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Hemodynamic consequences of postoperative inflammation after pulmonary valve replacement

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Objective:

Outcome of pulmonary valve replacement is typically excellent, but freedom from reoperation is variable and not predictable for the individual patient. There are some patients who develop early restenosis of the conduit valve or relevant conduit valve insufficiency.

We speculated that the severity of inflammation immediately after the valve implantation may contribute to the early conduit failure.

Methods:

From 8/2008 to 12/2010 pulmonary valve replacement by xenograft was performed in 42 patients (30 males, mean age 13.8 ± standard deviation 6.6 years).

Fifteen patients underwent a Ross procedure, 11 patients suffered from severe pulmonary insufficiency after Fallot-repair, and 14 patients required conduit replacement after previous conduit implantation for right ventricular outflow tract reconstruction.

Hancock II®, Matrix-P/-P plus®, and Contegra ® pulmonary valve conduits were used in 29, 11, and 2 patients, respectively.

C-reactive protein (CRP) and white blood cell count (WBC) after surgery was retrospectively compared to the results of Doppler echocardiography during the follow-up after 3, 6, 12, and 24 months.

Results:

CRP increased in all patients significantly within 48 hours after surgery to mean 151 ± 69 mg/l. There was no significant difference in peak CRP value between the groups, neither between different surgical procedures nor between the different valves (fig. 1).

WBC was mean 12.6 ± 3.7 x 10^3/µl and 13.1 ± 4.2 x 10^3/µl at the first 2 days.

Mean follow-up was 12.4 ± 8.7 months. Flow velocity in the pulmonary conduit at 3, 6, and 12 months after surgery was mean 2.0 ± 0.5, 2.3 ± 0.5, and 2.4 ± 0.6 m/s, respectively (fig. 2).

No or trivial conduit valve insufficiency was detectable in all patients during follow-up.

Peak CRP but not peak WBC was significantly correlated with Doppler peak flow velocity in the pulmonary conduit at 3 months after surgery (r=0.33, p=0.03; fig. 3).

In addition, early CRP (r=0.42, p=0.005) and postoperative WBC (r=0.42, p=0.006) both evaluated 4 hours after admission to the Intensive Care Unit were significantly correlated with Doppler peak flow velocity at 3 months.

Conclusions:

Postoperative CRP level is correlated to the flow velocity in the conduit valve 3 months after replacement. However, long-term follow-up is necessary to answer the arising question whether postoperative inflammation is also negatively correlated to freedom from reoperation.

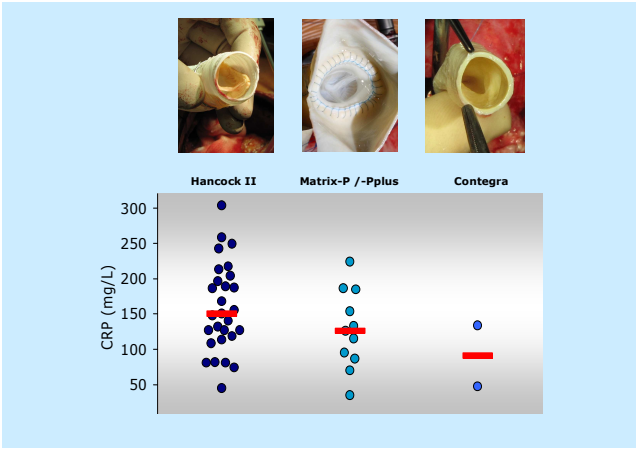

Fig. 1: Peak CRP respecting the type of valve

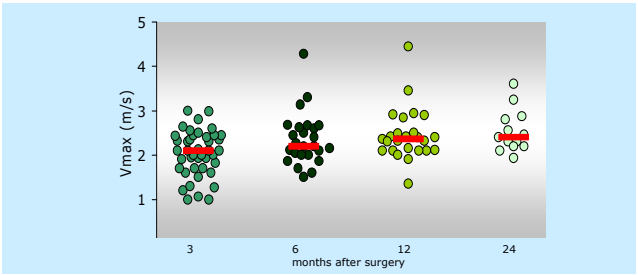

Fig. 2: Doppler peak flow velocity in the conduit during follow-up

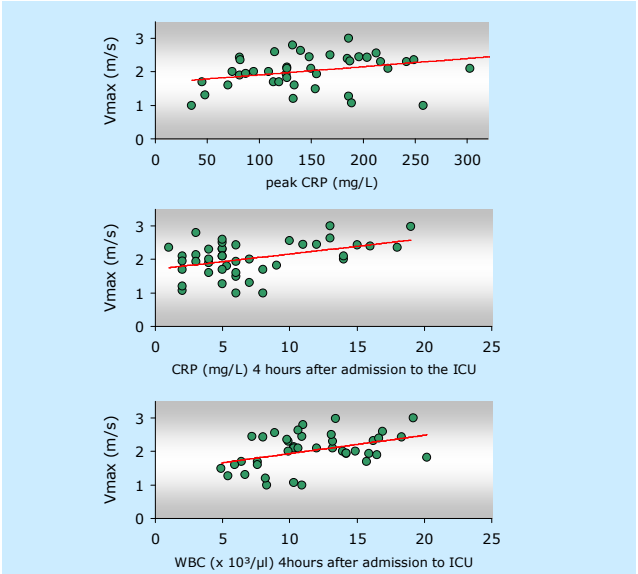

Fig. 3: Correlation between Doppler peak flow velocity in the conduit at 3 months after surgery and different parameters of inflammation

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