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REFERENCES

- 1 **Andrew RK**, Lopez JA, Berndt MC. Molecular mechanism of platelet adhesion and activation. *Int J Biochem Cell Biol* 1997;**29**:91–105.
- 2 **Afshar-Kharghan V**, Bray PF. Platelet polymorphisms. In: Michelson AD, eds. *Platelets*. London: Academic Press, 2002:157–80.
- 3 **Douglas H**, Michaelides K, Gorog DA, *et al*. Platelet membrane glycoprotein Ibb gene –5T/C Kozak sequence polymorphism as an independent risk factor for the occurrence of coronary thrombosis. *Heart* 2002;**87**:70–4.
- 4 **Kundu SK**, Heilman EJ, Sio R, *et al*. Characterisation of an in vitro platelet function analyser PFA-100. *Clin Appl Thromb Haemost* 1996;**2**:241–9.
- 5 **Ambrose JA**, Fuster V. The risk of coronary occlusion is not proportional to the prior severity of coronary stenoses. *Heart* 1998;**79**:3–4.
- 6 **Afshar-Kargan V**, Matijevic-Aleksic N, Ahn C, *et al*. The variable number of tandem repeat polymorphism of platelet glycoprotein Ibb and the risk of coronary heart disease. *Blood* 2004;**103**:963–5.
- 7 **Gonzales-Conejero R**, Lozano ML, Rivera J, *et al*. Polymorphisms of platelet membrane glycoprotein Ibb associated with arterial thrombotic disease. *Blood* 1998;**92**:2771–2.

IMAGES IN CARDIOLOGY

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Multiple complications of aortic valve endocarditis diagnosed from the ECG

The ECG is often overlooked as a diagnostic tool in conditions other than ischaemic heart disease and arrhythmias. A 48 year old man presented with symptoms of fever, headache, malaise, and abdominal pain. He was afebrile but was admitted for further investigation. His presenting ECG was unremarkable, showing sinus tachycardia but no other abnormal features. Twenty four hours later, blood cultures grew *Staphylococcus aureus* and the patient was referred for cardiology review at the suggestion of the microbiologists. The patient complained of no new symptoms. The ECG taken at this time showed tachycardia; it also showed notable PR interval prolongation and widespread ST elevation consistent with pericarditis. Consequently, a provisional diagnosis of aortic valve endocarditis with root involvement and pyopericardium was made based on the ECG findings. Subsequent clinical and echocardiographic examination confirmed severe aortic regurgitation and pericardial effusion, and the patient was taken to theatre. Operative findings were of free aortic regurgitation with pus tracking around the commissure between the non- and right coronary cusps and into the pericardium. The patient went on to have a successful aortic valve replacement and antimicrobial treatment. This case acts as a reminder of the diagnostic ability of the ECG, of the rapidly progressive course of staphylococcal endocarditis, and of the importance of daily ECG examination in patients with suspected aortic valve endocarditis.

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