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OVERACTIVE BLADDER SMOOTH MUSCLE CONTRACTION AFTER ATP STIMULATION UNDER A CALCIOUM FREE CONDITION

Aim of Study

We observed the overactive contractions of bladder muscle strips when extracellular Ca^{2+} was supplied after ATP stimulation under Ca^{2+} -free medium. It has been suggested that the depletion of intracellular calcium stores lead to the activation of secondary compensatory influx of Ca^{2+} across the plasma membrane. The role of purinergic contraction increases with age and/or an overactive bladder. Both inflammation and ishcemia are known to cause low intracellular Ca^{2+}. These findings prompted us to clarify the mechanism of the post re-perfusion of Ca2+ solution bladder overactivity.

Methods

Urinary bladders were obtained from Wistar rats at 24 weeks old. The bladder muscle strips 10x2 mm in size were made and transferred to10ml organ baths bubbled with 95%O_2 and 5%CO_2, filled with Krebs-Henseleit (K-H) solution. Changes in the force of contraction of the muscle strips were measured isometrically[1]. Calcium depletion was made by changing K-H to calcium free K-H and stimulated with 100 microM ATP. Then, the strip was washed with normal K-H. The strong post-perfusion contraction was modified with calcium mobilized antagonists and prostaglandin (PG) inhibitors.

Results

EGTA and okadaic acid (membrane phosphatase inhibitor) inhibited this post-perfusion contraction. However; H-7(protein kinase C inhibitor), ML-9 (myosin light chain kinase inhibitor), thapsigargin (intracellular calcium store Ca^{2+}-ATPase inhibitor) showed no effect on the contraction. Indomethacin and selective PG E2 antagonist showed an inhibitory effect, however PG E1 and F2arufa antagonists