Cardiol Young 2004; 14: 560–561

ISSN 1047-9511

Brief Report

Spontaneous echoes due to hypoalbuminemia

Amir Hadash, Yulia Braver, Avraham Lorber

Department of Pediatrics and Pediatric Cardiology, Meyer Children's Hospital, Rambam Medical Center, Haifa, Israel

Abstract We found spontaneous echoes in two teenagers with nephrotic syndrome and profound hypoalbuminemia, both having normal cardiac structure, function and output. The phenomenon disappeared after the level of albumin normalized. In one patient, all spontaneous echoes disappeared following convalescence, the level of albumin in the serum then being documented at normal levels. The second patient, who presented with profound hypoalbuminemia, was infused with human albumin because of oliguria, following which the spontaneous echoes disappeared.

Keywords: Nephrotic syndrome; hypoalbuminemia; spontaneous echoes

SPONTANEOUS ECHOES CAN BE SEEN IN THE HEARTS of patients with low cardiac output due to lesions such as tight mitral stenosis, atrial fibrillation, and reduced left ventricular function.[1,2] Such spontaneous echoes have also been observed in the setting of functionally univentricular hearts with low cardiac output. To the best of our knowledge, such spontaneous echoes have not previously been described in the setting of nephrotic syndrome, when patients can have profound hypoalbuminemia but with normal cardiac structure and function. The volume of intravascular blood may alter significantly in the course of nephrotic syndrome, particularly in the setting of hypoalbuminemia.[3,4] The reduced levels of albumin in the blood also lower the colloid osmotic pressure, thus leading to depletion of the intravascular space, and promotion of extravascular congestion and peripheral edema. There is also retention of salt and water, with increasing activity of the renin-angiotensin-aldosterone cascade.[4] This leads to hemoconcentration, albeit often without significant polycythemia or increased blood viscosity. We have now noted, however, that these changes, induced the phenomenon of spontaneous echoes in two patients with normal cardiac morphology and function.

Correspondence to: Dr Avraham Lorber, Department of Pediatric Cardiology, Meyer Children's Hospital, Rambam Medical Center, P.O. Box 9602, Haifa 31096, Israel. Tel: +972 4 854 2275; Fax: +972 4 854 2175; E-mail: a_lorber@rambam.health.gov.il

Accepted for publication 19 May 2004

Patient 1

A 15-year-old male was admitted to the hospital with edema of the limbs and the periorbital regions that started a month prior to hospitalization. His blood pressure was 157/87 mmHg and he had a functional apical systolic murmur. Initial analysis of the blood revealed hemoglobin at 15.5 g/dl, a normal white blood count, an erythrocytic sedimentation rate of 105 mm/h, hypercholesterolemia at 402 mg/dl, hypoproteinemia at 4 g/dl, hypoalbuminemia at 1.4 g/dl, and massive proteinuria. Laboratory screening for infectious, proliferative and collagen diseases was negative. An abdominal ultrasonography showed hepatic and splenic enlargement with minimal ascites. Echocardiography showed normal atrial arrangement and connections, no shunts, normal valvar function, no pericardial effusion, normal global and segmental ventricular function, normal ventricular compliance, and no hypertrophy. Spontaneous echoes were seen moving from the inferior caval vein to the right atrium and to the right ventricle (Fig. 1). Corticosteroid therapy was started, with a rapid and good response manifested in the improvement of edema and urinary output. Although no renal biopsy was performed, the patient was discharged from the hospital with the diagnosis of nephrotic syndrome. After a follow-up

