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LETTER TO EDITOR

Re: Complicated Subacute Bacterial Endocarditis in a Patient with Ventricular Septal Defect

رد: حالة معقدة لالتهاب جرثومي تحت الحاد للشغاف المبطن للقلب لطفلة لديها فتحة قلبية بالحجاب الحاجز البطيني

Sir,

I have two comments on the interesting case report published in the February 2014 issue of the Sultan Qaboos University Medical Journal (SQUMJ) by Al-Senaidi et al.1

First, echocardiography (ECHO) is included in the diagnostic algorithm of infective endocarditis (IE). In their case report, Al-Senaidi et al. noted that the ECHO showed a 4 mm perimembranous ventricular septal defect with a left-to-right shunt and a peak gradient of 85 mmHg. There were also two vegetations attached to the tricuspid valve with moderate regurgitation and no stenosis. The large vegetation measured 8 x 6 mm and the smaller one, 6 x 4 mm. During the patient’s clinical course, the large vegetation was dislodged and caused a pulmonary embolism.1 This embolic event is quite interesting, as anecdotal studies have shown that a mobile vegetation, or vegetations that are >10 mm in length, are significantly associated with embolic phenomena.2,3 I presume that the evolution of the patient’s pulmonary embolism, with a vegetation of less than 10 mm, could be explained by the observation that the different antibiotics used in the treatment of the IE had different effects on the size of the vegetation,4 and hence, the risk of vegetation dislodgement.

The studied patient was started on intravenous antibiotics: ampicillin (50 mg/kg, six-hourly), vancomycin (15 mg/kg, eight-hourly) and gentamicin (2.5 mg/kg, eight hourly). In an interesting German study, the effect of different antibiotic regimes on vegetation size was evaluated by transoesophageal ECHO in 183 patients with IE.4 Significant differences in vegetation size were noted with different kinds of antibiotics: treatment with vancomycin showed a 45% reduction; ampicillin a 19% reduction; penicillin a 5% reduction; penicillase-resistant drugs a 15% increase, and cephalosporin a 40% increase.4 Penicillin, cephalosporin and penicillase-resistant drugs were associated with an increased embolic risk, while vancomycin was associated with the formation of abscesses and cephalosporin with increased mortality.4 The authors observed that “Plotting changes in vegetation size against the incidence of embolism and mortality, linear regression analysis suggested a 40–50% reduction in vegetation size, thereby greatly reducing the risk of embolism and mortality.”4 I presume that both clinicians and echocardiographers should not only consider discernible vegetations as the sole diagnostic parameter for IE, but also the size and mobility of the vegetations. This would be helpful in the prediction of a patient’s pulmonary and systemic embolic risk. In addition, it may encourage the need to adopt a more aggressive antibiotic therapy and to perform an early surgery, if necessary.

Second, I do agree with Al-Senaidi et al.’s recommendation that clinicians should have a high index of suspicion for IE as the possible cause of prolonged fever, especially in the presence of congenital heart disease (CHD).1 Moreover, Al-Senaidi et al.’s case report could be added to the available literature on IE,1 as IE forming as a late presentation of CHD has rarely been reported.5

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References