PROTAMINE REACTIONS

Type 1: Hypotension with rapid administration (Histamine).

Type 2: Anaphylaxis (preformed antibodies and IGE).

Type 3: Acute pulmonary edema due to complement to be treated by Calcium: 2 mg/1mg protamine + Alpha agents + Milirinone, inamrinone + Re-administer Heparin +Steroids +Prepare to reinstitute CPB.

(One.Histamine.Two.anaphylaxis, Three.Edema.Complement).

- *(Prota.Ba.Po. Prota.Arginine. Prota.Salmon)*
- ***Protamine: It activates the complement system through the classic pathway.***
- *The alternative pathway of complement is activated by Bypass.*

Antithrombin III deficiency Syndrome: Heparin Resistance is the state of heparin resistance that requires 2-3X usual dose of heparin. U gave the usual dose of heparin and ACT is still low even after 3 times the usual heparin dose due to antithrombin III deficiency and previous exposure to heparin. Treatment is based on replenishing antithrombin III with FFP or concentrate. ***(Anti.Fresh.Anti.concentrate)***

- **The natural anticoagulants are Antithrombin-III, Protein C & S.** ***(C&S.anticoag.)***
- **Heparin acts on intrinsic pathway.** ***Heparin is acidic and negative charged. (Hepar.int) (Hepa.aci.ne)***
- **Extrinsic pathway: Factor *7*** ***(Ex.Seven)***
- **Intrinsic pathway: Factor12;** ***(Int.twelve)***
- **Common pathway: factor10.** **(Co.ten)**

Primary hemostasis: It is the platelet function. *(primary.Plate)*

Platelet adhesion, activation and aggregation.

©Secondary hemostasis*: It is the function of the plasma and plasma proteins. (Secondary.plasma.and.proteins)*

(BART TRIAL): Blood Conservation Using Antifibrinolytics in a Randomized Trial.

BART trial 2008 was terminated early because of the significant mortality of aprotinin compared with tranexamic acid and aminocaproic acid(6% Vs 3.9% Vs 4%). The primary outcome was massive bleeding and secondary outcome was mortality from any cause at 30 days. There was a modest reduction in the risk of massive bleeding in the Aprotonin group (9.5% Vs 12% Vs 12%). There was a strong and consistent negative mortality trend associated with aprotinin.

Mangano et.al., 2006 found the use of aprotinin associated with a double risk of renal failure requiring dialysis, 55 percent increase in the risk of myocardial infarction or heart failure and 181 percent increase stroke or encephalopathy and an increased mortality (2.8 vs 1.3%) Neither aminocaproic acid nor tranexamic acid was associated with an increased risk of these events. All the agents reduced blood loss. (Aprotonin.Renal.Infarction.Stroke)

The perfusionist must modify the standard techniques during bypass during using aprotonin by using ultrafiltration and cell saver salvaged blood and monitoring anticoagulation by heparin titration.

It may be wise to double the standard ACT.

(APRO.BEFORE.INCISION.APROTONIN.NEVER.REPEATED.Apro.saver.ultrafiltration.APRO.DoubleACT)

Dose of Aprotonin:

Low Dose: 1 million KIU IV loading dose, 1 million KIU into the pump prime volume, 250,000 KIU per hour of operation as continuous intravenous infusion.

High Dose: (exactly double Regimen B): 2 million KIU IV loading dose, 2 million KIU into the pump prime volume, 500,000 KIU per hour of operation as continuous intravenous infusion.

Aprotonin is anti-kinin and Kalekerine inhibitory unit(KIU)

- The predictors of AF in thoracic surgery are age >65 y, pneumonectomy, left sided operations.
- RISK FACTORS OF STROKE POSTCARDIAC SURGERY : ABCD2; Score between 0-7. Age, Blood pressure, Clinical Stroke, DM and duration of symptoms.
- The strongest factor is unilateral weakness(2 points) and duration>1 hour(2 points)
- The commonest factor for postoperative AF is sudden withdrawal of beta blockers.
- POAF is 30% after cardiac surgery.
- Embolic Manifestations with AF is due to LA thrombus.
- Embolic Manifestations with Sinus Rhythm is due to LA Myxoma.
- Labs for AF are ECG, Thyroid function tests, enzymes, electrolytes and CXR.
- Spontaneous Cardioversion may occur in 50-70% in the 1st 24 hours.

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THANK YOU!