

Reply to “Observations on native valve endocarditis caused by *Kocuria kristinae*”[☆]



Respuesta a «Observaciones sobre endocarditis nativa de la válvula causada por *Kocuria kristinae*»

Dear Editor,

I am grateful for the comments received by Rahim et al.¹ Our intentions with the letter sent was to point out the pathogenic significance *Kocuria kristinae* may have on the development of infectious endocarditis,² without any other apparent primary infectious focal point being measured, unlike the cases referenced by Rahim et al. in these cases obvious infectious niches exist (septic arthritis, infected catheter, infections of soft tissues) and appear to be the starting point of the bacteremia which will foster the development of endocarditis. The aim of our letter was to simply reiterate the importance of *Kocuria kristinae* as a potential pathogen of infectious endocarditis without the need for a clinically apparent primary reservoir. Also that its microbiological determination should be subject to an appropriate clinical interpretation which determines the realisation of the pertinent supplementary techniques.

We insist on several specific aspects: that on occasion this is attributed with a “contaminating” role without pathogenic involve-

ment; that identification should be made not just based on the metabolic study³ of isolation (as in several of the articles referenced by Rahim et al.); that there are no specific sensitivity guidelines (and for this reason it was relativised with *Staphylococcus aureus* cut-off points).

Our intentions was not to carry out a systematic review of the literature with respect to *Kocuria* spp and its pathological/microbiological spectrum, but to avoid under-estimations of *Kocuria kristinae* in isolation and its potential involvement in the development of infectious endocarditis, regardless of whether primary septic systemic outbreaks exist or not.

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Are the systemic inflammatory response syndrome valid in the assessment of febrile neutropenia?[☆]



¿Son válidos los criterios del síndrome de respuesta inflamatoria sistémica en la valoración de la neutropenia febril?

Dear Editor,

We read with interest the review recently published in your journal¹ on the management of infection and febrile neutropenia in the patient with solid cancer. In Figure 1 of the same an action algorithm is proposed for emergency service healthcare attention of patients with febrile neutropenia. In its central section there is a proposal for determination of systemic inflammatory response syndrome (SIRS) or severe sepsis prior to deciding which should be the following steps in the diagnostic and therapeutic process of the febrile neutropenia.

However, we wish to point out that the use of the criteria defined by the systemic inflammatory response syndrome for identifying patients with sepsis has currently diminished for several reasons. Thus, for example, among patients with suspected infection outside

of intensive care units, the predictive value of the in-hospital death rate mortality of other tools such as the *quick* SOFA (which only evaluates the Glasgow scale, systolic blood pressure and heart rate) is statistically superior to that of the Sequential (Sepsis-related) Organ Failure Assessment (SOFA) and the SRIS² itself. There are studies which show that the SRIS criteria lack sensitivity, as they do not determine one in every 8 patients with infection, organ failure and high associated mortality.³ Lastly, these criteria do not possess ideal specificity either, as almost half of hospitalised patients who have no infection develop SRIS criteria at least once during their hospital stay, which demonstrates that its use as a method of detection of sepsis is not overly useful.⁴

As a result, we believe there are better methods than the standard SRIS method for clinical evaluation of patients with febrile neutropenia who present at the emergency services.

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