Intracoronary Adenosine Induced hyperaemia reduces the incidence of myonecrosis due to micro infraction after elective Percutaneous Coronary Intervention

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Percutaneous Coronary Intervention (PCI) is associated with 5% to 30% incidence of elevation of serum Creatine Kinase MB fraction (CK-MB) fraction reflect the periprocedural myonecrosis. Appearance of Creatine Kinase MB (CK-MB) in the circulation is the strongest predictor of long term clinical outcome of PCI2-5, correlating the deleterious consequences of myonecrosis on Left Ventricular function or Electrophysiological stability6. Pathophysiology of myonecrosis during PCI has been related to side branch closure or compression, major intimal dissection, intramural thrombus formation, coronary spasm, distal atheroembolization. The first four causes may be clinically apparent and measures can be taken during the procedure, the latter may be entirely asymptomatic and occur even angiographically uneventful procedure. Contrast enhanced Magnetic Resonance Imaging suggest that distal embolization of atherogenic materials from plaque disruption7, causing micro infarction by obstructing the blood flow at capillary level8,9.

Studies show that intra venous Adenosine infusion as an adjunct to reperfusion in the treatment of acute myocardial infarction associated with reduction of heart muscle damage10,11.

Adenosine is a naturally occurring nucleoside with a half life in blood of less than 10 seconds. Adenosine may be administered via intravenous or Intracoronary route, produce hyperemic effect that is commonly used to measure the coronary flow reserve during PCI. The mechanism of the cardio-protective effect of adenosine include preconditioning17, antiplatelet activity18, anti inflammatory effect19 and hyperaemia20. There is a evidence from previous study that despite a short serum half life the cardio-protective effect the effect of Intracoronary adenosine infusion in human subjects persist much longer17.

Hyeraemic effect of Adenosine may be beneficial to ameliorate distal embolization of platelet thrombi following elective PCI. One study suggested that endogenous release of adenosine could compensate distal embolization of small particles21. The minimum dose is required to achieve minimum Fractional Flow Reserve (or maximum hyperaemia), 2 micro grams for both Left and Right Coronary artery in human without any significant side effect22, 50 micro grams bolus administration of adenosine through the guiding catheter before elective PCI to ameliorate distal embolization of relatively small platelet thrombi6, Although only a fraction of the administered adenosine would reach the target distal lesion, this above dosage (50 micro grams) and route of administration was sufficient to achieve maximum hyperaemia. Moreover, an Intracoronary bolus approach to administration, lower cost and reduce risk of bradycardia.

Adenosine induced hyperaemia can potentially ameliorates the deleterious effect of distal embolization associated with non-urgent elective Percutaneous Coronary Intervention (PCI) through dilatation of microvasculature. This may reduces capillary obstruction by facilitating the throughout passage of embolized platelet thrombi out to the venous end of coronary circulation, there by reduce the incidence of post procedural micro infraction and myonecrosis.

References:


