Case Report

Angina Pectoris Induced by Intravenous Administration of 50% Glucose

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SUMMARY

Frequent tracings of ECG during attacks of the nocturnal angina pectoris of a 69-year-old man failed to show any evidence of angina pectoris electrocardiographically. Angina pectoris was, however, induced by an intravenous administration of 50% glucose unexpectedly, and relieved by sublingual nitroglycerin and oxygen inhalation. The mechanism of this unusual phenomenon was discussed in relation to the serum-potassium, plasma-insulin, and the spasm of the coronary artery.

Additional Indexing Words:

Glucose load	Carbohydrate	Plasma-insulin	Serum potassium
ST segment	Nitroglycerin		

THE changes of ST segment and T wave associated with anginal attacks after glucose load have not been documented in literatures. We recently encountered a patient who suffered from occasional nocturnal angina pectoris over several years, and episodes of angina pectoris increased up to every night or every early morning without any evidence in electrocardiogram obtained during attack, but was both electrocardiographically and clinically diagnosed as true angina pectoris by an unexpected intravenous infusion of 60 ml of 50 per cent glucose solution for nutritional supply. This experience is so rare to force the following case report.

CASE REPORT

A 69-year-old man was admitted on April 13, 1968, because of frequent severe episodes of nocturnal precordial pain. He had experienced occasionally a nocturnal precordial pain of short duration for several years. He used to smoke 2 packs of cigarette and drink 2 liters of Japanese sake daily. The precordial pain occurred

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substernally and radiated left arm down, and subsided by sublingual nitroglycerin. Episodes of this pain increased up to every mid-night and/or every early morning since March of 1968. The electrocardiogram of routine 14 leads (standard 12 leads + V_{4R} and V_{3R}) and of the continuous monitoring of single precordial lead (C4) were recorded intensively, but failed to elucidate the nature of episodes electrocardiographically.

Physical findings on admission revealed small man with moderately developed musculature, tooth caries, and mild gingivitis. Physical examination of chest disclosed normal lungs and heart with normal size and no murmurs. Blood pressure was 110/70 mmHg. No abnormalities were confirmed in abdomen.

Laboratory tests on admission disclosed normal blood cell counts and normal differentials of white blood cell, erythrocyte sedimentation rate of 12 mm per 1 hour. Blood chemistry revealed almost normal including total cholesterol 194 mg/100 ml, GOT 25 R. F. Unit, GPT 8 R. F. Unit, LDH 317 Unit, and glucose 101 mg/100 ml. Serological tests disclosed negative STS, CRP negative and ASL-O less than 80 T. U. Urinalysis was negative, and specimens of stool was negative for occult blood reaction and parasites. Standard oral glucose tolerance test of 50 Gm glucose was normal. X-ray of chest disclosed normal size and configuration of the heart. Electrocardiogram at rest and during attack of chest pain disclosed only minimal but physiological elevation of ST segment in V_1 - V_6 (Fig. 1). In early evening of the 6th hospital day, 60 ml of 50 per cent glucose solution was administered intravenously

Fig. 1. Electrocardiogram at rest.

Fig. 2. Electrocardiogram during angina pectoris induced by intravenous administration of 50% glucose.

for nutritional supply because of poor appetite, but induced severe attack of substernal pain immediately and blood pressure was 130/80 mmHg during attack. ECG during this attack disclosed the elevation of ST segment in leads II and III with an increase of amplitude of R wave and reciprocal ST depression in I, V₃, V₄, and V₅, and occasional ventricular premature beats (Fig. 2). Lead V₆ could not be recorded during this attack. The present attack of chest pain and electrocardiographic alterations subsided soon after sublingual nitroglycerin and oxygen inhalation. Blood chemistry at the end of this attack revealed glucose 179 mg/100 ml, GOT 28 R.F. Unit, GPT 12 R.F. Unit, but serum electrolytes were not measured.

Patient was diagnosed as angina pectoris by this unexpectedly induced episode, and subsequently transferred into the oxygen tent with 30–40% oxygen, and was on 2 mg of sinthrom (anticoagulant), isosorbide-dinitrate 20 mg, and 1 Gm of chloramphenicol daily. Episodes of chest pain decreased rapidly by these intensive therapies, and was no more complained on the 18th hospital day, and thereafter the oxygen tent was removed, but no more typical episodes occurred. Patient discharged on May 16, 1968, because of his personal reason without doctor's permission. Therefore, further examinations such as coronary arteriography have not been performed. Patient has, however, been on isosorbide-dinitrate and dipyridamole, and doing well without episodes of angina pectoris any more.

DISCUSSION

Although alterations of ST segment and T wave after carbohydrate and glucose load have well been noted in not only persons with coronary heart disease but also normal persons (Simonson E, Keys A 1950,¹⁾ Chen T et al 1962,²⁾ Ostrander LD, Weinstein BJ 1954,³⁾ Kuramochi M et al 1973³⁾), these alterations were minor in degree, and not likely accompanied by episodes of angina pectoris. Therefore, our case of angina pectoris induced by an intravenous infusion of 50% glucose is quite unusual case. The mechanism of this rare phenomenon is not clear, but might be partially due to that of alterations in ST segment and T wave after meals and/or glucose load which is presumed to be the lowering serum potassium level relating to plasma insulin level (Dear HD et al 1969,⁵⁾ Passa P, Czvinet J 1972⁶⁾). Peters and Hales (1965)⁷⁾ measured plasma-glucose and insulin concentration during oral glucose tolerance test on a group of patients 6–17 months after myocardial infarction, and got results that the mean plasma-insulin level rose significantly from fasting to 150 min after glucose, and Nikkilä et al (1965)⁸⁾ confirmed that, in non-obese survivors of myocardial infarction, the response of immunoassayable plasma-insulin to oral glucose was abnormally high in 50% and subnormal in 10%, but the response to intravenous administration of glucose was within normal range. Dear et al (1969)⁵⁾ studied on the effect of standard glucose tolerance test on ST segment and T wave of ECG and serum potassium level, and confirmed the greater changes in group of persons with heart disease than in group of persons with diabetes mellitus and normal control.

Another possible mechanism of induction of anginal attack by glucose load might be related to an expansion of the circulating blood volume caused by infusion of high osmotic glucose solution which resulted in an increase of myocardial oxygen consumption, because anginal episode disappeared soon after inhalation of oxygen with sublingual nitroglycerin intake. This fact also suggests the possibility of the spasm of coronary artery by intravenous glucose load, because sublingual nitroglycerin was likely effective.

This rare case may warn us that an intravenous injection of high osmotic glucose solution to persons with coronary artery disease should be cautious.

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