

Comment on: “Transfusion associated circulatory overload”

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 profile 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]

James K. H. Ho, J. Henry G. Antrum

Department of General Medicine, University of Edinburgh, Royal Infirmary of Edinburgh, Edinburgh, EH16 4SU, United Kingdom

Correspondence:

Mr. James K. H. Ho,
Flat 2F1, 50 George Street, Edinburgh, EH22LR, United Kingdom.
E-mail: s0807475@sms.ed.ac.uk

References

1. Agnihotri N, Agnihotri A. Transfusion associated circulatory overload. *Indian J Crit Care Med* 2014;18:396-8.
2. Popovsky MA. Transfusion-associated circulatory overload. *ISBT Ser* 2008;3:166-9.
3. Zhou L, Giacherio D, Cooling L, Davenport RD. Use of B-natriuretic peptide as a diagnostic marker in the differential diagnosis of transfusion-associated circulatory overload. *Transfusion* 2005;45:1056-63.
4. Azevedo A, Bettencourt P, Barros H. Demographic, clinical and echocardiographic determinants of B-type natriuretic peptide plasma concentration. A population-based study. *Rev Port Cardiol* 2007;26:105-13.
5. Roback JD, editor. Non-infectious complications of blood transfusion. *AABB Technical Manual*. 17th ed., Ch. 27. Bethesda: AABB; 2011.

Access this article online

Quick Response Code:



Website:

www.ijccm.org

DOI: 10.4103/0972-5229.140163