for at least six weeks. Patients were reexamined after 1.5, 3, 6, 12 months and x-rays, ESR and CRP measurements were done for follow-ups. The patients with higher values than normal were consulted by infectious disease specialist and AB treatment was carried on, in corrobation when needed. For patients with activation during the follow ups, debridement

was performed when required. For the rest of the patients, yearly follow ups were adviced when there was no additional complaint. Parenteral (PE) administration of ABs was preferred during initial three weeks and continued with oral pharmacetic forms later on. Twelve patients needed debridement procedures more than one. Simple secondary grafting procedures were enough for five cases to achieve bone healing. When there was cortical continuity on x-ray in two planes, immobilization was provided by casts. When it was destroyed, external fixation was used.

Segmentary bone loss was faced in nine patients. One of them was a four-year-old boy, with a sequester of 6 cm on his left ulna (Figure 1a). He has had complaints for 3 months. On x-rays, there was involucrum surrounding all over the sequester, as forming a bridge between the healthy bone ends. For this patient, his ulna was stabilized with a unilateral external fixator and sequestrectomy was done (Figure 1b). The periosteal continuity was protected and sutured as if to form a tubular structure. Spontaneous regeneration of the shaft took place in three months (Figure 1c).

For seven patients (four tibias and three femurs), distraction-compression osteogenesis was performed to reconstruct the bone (Figure 2a). Uniplanar- bilateral fixators, for tibias and unilateral fixators for femurs were used for the procedures. The fixators were applied to the proksimal and distal healthy bone ends, through two holding clamps, with three schanz pins on each side. Care was taken not to damage the epiphyseal plates. Lengthening clamp was applied just below the proximal holding clamp and an osteotomy was performed between these two clamps (Figure 2b). Lengthening was started on the tenth day, with the rate of 1mm/day in four sessions. The procedure was stopped when the predicted length was obtained; the fibrous tissue, between the confronting bone ends, was excised surgically and cancellous grafting was done. Immobilization was carried on, until the consolidation was confirmed radiographicaly (Figure 2c). In one patient with tibial osteomyelitis, whose defect

