HEPARIN RESISTANCE DURING OR AFTER CARDIAC SURGERY

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Thrombine activation is a key mediator during cardiopulmonary bypass (CPB) and may explain some cases of heparin resistance during CPB or thromboembolism after cardiac surgery. Antithrombine (AT) is the main cofactor of heparin, influenced by systemic inflammatory syndrome or hepatic dysfunction, with a small plasmatic pool. Some clinical cases are described but observational or clinical trial are scarce.

Based on a literature review, on a personal cohort of mechanical cardiac assistance in adults and children, and on a multicenter observational study we proposed a practical approach of heparin monitoring and antithrombin substitution.

A French multicenter study including 450 patients, confirms that antithrombin could decrease during standard CPB in 30%, and not only in case of severe multiorgan dysfunction. Hemodilution could be a partial explanation. Mean SAPS score was 24 and 14% had low cardiac output. 21% of patients had clinical symptoms of hemorrhage or thrombo-embolic events during the 3 month follow up.

A second personal cohort of 25 patients treated by mechanical cardiac assistance presented an acquired AT deficiency (<60%). A single perfusion of AT (27±12 UI/kg) increased the AT plasma level from 47±10 to 75%. aXa therapeutic level was obtained in 19±10 hours with an heparine dose of 260±135 UI/kg/24h. As an increased and rapid efficiency of anticoagulation is obtained, AT perfusion should be slower than initially recommended.

In severe patients aPPT test is not adequate to monitor non fractionned heparin. Heparine resistance seems to be linked with an increased risk of thrombo-embolic event. Double blinded trial including AT substitution is needed but difficult.