Background

Infective endocarditis (IE) is a severe infection where bacteria colonize the heart valves leading to the formation of vegetations containing bacteria, fibrin, and platelets. Septic embolization or heart failure can complicate the infection. Treatment relies on long courses of intravenous antibiotics and sometimes valvular surgery is necessary. The Swedish Society for Infection Medicine keeps a Registry of the clinical presentation of IE cases.

Platelet aggregation is thought to be important for the pathogenesis of IE with Staphylococcus aureus and alpha-haemolytic streptococci (1) and the mechanisms behind platelet aggregation by bacteria have been extensively studied. Most described mechanisms involve the simultaneous binding of specific IgG and of another plasma protein such as fibrinogen to the bacterial surface. The plasma proteins then signal through platelet receptors leading to activation and aggregation (1).

Our interest in platelet-bacterial interactions

When the PhD student Daniel Johansson was recruited, Oonagh Shannon and myself noted that despite that enterococci are the third most common pathogen in IE, nothing was known about the potential interactions between these bacteria and human platelets. We investigated this and found that many isolates of E. faecalis aggregate and activate human platelets through an IgG-dependent mechanism (2). This ability was shared among isolates from the normal flora and isolates causing IE (3). We have also shown that Streptococcus dysgalactiae subspecies equisimilis (group G Streptococcus) and A. urinae both have this ability (4,5). In the case of A. urinae platelet activation was dependent both on IgG against the bacteria and on complement activation (5). Since it was well known that platelet aggregation by bacteria was highly donor-dependent, we preceded to study how bacteria affect platelets in patients with bacteraemia. More specifically, we investigated the response of platelets from patients with bacteraemia to the bacterial isolate causing the infection and we could conclude that platelet aggregation and activation can occur also in these patients (6).

We will now continue to define interactions between platelets and aerococci in a whole-blood aggregometer system.
References


