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Dermcidin isoform-2 induced nullification of the effect of acetyl salicylic acid in platelet aggregation in acute myocardial infarction

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The aggregation of platelets on the plaque rupture site on the coronary artery is reported to cause both acute coronary syndromes (ACS) and acute myocardial infarction (AMI). Acetyl salicylic acid has beneficial effect in the inhibition of platelet aggregation in ACS but it failed to do in AMI. The concentration of a stress induced protein (dermcidin isoform-2) was much higher in AMI than that in ACS. Incubation of normal platelet rich plasma (PRP) with dermcidin showed one high affinity ($K_d = 40$ nM) and one low affinity binding sites ($K_d = 333$ nM). When normal PRP was incubated with $0.4\mu\text{M}$ dermcidin, the platelets became resistant to the inhibitory effect of aspirin similar to that in the case of AMI. Incubation of PRP from AMI with dermcidin antibody restored the sensitivity of the platelets to the aspirin effect. Incubation of AMI PRP pretreated with $15\mu\text{M}$ aspirin, a stimulator of the NO synthesis, resulted in the increased production of NO in the platelets that removed the bound dermcidin by 40% from the high affinity binding sites from AMI platelets. When the same AMI PRP was retreated with $10\mu\text{M}$ aspirin, the aggregation of platelets was completely inhibited through NO.

Biography

Sarbashri Bank has completed his M.Sc. (gold medalist) in Biochemistry in 2012 at the age of 24 from Vidyasagar University and currently he is pursuing PhD in the area of cardiovascular diseases under the mentorship of Prof. Asru K Sinha in Sinha Institute of Medical Science & Technology, Kolkata since July 2012.

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