Nosocomial candiduria in chronic liver disease patients at a hepatobiliary center

Sir,

We read the article “Nosocomial candiduria in chronic liver disease patients at a hepatobiliary center” with great interest. This article clearly highlights the importance of repeat sampling in patients who are reported as having candiduria. Candiduria can occur due to contamination, colonization (of the indwelling catheter or bladder), and infection (Candida cystitis or ascending pyelonephritis or renal candidiasis) as discussed in this article. Taking a repeat sample after insertion of fresh catheter eliminates the chances of contamination, but colonization can still be present without any clinical manifestation of disease.

Differentiation between the colonization and infection is difficult one and should be interpreted in the light of clinical features. Frequently, due to the presence of multiple co morbidities the line of demarcation between Candida colonization and infection is blurred, more so in critically ill patients.

Pathogenesis of urinary tract and renal infections caused by Candida involves hematogenous spread as well ascending infection. When suspecting hematogenous spread various risk prediction scores (colonization index, Candida score) have been designed to guide therapy. Candida colonization has been shown to be a risk factor for invasive candidiasis, but multiple site colonization and heavy colonization is considered more significant than single site colonization. Candiduria represents single site colonization and should not trigger initiation of treatment unless other symptoms are also present. When suspecting ascending infection, one should look for predisposing factors like presence of stone or presence of obstruction, urinary drainage devices, prior surgical procedures, broad spectrum antibiotic use, old age, and diabetes mellitus.

Infectious Disease Society of America 2009 guidelines recommends that asymptomatic candiduria should not be treated unless the patient is at high risk of invasive candidiasis (neutropenia, low birth weight, and patients who undergo urologic procedures). The reason for treating asymptomatic candiduria in high risk patients is mainly prophylaxis.

Chronic liver disease is a very broad terminology and includes various etiologies and stages of evolution in its natural history. Whether all chronic liver disease patients should be included in the high risk remains to be established. Currently, there is a paucity of literature regarding predisposition for invasive Candida infections among chronic liver disease patients. It would have been enlightening if the patient characteristics and stages/classification of liver disease were also mentioned in this study.

The study also reports that 11 (3.5%) patients of candiduria evolved to candidemia. Though it is difficult to perform, genotypic identification is ideally required before one can say that species colonizing is the same as the species responsible for blood stream infection.

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Sir,

I read with great interest the recent case report regarding the serious yet under-recognized acute phenomenon of transfusion associated circulatory overload (TACO).[1] Paramount to the appropriate identification of TACO is the awareness of common precipitants to this transfusion reaction. While rapid and large volume infusions have been thought to precipitate TACO, relatively small volume transfusions (1-2 units) are sufficient to evoke a reaction.[1,2]

What poses a great diagnostic challenge is differentiating a cardiogenic etiology (TACO), to that of permeability pulmonary edema (transfusion-related acute lung injury [TRALI]). This distinction is made, especially difficult since the two conditions present similarly and may coexist.

The case report[1] briefly mentions the notion of B-type natriuretic peptide (BNP) testing in order to differentiate between TACO and TRALI. BNP is a cardiac neurohormone specifically secreted from the ventricles in response to volume expansion and pressure overload. Brain natriuretic peptide is elevated in TACO (<250 pg/mL) and is itself a sensitive and specific indicator of cardiogenic pulmonary symptoms. Moreover, a post-to-pre-transfusion ratio of 1.5 was found to be indicative of TACO, with a sensitivity of 81% and a specificity of 89%, respectively.[3] This presents as a simple and noninvasive clinical test to diagnose or exclude cardiogenic pulmonary edema (TACO) after transfusion.

There is, however, no sole feature that distinguishes TACO from TRALI. TRALI is a diagnosis of exclusion, and requires a multi-faceted approach consisting of a thorough clinical file to include the patient’s fluid and cardiac status, appropriately indicated chest X-ray and pulmonary wedge pressure measurements, and possibly the inclusion of the measurement of BNP. However, possible biological variability in BNP levels[4] may warrant further studies on the efficacy of BNP in TACO diagnosis. Moreover, the feasibility of BNP measurement both before and after a transfusion may be questioned. It may be worthwhile, nonetheless, to measure BNP levels in susceptible populations of patients undergoing blood product transfusion, namely patients at the extremes of age or who are severely anemic.[2,5]

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