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QUESTION 4: What differentiates acute from chronic osteomyelitis (OM)? Is it clinically important to distinguish one from the other?

RECOMMENDATION: Current literature is lacking consistent criteria for a distinct time point that differentiates the acute and chronic forms of infection. Differentiating between acute and chronic types may have practical implications on treatment plan and final prognosis.

LEVEL OF EVIDENCE: Limited

DELEGATE VOTE: Agree: 95%, Disagree: 5%, Abstain: 0% (Unanimous, Strongest Consensus)

RATIONALE

To address this question an extensive search of the literature was conducted. Our search aim was to identify articles reporting on the diagnostic criteria for acute or chronic osteomyelitis. A clear definition of OM in terms of temporal evolution was considered mandatory. Furthermore, in order to investigate the potential practical significance of the temporal distinction of OM into acute or chronic types, we aimed to identify papers reporting on the outcome of antimicrobial therapy or combined treatment (antimicrobial plus surgical intervention) of acute osteomyelitis. Our exclusion criteria included case reports, expert opinions, experimental studies, infections associated with prosthetic implants, diabetic ulcers and non-orthopaedic bone infections (facial, cranium, ribs).

We searched the Medline, Embase, Ovid, Cochrane and Google Scholar databases using the PubMed search engine. Our search strategy included the following Medical Subject Headings (MeSH) terms and Boolean operators: ("osteomyelitis"[MeSH Terms] OR

"osteomyelitis"[All Fields]) OR "bone infection"[All Fields] OR "osseous infection"[All Fields] AND ("classification"[Subheading] OR "classification"[All Fields] OR "classification"[MeSH Terms]). This search process yielded 856 records. After rejection of duplicates and irrelevant articles by their title or abstract, there remained 45 papers for which full text was obtained. After careful screening against the eligibility criteria, there were ultimately eight eligible articles left.

A second search process was run in parallel, as follows: acute [All Fields] AND ("osteomyelitis"[MeSH Terms] OR "osteomyelitis"[All Fields]) AND "humans"[MeSH Terms]. It yielded 3,339 results. After removal of duplicates and rejection of irrelevant articles based on their title or abstract, there were 56 studies remaining, for which a full text was obtained. Eventually, after screening of these manuscripts against the eligibility criteria, another 11 eligible articles were obtained. In addition, another 4 articles were added from handsearch of the relevant bibliographies, leading to a total of 23 eligible articles (see Fig. 1).

OM is an inflammation of the bone and bone marrow caused commonly by pyogenic bacteria, and rather infrequently by mycobacteria or fungi [1,2]. It is classically classified by the duration of its clinical course as acute or chronic. Acute osteomyelitis represents the early stage of the evolutionary process of the disease, usually characterized by an intense clinical picture. Its diagnosis is based on a combination of clinical, laboratory and imaging findings, with a definitive diagnosis established by positive bacterial cultures of aspirate, bone or blood samples [3]. A longstanding infection which progresses to bone necrosis and sequestrum formation is termed chronic OM [1,2,4]. This condition is usually characterized by more subtle clinical findings, occasionally the presence of draining sinus tracts, or may progress intermittently [5]. While the clinical differentiation is marked by necrosis and sequestrum formation, defining a specific time threshold beyond which an acute infection could be considered chronic is difficult [1,2]. The current literature is lacking consistent criteria for a distinct time point that differentiates the acute and chronic forms of infection. Nevertheless, this distinction is of only limited value in adults as they are very rarely affected by acute OM and, even if this does occur, prompt diagnosis before transition to chronicity is often missed. On the contrary, in children, who are frequently affected by acute hematogenous OM, differentiating between acute and chronic types has practical implications regarding the treatment plan and final prognosis. This is mainly due to the fact that younger patients have the ability to resorb, at least to some degree, devitalized bone tissue, thereby removing foci of "biofilm type" of bacterial growth and potentiating the effectiveness of early-instituted antimicrobial treatment [6]. Additionally, the duration of this antimicrobial treatment differs between acute and chronic OM, with the acute form being treated with three to six weeks of specific antimicrobials targeted at identified pathogens after initial empiric formulations, and the chronic form being treated for up to six months with targeted antimicrobial therapy without initial empiric therapy [7]. This is due to the fact that certain pathophysiological changes that occur during the evolution of the inflammatory process (such as pus formation, reparative reaction, formation of involucrum and bone sequestration), which dictate the treatment plan and prognosis, are time-dependent [8]. Consequently, the differentiation between an acute and chronic form, especially in children, has important implications on the treatment plan.