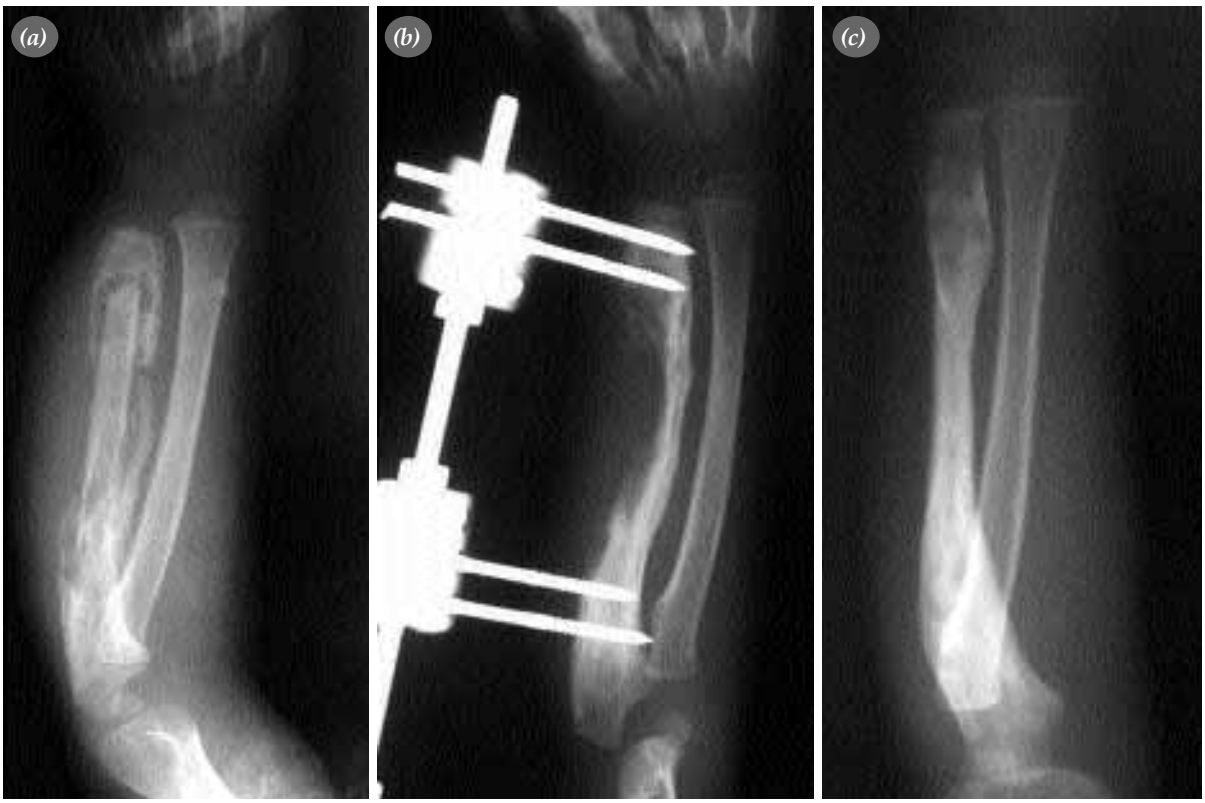

Figure 1. (a) A-P x-ray of the case with OM after ulnar fracture, showing the sequester and the involucrum formation around it. **(b)** Postoperative A-P forearm x-ray of the same case. Sequestrectomy was done and uniplanar external fixation was performed. **(c)** Shaft regeneration took place after three months.

was 9 cm, fibular bone transposition was performed. He was lost in follow up after 18 months.

Results

In thirteen patients (59.1%) surgical debridement procedures, AB treatment for 6 weeks, and cast immobilization were sufficient for clinical improvement. Secondary grafting procedures were needed for five of them. Twelve patients (54.6 %) needed debridement procedures more than one time. They went back to their normal daily lives after a mean treatment period of 5.1 months (from 3 to 8).

Pathogen bacteria were isolated in 11 patients (50 %). Methicilline sensitive in six, and methicillin resistant Staphylococcus aureus were isolated in four patients. In one patient, Pseudomonas aureginosa, Proteus mirabilis, methicillin resistant Staphylococcus aureus were isolated altogether. She had been suffering from this severe infection for four years and had had several debridement procedures before her admittance to our hospital.

Out of the patients with segmentary loss, the boy with the ulnar infection healed completely by spontaneous shaft regeneration, without any restriction of movement. No additional surgical reconstruction was done for him but sequestrectomy and external fixation. Reconstruction was performed for the remaining patients (3 femurs, 4 tibias) by distraction and compression osteogenesis methods, and they are now full weight bearing without any limb-length discrepancy. Proximal bone union couldn't be achieved for the patient with fibulary transposition. He was lost in follow up after 18 months. Except him, the mean recovery period for the remaining eight patients was 10 months (between 9.5 and 11). Postoperative complications were, scar formation in 16 cases, diaphyseal bowing over 10 degrees in four cases (3), and non-union of proximal tibio-fibular osteosynthesis in one case.

