Prognostic factors associated with mortality in patients with arthritis: a descriptive cohort study – comments on the article by Andreasen et al

RL Roerdink & M Dietvorst


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LETTER

Prognostic factors associated with mortality in patients with arthritis: a descriptive cohort study – comments on the article by Andreasen et al

With great interest we read interest the study by Andreasen et al (1). The aim of the study was to explore the risk factors for the 30 day mortality rate at the time of septic arthritis (SA) presentation. The secondary objective was to describe the microbiological aetiologies, systemic signs of inflammation, and comorbidities.

An electronic database was used to identify patients with SA in Denmark. This study included 215 patients, of whom 101 (47%) had an infection of their joint prosthesis. SA in total joint arthroplasties (TJAs) are actually periprosthetic joint infections (PJIs). The authors did not differentiate between native joint infections and PJIs. These are, however, different kind of infections.

Definitions, pathology, diagnostic work-up, treatment, and prognosis of PJIs differ significantly from SA in native joints (2). Therefore, we argue in this letter why patients with SA and PJI should have been differentiated in this study.

SA is considered to be confirmed in case of the following findings: positive synovial fluid cultures, positive blood cultures with negative synovial fluid cultures, or negative cultures due to previous use of antibiotics, but with purulent drainage of the joint. A definite radiological or postmortem finding can also confirm the diagnosis of SA (3, 4). In this study, the authors chose to define SA as a clinically inflamed joint combined with a positive culture. All patients without bacteria in the synovial fluid were excluded. Their reasons are explained in the discussion.

For PJIs, the Workgroup of the Musculoskeletal Infection Society released the most recent, and widely accepted, definition: a sinus tract communicating with the prosthetic joint or two positive periprosthetic cultures with identical organisms or three out of the five following minor criteria: (i) elevated serum erythrocyte sedimentation rate and C-reactive protein; (ii) elevated synovial fluid white blood cell count (WBC) or change on leucocyte esterase test strip; (iii) elevated synovial fluid polymorphonuclear neutrophil (PMN) percentage; (iv) a single positive periprosthetic culture; and (v) positive histological analysis of periprosthetic tissue (2).

There are different types of PJIs. Acute postoperative infections are defined as infections of the prosthesis that occur within 90 days after surgery (2). Infections diagnosed later than 90 days after surgery are defined as chronic PJIs (2).

Each type has its own treatment modality. In acute PJIs, usual care means debridement, antibiotics, and implant retention (2, 5). Chronic PJIs are treated with implant revision surgery (2, 5). In general, antibiotics are administered for a minimum of 3 months in both acute and chronic PJIs (2, 5). This is due to biofilm formation that occurs on the prosthesis, which functions as a foreign body (2, 5). Therefore, antibiotics that penetrate biofilms are recommended (2, 5). Treatment of native SA generally consists of 6 weeks of antibiotics with needle aspiration or arthroscopy of the joint (6).

Furthermore, there are other factors that may influence the primary outcomes of this study. The characteristics of patients with a TJA in the general population may be different from those of the native joint group. The threshold for operating on vulnerable patients with chronic diseases has decreased (7). So, within the population of patients who receive TJAs, comorbidities may be more often present and they generally have a higher age (7).

The conclusion of the authors that the 30 day mortality rate in their study could not be explained by comorbidities or old age in native SA may have to be reconsidered after eliminating these factors.

Concerning their secondary outcomes, there also are some points of discussion that may be important in interpreting the results of this study.

Systemic signs are less common in chronic PJIs (2). Therefore, the secondary objective, to describe systemic signs of inflammation, could be biased in case a significant number of the included patients had chronic PJIs.

Furthermore, the authors have given averages for several microorganisms for the WBC count in synovial fluid, but it is not known whether they were aware that the cut-offs for WBC and PMNs in synovial fluid are different between acute and chronic PJIs (2). The cut-offs presented in this study may also be biased because of this phenomenon.

In conclusion, this study could be a valuable addition to the current literature in determining prognostic factors associated with mortality in patients with SA in native joints and in PJIs. A recommendation would be to split these different pathologies into separate groups. For the group of PJIs it is recommended also to differentiate between acute and chronic joint infections.

RL Roerdink¹, M Dietvorst²

¹Department of Orthopaedic Surgery, Jeroen Bosch Hospital, ’s-Hertogenbosch, The Netherlands
²Department of Orthopaedic Surgery, Máxima Medical Centre, Eindhoven, The Netherlands
References


Ramon Lucas Roerdink, Department of Orthopaedic Surgery, Jeroen Bosch Hospital, Henri Dunantstraat 1, 5223 GZ ’s-Hertogenbosch, The Netherlands.
E-mail: r.roerdink@hotmail.com