Some authors do not utilize strict temporal criteria for defining OM. In 1970, Waldvogel et al. emphasized the difficulty in distinguishing between acute and chronic OM in terms of clinical course (type and duration of symptoms) or histologic findings [9,10]. They classified all cases as either "initial episodes" or "recurrences." An initial episode was thought of as representing an acute type of the disease spectrum, while recurrences represented chronic cases. They documented significantly higher treatment failures in "recurrences" as compared to the "initial episodes" for both hematogenous cases ($p = 0.003$) and those secondary to a contiguous focus of infection ($p = 0.0005$). The same definition of acute OM as "initial episode" was adopted by Lieu et al. in a retrospective study of 95 patients aged less than 17 years [11]. Fifty-five percent of them had been treated conservatively, while the remaining 45% had received combined treatment (antimicrobial therapy plus surgery). A recurrence rate of only 8.5% was documented. Other authors utilized a list of clinical, laboratory and imaging criteria to define acute OM in children and adolescents [12–14].

Various temporal thresholds have been used to define acute OM (Fig. 1). The shortest time threshold was one week, and was documented in three studies reporting on pediatric populations (584 children) [15–18]. The percentage of surgical intervention across all three studies ranged from 5.3% to 56%, and the recurrence rate of the infection ranged from 0 to 12% (pooled estimate of effect size for recurrence rate [random effects model]: 3.5%, 95% confidence interval (CI): 0.1 to 11.5%, with significant statistical heterogeneity: $I^2 = 87\%$). In one study, a sub classification of acute hematogenous OM was proposed into early-acute OM (diagnosed within 48 hours of onset in children over one year of age), late-acute OM (diagnosed at 5 days or more in children over 1 year of age) and neonate-infantile type [16]. The rationale for this classification was based on the findings of the study that the success rate of antimicrobial treatment was 92% for early-acute type versus 25% for late-acute OM. Another commonly-used threshold was two weeks and was utilized by studies reporting on either pediatric [19–21], adult [22] or mixed populations [23]. Two out of the three studies dealing with the pediatric population reported on the recurrence of the acute infection, which ranged from 0 to 7% (pooled estimate of effect size [random effects model]: 3.6%, 95% CI: 0.02 to 13%, $I^2 = 79\%$), with the rate of operative intervention ranging from 8 to 44% [19,20]. Finally, in one study reporting on open, infected bone wounds of the distal fibula/tibia, an acute infection was considered when the duration of open wound drainage was less than six weeks [24].

The definition of chronic OM is much more variable in the literature. Various lower limits of duration of symptoms exist, above which a chronic osseous infection is considered (Figs. 2 and 3). These range from at least a week in one study [17] to at least six months in three studies [25–27]. In-between, there are studies using the lower limits of two weeks [23], six weeks [24], one month [22] and two months [28]. However, in all studies the most consistent sign of chronicity of infection was bone sequestration. In a recent systematic review of the literature on the classification on the long bone OM the authors concluded that the terms acute/chronic OM are unreliable and do not influence the diagnostic workup or the principles of medical or surgical management [29].

Given the great variability of definitions for acute and chronic OM existing in the literature, we conclude that these terms are impractical in most cases as they lack accuracy in describing the underlying disease, and cannot dictate the treatment plan or predict prognosis. An exception to the above conclusion is the pediatric cases of acute OM due to the greater capacity of the younger patients to absorb necrotic bone and, therefore, to potentiate the effects of medical treatment. Additional variation in the treatment plan between acute and chronic forms of OM is in the duration of antimicrobial treatment. Lima et al. concluded that in acute cases patients should be given initial empiric antimicrobial treatment followed by targeted treatment for three to six weeks, while chronic cases require up to six months of targeted therapy [7].