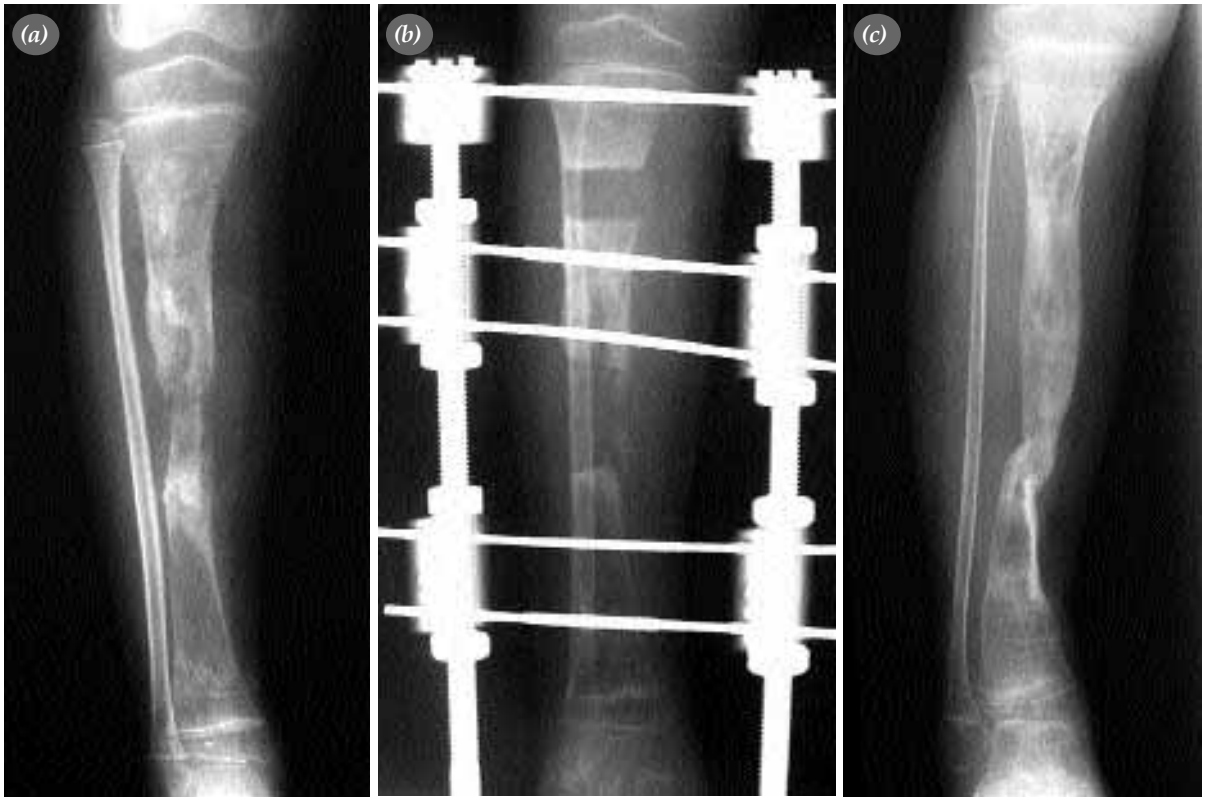

Figure 2. **(a)** A-P tibial x-ray of a case with chronic OM developed after hematogenous spreading , presenting the large sequester an no involucrum formation **(b)** Segmentary defect was reconstructed by distraction-compression osteogenesis method, bilateral uniplanar external fixator was used, lengthening was performed selectively on tibia, the external fixator pins were applied directly to tibia, missing the fibula on purpose. **(c)** x-ray of the same case after the healing was completed.

Discussion

Although definitive cause of OM has not been identified yet, it is clear that socioeconomic and environmental factors and biologic and hereditary features of the host have influence on the initiation of the disease.[1,2,4] Traumatic causes and surgical interventions are the leading etiologic factors nowadays. Etiologic causes were trauma and surgery for sixteen of our patients (72%). Staphylococcus aureus strains are the most frequently isolated bacteria in bone infections and have receptors for collagen, fibrinogen, fibronectin, bone-sialoprotein and heparin-sulphate. Injury to the bone possibly may expose binding sites for this organism, and becomes a predisposing factor for bone infection. Adhesion is reversible at the very beginning. Then, a 'biofilm' is formed by the synthesis of capsular polysaccharides, that acts to protect the microorganism from host defence and antibiotics and the adhesion becomes irreversible.[4,8-10] S. aureus was isolated as pathogeneous agent in 10 out of 11 patients in this study. After this irreversible phase, the vicious cycle of OM process begins with the vascular and cellular responses of the host. Prevention is currently not possible to this phase. On some occasions, doctors who are using AB prophylaxis before the surgery or immediately after the trauma may have some role in preventing OM. It is not as challenging to diagnose chronic OM as acute forms of bone and joint infections. Blood tests and x-rays are usually enough for certain diagnosis.[11] Initial diagnosis were made by clinical examination, blood test and x-ray for all our patients. Bone aspiration is important if the patient has no draining sinuses with atypical clinical presentation. Ultrasonography is reported to be used especially to diagnose the acute cases in children.[12] Although advanced imaging techniques are rarely used for chronic OM, magnetic resonance imaging (MRI) technique, which has been proven to be highly sensitive and specific, is preferred to find out the extension of the bone destruction and pus localization.[13,14] Scintigraphy methods are also suitable. [4,5,8] The patient, who has OM, has usually pus formation and bone necrosis at the time of diagnosis. At this stage, surgical debridement is necessary for the drainage of the pus and debridement of the necrotic tissues, besides AB treatment.[11,15] Although surgery planning, has great influence on the prognosis of the patient, depending on the x-ray findings, aspiration, and MRI, this couldn't be done for our patients because of the emergency conditions and implant existence.[13,14] Under those circumstances, evaluation of the environmental soft tissues and bone during the surgery carry importance for comprehensive debridement. If there is no or little detachment of the periosteum from the bone; draining the pus, fenestration of the bone, curettage and irrigation are usually enough, as surgical treatment procedures. If there are sequestration and large detachment of the periosteum; the involucrum formation and the quality of the soft tissue coverage over the bone should be considered to determine the surgical procedure. Tubular sequestration is usually observed in children, unlike adult OM. If the involucrum formation is forming a bridge between the healthy bone ends, and lying all over the sequester as a sign of periost continuity, the prognosis is usually good. 'Adequate involucrum' needs some preconditions. These are, an intact periosteum with a healthy overlying tissue, and a sequester acting like a model for the periosteum to produce involucrum and preserving the area for involucrum formation by preventing the periosteal tube from collapsing. [5,11,16-18]

