Is congenital tricuspid insufficiency (CTI) as rare as it seems to be?
Andrew C. Chatzis, Nicolas M. Giannopoulos and George E. Sarris

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Reply to the Letter to the Editor

Reply to von Heymann et al.

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I would like to thank Dr von Heymann [1] and colleagues for this interesting interaction. Let me start by confirming to Dr von Heymann the following:

1. Pre operative coagulation assessment were done and patients on anticoagulation or abnormal results were excluded from the study.
2. We did take into consideration Gibbs [2] paper. This is one of the reasons why we continue with aspirin till the day of the operation in all patients. Gibbs paper confirms the universal inhibition of platelet function by aspirin. And since Aspirin inhibition to platelet function is irreversible, the individual variability in platelet function reflects the rate of regaining function by producing new platelets.
3. The other aim of inhibiting platelet is to cancel out their contribution to the activation of neutrophils and hence a beneficial effect on the inflammatory response.
4. There is no strong evidence [3] today to suggest that a decrease in anti thrombin III levels will translate into a decrease in soluble Fibrin level which is the end point of the clotting cascade and have the real effect in haemostasis.
5. The results from Despotis [4] paper can not be compared with ours, his population is smaller and different (re-operations vs. primary CABG).

All in all this is a large study which we set out to exclude most of the confounding variables rather than trying to give plausible explanations. Activated Clotting Time measurement is still in use by the majority despite its critics. The clotting cascade remains a complex multi-factorial system that we have yet to elucidate comprehensively. ACT is a simple test and a good indicator of the haemostatic system.

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References

pericardial defect [2–4]. A case of congenital tricuspid valve can be found in about 30% of the cases reported with congenital defects, patent ductus arteriosus and tetralogy of Fallot. Associated anomalies including mitral stenosis, atrial septal defect, complete absence of pericardium must be considered. We appreciate your report ‘Tricuspid valve repair in a case with congenital tricuspid annulus’. There existed no further congenital anomalies in this case beside the absence of left thoracic pericardium. The anterior tricuspid leaflet appeared of normal structure. The anterior tricuspid leaflet had a normal size and shape but was disrupted from the tricuspid annulus. There existed no further congenital anomalies in this case beside the absence of left thoracic pericardium.

It is to emphasize that any larger pericardial defect including right sided absence of pericardium has an impact on the geometry of the heart and may alter the integrity and function of the atrioventricular valves although a major cardiac displacement is not apparent. In addition we have to be aware that the risk for valvular endocarditis is increased in valvular heart disease and observed valvular thickening or attenuation can be caused by endocarditis [5].

References


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