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Poster Sessions

Effect of pretreatment with diltiazem on mast cell degranulation by adrenaline treatment in anaphylactic rats.

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Anaphylaxis (ANA) induced myocardial ischemia, and adrenaline (Ad) administration may exacerbate myocardial ischemia. Because elevated ST-wave was suppressed by pretreatment with diltiazem in the anaphylaxis leading to myocardial ischemia, therefore, investigating pretreatment with diltiazem on Ad administration after compound 48/80 induced ANA. The β -hexosaminidase (β -hex) activity was significantly increased by ANA induction, and degranulation was observed. Contrary to expectations, a significant increase in β -hex activity was promoted by the administration of Ad under the ANA induction. Interestingly, the increasing β -hex activity due to Ad administration during ANA was significantly suppressed by pre-treatment with diltiazem. The blood pressure immediately increases after the Ad administration rapidly due to the sympathetic nerve like action, therefore considering the parasympathetic nervous system working accordingly, decreasing heart rate. Whereas, when parasympathetic nerves become dominant, β -hex activity is thought to increase by stimulating Gq protein-coupled acetylcholine M1 receptors to introduce Ca ions into mast cells and promote degranulation. ☒Because pretreatment with diltiazem blocked the influx of calcium ions from outside the mast cells by blocking the calcium ion channel, therefore, it is considered that diltiazem suppressed the increase in β -hex activity caused by adrenaline administration after anaphylaxis induction.