If there is a healthy soft tissue coverage and an adequate involucrum formation; sequestrectomy, drainage, debridement of the necrotic tissue and vigorous irrigation should be done. Attention should be paid for not giving any damage to the soft tissue overlying the periost and breaking the involucrum. The periost should usually be preferred to be incised longitudinally to take the sequester out. Leaving the sequester inside the involucrum is not suggested because it may form a shelter for the dormant bacteria. The periosteum should be preserved and sutured to form a tubular structure. Immobilization is important for postoperative care. Plaster casts may be used to provide it.

If there is a tubular sequester but not enough involucrum formation and if the soft tissue coverage is healthy without draining sinuses; the surgical procedure should be limited to drainage, irrigation and immobilization. Sequestrectomy should be delayed for 3 to 12 months to give an opportunity to involucrum formation with AB treatment.[2,19]

If there is sequester and unhealthy soft tissue coverage with draining sinuses, insufficient blood

circulation and inadequate involucrum formation; it is possible to presume that the patient will have a segmentary defect after the debridement procedure. In those cases, the surgeon should not hesitate to perform sequestrectomy which will lead to healing of sinuses.[2,7] Irrigation with saline and sinus tract excisions should also be performed. Immobilization also stabilization when necessary should be provided. After improving the infection by the admittance of AB's, a treatment plan should be developed to cope with the segmentary defect.[11] If the defect is less than 2 cm, grafting can usually be carried on. But if the defect is larger, we prefer lengthening procedures for reconstruction.[20-22] Uniplanar fixators are usually the choice of device for our clinic. Complications are unavoidable while treating patients with chronic OM. High complication rates were reported in the literature.[11,16,19] More than one complications may happen in a case. Therefore, treating OM, is a long battle for both of the patient and the doctor, which is given not only against the disease itself, but also the complications.

Surgical debridement is one of the main steps in the treatment of chronic OM besides AB treatment and immobilization in children. Besides, their responses to bone reconstruction procedures with external fixators are better than the adults.[23] Timing of the sequestrectomy has definitive influence on involucrum formation, regeneration, and preserving the length of the bone. More than half of the pediatric patients with chronic OM can be treated by debridement and AB treatment. Cases with adequate involucrum formation and healthy soft tissue coverage have good prognosis. Segmentary bone loss is seen, when there is inadequate involucrum formation and poor soft tissue coverage. Those cases need bone reconstruction procedures.