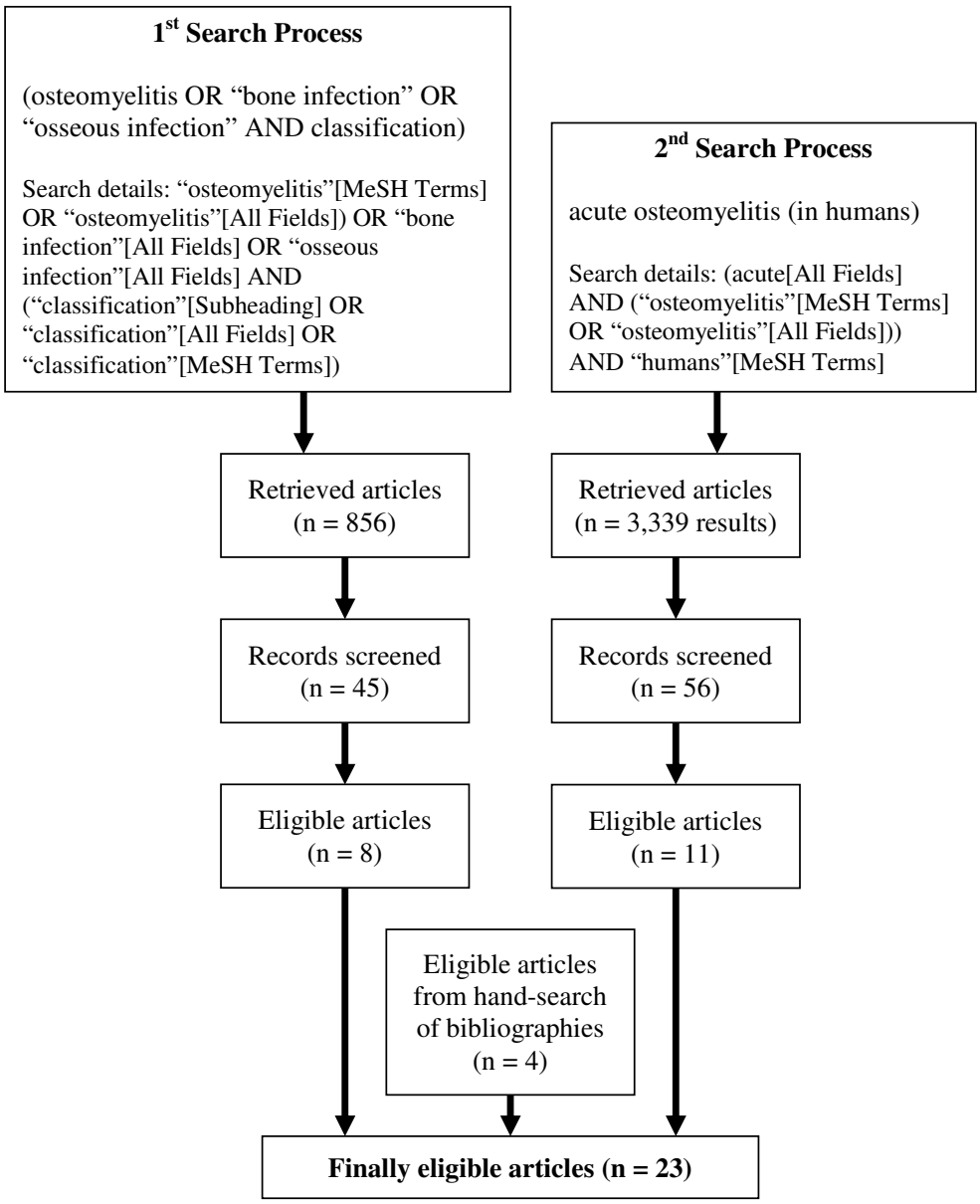

FIGURE 1. Search strategy flow chart.

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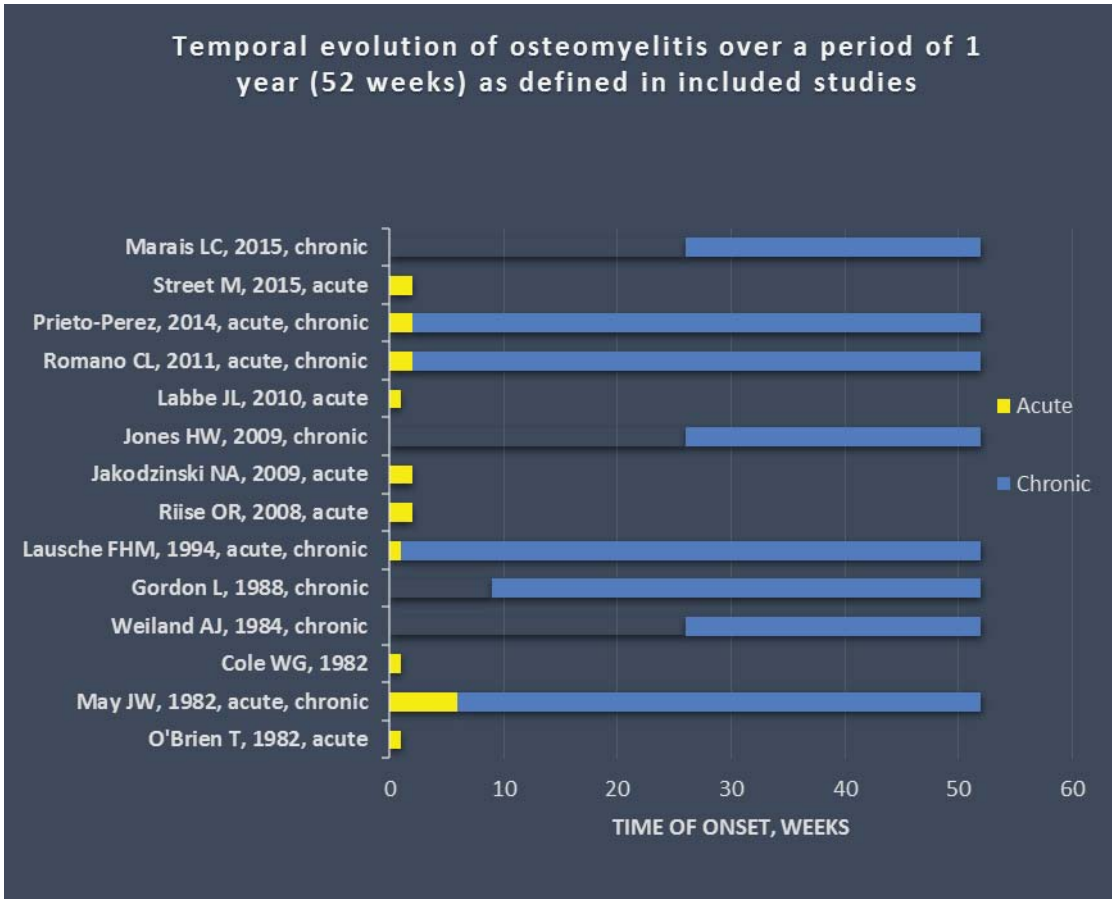

FIGURE 2. Temporal evolution of osteomyelitis over a period of one year as defined in included studies.