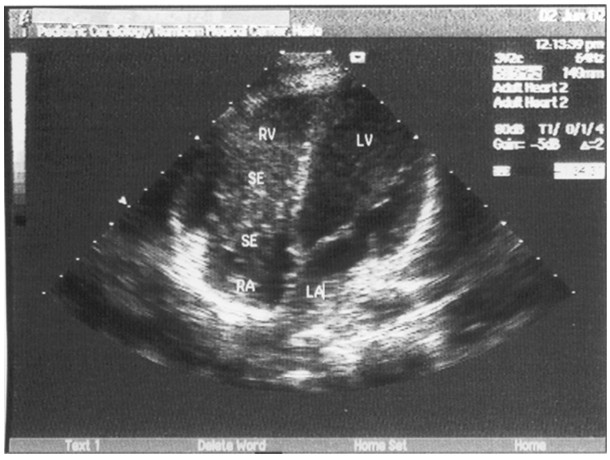

Figure 1.
Spontaneous echoes (SE) progressing as a cloud in diastole from the right atrium (RA) to the right ventricle (RV).

of 2 months, the serum albumin had risen to 4.1 g/dl, and repeated echocardiography revealed resolution of the spontaneous echoes.

Patient 2

Another 15-year-old male was admitted to the hospital with peripheral and periorbital edema, which had lasted for 3 weeks. His blood pressure was 154/95 mmHg and there was pitting edema of the feet. Blood analysis showed hemoglobin of 14.7 g/dl, a normal white blood count, hypercholesterolemia at 463 mg/dl, hypoproteinemia at 3.8 g/dl, hypoalbuminemia at 1.7 g/dl, but normal levels of creatinine, urea and electrolytes. Urinanalysis showed many erythrocytes, 5–8 white blood cells per high power field, while a 24 h sample revealed 11 g of protein. A renal biopsy confirmed the minimal changes of the nephrotic syndrome, and we started treatment with corticosteroids. Echocardiography demonstrated usual arrangement and connections, no shunts, normal valvar function, no pericardial effusion, normal global and segmental ventricular function, normal ventricular compliance, and no hypertrophy. Spontaneous echoes were again seen running from the inferior caval vein to the right atrium and to the right ventricle (Fig. 1). We infused human albumin and furosemide because of the oliguria and generally edematous state. Follow-up echocardiography performed on the next morning showed no spontaneous echoes. His level of albumin in the serum decreased dramatically, to 1.7 g/dl, during the following week, as was expected in the course of active nephrotic syndrome. Repeated echocardiography showed the re-occurrence of spontaneous echoes from the inferior caval vein to the right atrium and the right ventricle. This patient did not respond to corticosteroid therapy. Dramatic clinical improvement, with normalization of levels of albumin in the serum followed, treatment with cyclosporine.

Discussion

Spontaneous echoes are reported extensively in patients with low cardiac output, reduced left ventricular function, mitral valvar stenosis and are also associated with atrial fibrillation. Patients with functionally univentricular hearts have also been shown to have spontaneous echoes, either after a total cavopulmonary connection or in the setting of low cardiac output. As far as we know, the phenomenon has not previously been encountered in patients with normal hearts and profound hypoalbuminemia. The depletion of blood volume, and retention of extravascular fluid which accompanies hypoalbuminemia, may cause other changes in the content of the blood, and may account for echogenic properties, changes for which no satisfactory hypothesis has otherwise been found. We did not find significant hemoconcentration, since the levels of hemoglobin did not alter significantly during the active phase of the nephrotic syndrome, nor during recovery. The only convincing correlation was demonstrated between spontaneous echoes and profound hypoalbuminemia. We lack, therefore, a better understanding of the physiology of this peculiar phenomenon. Further research is needed to elucidate the reasons for the spontaneous echoes, particularly in the setting of hypoalbuminemia and nephrotic syndrome.

References

1. Peverill RE, Graham R, Gelman J. Hematologic determinants of left atrial spontaneous echo contrast in mitral stenosis. Int J Cardiol 2001; 81: 235–242.
2. Breuer HW. Spontaneous echo contrast and atrial fibrillation. Circulation 1999; 99: 975–978.
3. Behrman RE, Kliegman RM, Jenson HB (eds). Nelson's Textbook of Pediatrics, 16th edn. W.B. Saunders Company, Philadelphia, 2000, pp 1592–1593.
4. Orth SR, Ritz E. The nephrotic syndrome. New Engl J Med 1998; 338: 1202–1210.

https://doi.org/10.1017/S1047951104005153 Published online by Cambridge University Press