

1. What is the current thought on horizontal earlobe creases as markers for CAD?

Question submitted by Dr. Tom Simpson, St. Jacobs, Ontario

Numerous clinical and autopsy studies have evaluated the relationship between an earlobe crease (defined as a unilateral or bilateral oblique or diagonal crease extending a distance > half the diagonal length of the earlobe) and coronary artery disease (CAD).

Although there is some conflicting evidence, the majority of data suggest there is an association between the two. A review of 1,424 patients showed an earlobe crease has a sensitivity of 65% and a specificity of 72%, with a positive predictive value of 42% for the presence of CAD.¹

However, given that the prevalence of both an earlobe crease and CAD increases with age, it is unclear as to whether this association reflects diffuse, underlying atherosclerosis in elderly patients.

Nonetheless, an earlobe crease is clearly not a modifiable risk factor, nor does this association imply causality. Therefore, if anything, its utility serves to alert a clinician to measure well-studied, powerful, and modifiable predictors for cardiovascular morbidity and mortality (*i.e.*, abdominal circumference, lipid levels, and blood pressure).

Reference

1. Tranchesi JB, Barbosa V, de Albuquerque CP, et al: Diagonal earlobe crease as a marker of the presence and extent of coronary atherosclerosis. *Am J Cardiol* 1993; 70(18):1417-20.

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2. Could you provide an update on the approach to Dressler's syndrome?

Question submitted by Dr. Maria Valois, Ottawa, Ontario

The diagnosis of Dressler's syndrome is based on the development of pleuritic chest pain, typically with fever, occurring at least one week after a known cardiac insult. Investigations are frequently non-specific, but can include:

- electrocardiogram demonstrating pericarditis,
- chest X-ray showing pleural effusion and/or pulmonary infiltrates,
- elevated white blood cell count, and
- elevated erythrocyte sedimentation rate.


Treatment goals include pain control and suppression of inflammation. First-line treatment consists of high-dose acetylsalicylic acid (ASA), up to 650 mg orally four times/day. While other non-steroidal anti-inflammatory drugs (NSAIDs) may be used, indomethacin is not advised as it has demonstrated adverse effects on ventricular remodeling.¹

The duration of high-dose treatment should be one to two weeks, with rapid tapering. For

additional pain control, acetaminophen and, rarely, narcotics may be necessary.

Corticosteroids may be used in patients refractory to ASA or when ASA is contraindicated. Oral prednisone is initiated at relatively high doses (0.25 mg/kg/day to 0.75 mg/kg/day), with subsequent rapid taper.

Since the principal morbidity with Dressler's syndrome is related to pleuritic chest pain, with no effect on overall prognosis, colchicine should be considered as an alternative to steroids in refractory cases. Chronic oral colchicine, 1 mg/day for up to one year, as monotherapy or in combination with corticosteroids (discontinued after six weeks) effectively treats symptoms and prevents recurrence.

Although routine investigations are not warranted, surveillance through clinical assessment for acute (cardiac tamponade) and chronic (constrictive pericarditis) complications should be maintained. 

Reference

1. Jugdutt BI, Basualdo CA: Myocardial infarct expansion during indomethacin or ibuprofen therapy for symptomatic post infarction pericarditis. Influence of other pharmacologic agents during early remodelling. *Can J Cardiol* 1989; 5(4):211-21.

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