**Short Report**

**Complete Prevention of Acetylcholine Induced Coronary Artery Spasm with Nifedipine**

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SHIMIZU, K., OGAWA, K.B., KATO, T., LEE, J-D., HARA, A. and NAKAMURA, T. Complete Prevention of Acetylcholine Induced Coronary Artery Spasm with Nifedipine. Tohoku J. exp. Med., 1988, 154 (1), 93-94 —— A 63-year-old woman has been known to have angina pectoris due to coronary artery (CA) spasm. An intra-left CA infusion of acetylcholine (ACh) provoked spasms of the left CA and an anginal attack. After daily oral administration of nifedipine (NIF) for 2 weeks, intra-CA re-infusion study of ACh did not provoke any CA spasms. This is the first report to demonstrate the complete prevention of ACh-induced CA spasm with nifedipine. ——— acetylcholine ; coronary artery ; spasm ; nifedipine ; vasospastic angina

**Case Report**

A wide variety of provocations can induce coronary artery spasm in susceptible patients, including stimuli such as hyperventilation, cold pressor and exercise, as well as agents such as ergonovine, histamine and acetylcholine (ACh) (Yasue et al. 1986 ; Hackett et al. 1987 ; Miwa et al. 1988). Recently intracoronary ACh infusion has been used as a powerful method for provoking coronary artery (CA) spasms, which have been diagnosed as vasospastic angina (Miwa et al. 1988). Furthermore, Yasue et al. (1986) postulated some possible role of the parasympathetic nervous system in the pathogenesis of a coronary artery spasm (Yasue et al. 1986). On the other hand, the administration of nifedipine (NIF), a powerful calcium channel blocking agent, is widely used for the prevention of CA spasms, and provides good results in the treatment of variant angina (Kimura and Kishida 1981).

We recently encountered a patient, whose CA spasm and anginal attack induced by intra-CA ACh administration anginal attack had been blocked completely by oral administration of NIF.

A 63-year-old woman, who had been diagnosed as having suscetable vasospastic angina pectoris, was admitted to our hospital for further examinations. The exercise stress test could not provoke any symptoms. During the exercise, electrocardiogram monitoring showed a transient left bundle branch block though the 201-thalium myocardial scintigram did not indicate any perfusion defects. The first cardiac catheterization including left ventriculography (LVG) and coronary arteriography (CAG) without medication was performed. Slight hypokinesis of the left ventricular wall was found in the apical area by LVG, but pressure measurements and routine CAG were normal. After routine CAG, 25 µg of ACh was infused into the left CA. After the infusion of ACh, an anginal attack was
provoked and left CA narrowings were found at segments 8 and 11 (Austen et al. 1975) by CAG (Fig. 1a). The patient was diagnosed as having vasospastic angina. Treatment with daily oral administration of nifedipine (NIF) (slow-release tablet, 20 mg b.i.d) was started, and the patient has been relieved from her anginal attack. Two weeks later, the second cardiac catheterization and CAG were performed under the administration of NIF. The patient was given a soft capsule of NIF (10 mg) 1 hr before the second CAG in addition to her daily nifedipine tablet. After control CAG, ACh was infused to the left CA in doses of 25 μg and 50 μg. However, even the latter dose of ACh was unable to provoke any chest symptoms or CA spasm (Fig. 1b).

This is the first report demonstrating the preventive efficacy of NIF against anginal attacks due, not only to spontaneous CA spasm, but also to spasm provoked by administration of ACh to the intra-CA. Though the effectiveness of NIF administration to the vasospastic angina is clearly demonstrated in this case report, the etiology of CA spasm and the relationship between the autonomic nervous system and calcium channels concerning CA spasm remains unclear. We look forward to further studies on vasospastic angina with great interest.

References