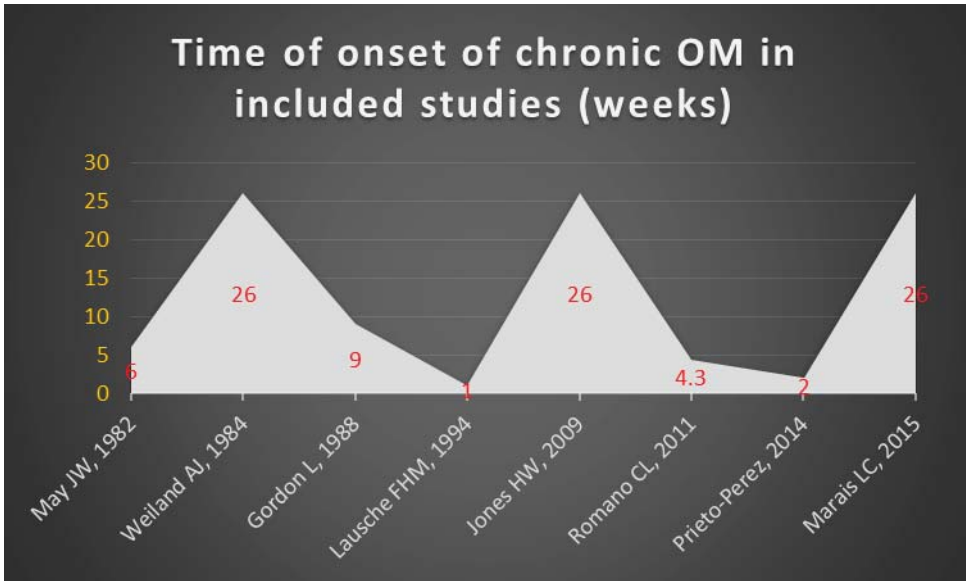

FIGURE 3. Time of onset (weeks) of chronic osteomyelitis, as defined in the included studies.

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QUESTION 5: Is synovial fluid or fracture hematoma always aseptic? If not, could this play a role in acute infection or periprosthetic joint infection (PJI) after open reduction and internal fixation (ORIF)?

RECOMMENDATION: Fracture hematoma is not always aseptic. It is unknown if synovial fluid is always aseptic. In addition, it is unclear if this plays a role in acute infection or fracture-related infection (FRI) after ORIF.

LEVEL OF EVIDENCE: Moderate

DELEGATE VOTE: Agree: 95%, Disagree: 5%, Abstain: 0% (Unanimous, Strongest Consensus)

RATIONALE

The association between soft-tissue conditions and infection has been well-known since the 1970s, when Gustilo and Anderson described how the major risk factor for post-traumatic infection following open fracture was the quality of the soft tissue envelope [1]. More recent evidence has demonstrated how traumatized host tissue can result in altered vascularization, decreased perfusion, increased endothelial permeability and decreased oxygenation; all of which can compromise the body's innate ability to resist local infection [1,2]. The prevailing theory of infection is that it is secondary to inoculation of pathologic microorganisms in traumatized tissues; however, it is unclear how infection occurs in closed trauma if there is no bacterial contamination through an open wound [2]. Some have questioned the common belief that synovial fluid and fracture hematoma is always aseptic based on evidence from other surgical fields that demonstrated how bacterial balance within presumably clean soft tissues affects the likelihood of soft tissue healing versus infection [3].

Two recent studies explored if fracture hematoma or callus was aseptic. In contrast to the prevailing view that these tissues are always clean, both studies found that 14 to 40% of the deep tissues grew bacteria when cultured, but no study has replicated these findings with synovial fluid. Szczesny et al. used conventional and molecular bacterial detection methods to determine if bacteria colonized lower limb soft tissues and bone following closed fractures in 71 patients. Cultures of fracture callus were positive in 26.7% of patients and bacterial rRNA was isolated in 41% of patients [4]. Similarly, Font-Vizcarra et al. evaluated the presence of positive cultures from hematoma in 109 patients with femoral neck fractures. They found that fracture hematoma was positive in 31.2% of all patients [2]. In both studies, the most common cultured organism was *S. epidermidis*. Based on recent basic science data, the presumed mechanism of infection of the deep tissues was that high-stress conditions resulted in decreased ability to contain skin and mucosal flora, leading to seeding of traumatized soft tissues/hematoma by lymphatic spread or transient bacteremia [1,2,4].

Although there is good evidence that fracture hematoma is not always aseptic, it remains unclear if the bacteria within the deep tissues play a role in acute infection or PJI after ORIF. Font-Vizcarra et al. did not find that culture positivity was a risk factor for early post-traumatic infection unless the specimen grew gram-negative rods [2]. Similarly, positive cultures from the fracture callus was not associated with non-union following closed tibia or femur fractures