References

1. Jawetz E. Penicillins and cephalosporins. In: Katzung BG, editor. Basic and clinical pharmacology. 5th ed. Norwalk: Appleton & Lange; 1992. p. 626-38.
2. Kharbanda Y, Dhir RS. Natural course of hematogenous pyogenic osteomyelitis (a retrospective study of 110 cases). J Postgrad Med 1991;37:69-75.
3. Lew DP, Waldvogel FA. Osteomyelitis. Lancet 2004;364: 369-79.
4. Norden CW. Acute and chronic osteomyelitis. In: Armstrong D, Cohen J, editors. Infectious diseases. Barcelona: Mosby, Harcourt Publishers; 1999. p. 2.43.1-2.43.9.
5. Bobechko WP. Infections of bone and joints. In: Lovell WW, Winter RB, editors. Pediatric orthopedics. 2nd ed. Philadelphia: J. B. Lippincott; 1986. p. 437-56.
6. Green EN. Bone and joint infections in children. In: Weinstein SL, Buckwalter JA, editors. Turek's orthopaedics. 5th ed. Philadelphia: J. B. Lippincott; 1994. p. 127-40.
7. McNally MA, Small JO, Tofighi HG, Mollan RA. Two-stage management of chronic osteomyelitis of the long bones. The Belfast technique. J Bone Joint Surg [Br] 1993;75:375-80.
8. Tachdjian MO. Bone and joint infections. In: Herring JA, editor. Tachdjian's pediatric orthopaedics. 3rd ed. Texas: W. B. Saunders; 2002. p. 1841-77.
9. Auh JS, Binns HJ, Katz BZ. Retrospective assessment of subacute or chronic osteomyelitis in children and young adults. Clin Pediatr 2004;43:549-55.
10. Resnick D, Niwayama G. Osteomyelitis, septic arthritis, and soft tissue infection: mechanisms and situations. In: Resnick D, editor. Diagnosis of bone and joint disorders. 3rd ed. Philadelphia: W. B. Saunders; 1995. p. 2325-418.
11. Lazzarini L, Mader JT, Calhoun JH. Osteomyelitis in long bones. J Bone Joint Surg [Am] 2004;86:2305-18.
12. Mah ET, LeQuesne GW, Gent RJ, Paterson DC. Ultrasonic features of acute osteomyelitis in children. J Bone Joint Surg [Br] 1994;76:969-74.
13. Dangman BC, Hoffer FA, Rand FF, O'Rourke EJ. Osteomyelitis in children: gadolinium-enhanced MR imaging. Radiology 1992;182:743-7.
14. White LM, Schweitzer ME, Deely DM, Gannon F. Study of osteomyelitis: utility of combined histologic and microbiologic evaluation of percutaneous biopsy samples. Radiology 1995;197:840-2.
15. Shuford JA, Steckelberg JM. Role of oral antimicrobial therapy in the management of osteomyelitis. Curr Opin Infect Dis 2003;16:515-9.
16. Daoud A, Saighi-Bouaouina A. Treatment of sequestra, pseudarthroses, and defects in the long bones of children who have chronic hematogenous osteomyelitis. J Bone Joint Surg [Am] 1989;71:1448-68.
17. Eyre-Brook AL. The periosteum: its function reassessed. Clin Orthop Relat Res 1984;(189):300-7.
18. Peltier LF. The classic. The growth of bone. Chapter III. Osteogenic power of bone bereft of periosteum. William Macewen, F.R.S. Clin Orthop Relat Res 1983;(174):5-14.
19. Fowles JV, Lehoux J, Zlitni M, Kassab MT, Nolan B. Tibial defect due to acute haematogenous osteomyelitis: treatment and results in twenty-one children. J Bone Joint Surg [Br] 1979;61:77-81.
20. Tezer M, Ozturk I, Kucukkaya M, Kabukcuoglu Y, Eren T, Kuzgun K. Bone transport techniques in defect pseudarthroses in children. [Article in Turkish] Acta Orthop Traumatol Turc 1999;33:23-9.
21. Villa A. Bone lengthening. In: Maiocchi AB, Aronson J, editors. Operative principles of Ilizarov. Milan: Williams & Wilkins; 1991. p. 288-309.
22. Yeargan SA 3rd, Nakasone CK, Shaieb MD, Montgomery WP, Reinker KA. Treatment of chronic osteomyelitis in children resistant to previous therapy. J Pediatr Orthop 2004; 24:109-22.
23. Kucukkaya M, Kabukcuoglu Y, Tezer M, Kuzgun U. Management of childhood chronic tibial osteomyelitis with the Ilizarov method. J Pediatr Orthop 2002;22:632-7.