A CASE OF JUVENILE ANGINA PECTORIS
PROBABLY DUE TO CONGENITAL SYPHILIS*

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CARDIOVASCULAR diseases due to congenital syphilis have been seen extremely rare\(^1,2\); it is considered that the most cases complicated cardiovascular disorders are dead in embryo or early infancy and syphilis itself is decreased today.

In the following case severe chest pain was the only symptom and sign, and there was no evidence of syphilitic aortitis (aortic insufficiency and aneurysma, etc) in usual physical and laboratory examination. It's syphilitic origin, coronary ostial narrowing and aortitis, could only be inferred by aortography and selective coronary arteriography.

CASE REPORT
A 29 year old male was admitted to the Nagoya University Hospital, Nagoya, Japan on March 17, 1970, because of chest pain. He gave no history of rheumatic fever or venereal disease. Sometimes vague discomfort in anterior chest occurred since 1967. Four months prior to admission severe chest pain developed. Its quality was constricting or burning and it was located in epigastrum and radiated to the neck. Chest pain was aggravated in course of time and precipitated on slow walking, eating and resting. After disappearance of anginal attack in about half one hour, anterior chest discomfort continued, and he sought hospital admission.

Family history is as Table I.

His sister said that father had venereal disease and she was diagnosed congenital syphilis and treated with penicillin during her first pregnancy.

TABLE I FAMILY HISTORY

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<th>mother</th>
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<th>father</th>
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<tr>
<td></td>
<td>68 year old</td>
<td>STS (2+~4+)</td>
<td>75 year old</td>
</tr>
<tr>
<td>female</td>
<td></td>
<td></td>
<td>male (patient)</td>
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<tr>
<td>34 year old</td>
<td>STS (1+~4+)</td>
<td>29 year old</td>
<td>STS (1+~3+)</td>
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<td>children 3</td>
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The 2nd Department of Internal Medicine, Nagoya University, School of Medicine, Nagoya
* An outline of this case was exhibited at the 34th Tokai Regional Meeting of the Japanese Circulation Society, in 1970.
On physical examination, the patient was a small man, weighing 49 kg, and 156 cm tall. His nose was low, but not saddled. Hutchinson's triad, rhagades, Argyll-Robertson's phenomenon, and mental retardation were not recognized. Ocular ground was normal. Blood pressure was 110/60 mmHg, pulse was 74/min and regular. On auscultation, there was a grade 2/6 early
systolic murmur in the left precordial area and the second sound was increased in 2LSB. Reflexes were all normal.

All laboratory data as follows:
1) Urine; protein (−) sugar (−) urobilinogen (n)
2) Blood: RBC 478 × 10⁶ Hb 96% WBC 9500 Hemogram: st: 5% N: 61% L: 33% M: 1%
3) Blood sedimentation rate: 6 (1 hour)
4) Biochemical examination of blood; protein fraction; total protein 7.7 mg/dl, Albumin 4.9 mg/dl, 63.2%, globulin α-1 0.3 g, 3.8%, α-2 0.7 g, 8.5%, β 0.8 g, 10.4%, γ 1.1 g, 14.2%, urea N 16.2 mg/dl, CCF (−), TTT 2+, GGT 27u, GPT 21u, LDH 235u, alkaline-phosphatase 6.7u, Na 138 mEq/L, K 4.2 mEq/L, Cl 96 mEq/L, lipid fraction; T.C 185 mg/dl F.C 50 mg/dl, P 190 mg/dl, T.L. 590 mg/dl TFA 306 mg/dl TG 117 mg/dl glucose tolerance test: before 82 mg/dl, 30' 141 mg/dl, 60' 145 mg/dl, 90' 122 mg/dl, 120' 90 mg/dl
5) Serologic test: for Syphilis; Wa-R (3+), FTA (3+), TPHA (1+) for Rheumatismus; CRP (−) ASLO (12u) RA (−)
6) Chest X-ray film: almost normal
7) Electrocardiogram (Fig. 1); there was slightly

ST depression in II, III, aVF at rest and after Master's single exercise ST depressions were remarkably in II, III, aVF and V₄,₅,₆, at the same time severe chest pain occurred (Fig. 1-a). During chest pain after breakfast, ST in leads II, III and aVF were depressed and T waves were taller in left precordial leads (Fig. 1-b).
8) Phonocardiogram: early systolic murmur was recorded at apex, and mid systolic murmur and accentuated IIa sound at 2LSB.
9) Cardiac output and circulatory volume: 6.824 L/min, 4.185 L in the supine position and 4.910 L/min, 4.133 L in the stand position.
10) Myocardial scincigram; almost normal.

In spite of the admission and treatment, chest pain developed frequently and its quality, duration and occurrence were variously (Fig. 2). ST-T changes in ECG demonstrated also two different type; ST depression in II, III, aVF occurred easily and continued longer, on the other hand, ST-T changes in V₄₋₆ appeared only during exercise and severe chest pain, and then its duration was relatively short.

In the previous present illness, family history and laboratory examination, both coronary ostial
narrowing probably due to congenital syphilis was suggested, and then aortography was two times performed. Coronary arteries were not almost visualized, however, there were convex defects of the sinus of Valsalva in RAO view and irregularity of the lumen above the aortic sinus in LAO view (Fig. 3-a, 3-b).

To ascertain the coronary disease, selective coronary arteriography was made. The coronary arteriograms showed a severe obstruction at the orifice of the left coronary artery and peripheral vessels were as a whole narrow and it's opacifications were then (Fig. 3-c, 3-d).

The right coronary artery showed the same change on the fluoroscopy.

During these procedures, patient complained severe chest pain and ST depression occurred more remarkably. The patient was attentively treated with small dosage of Aminobenzyl penicillin (750 mg/day) for 4 weeks, but quantitative blood STS were not so changeable. Moreover, procain penicillin G 400,000 units i.m. daily was administered for 2 weeks. A rise of STS titer

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A case of juvenile angina pectoris

Aortography demonstrated convex defect of the lumen of the aortic origin and irregularity above the aortic sinus though the former finding is not entirely characteristic in the luetic aortitis. Both coronary artery was not revealed except thin opacification considered probably left main coronary trunk.

Coronary arteriography was performed by the use of Sone's catheter. The obtained coronary arteriogram was not so clearly visualized except severe coronary ostial stenosis.

These observation was suggested that only a little contrast medium could be perfused into the coronary artery through ostial stenosis.

The presence of this obstructive lesion could be inferred from chest pain attack and remarkable ST depression when the catheter was inserted.

However, it is not confirmed whether the peripheral coronary vessels are intact because of reports of peripheral vascular involvements of syphilis.

This patient has been treated with penicillin. STS titers were low at first, therefore, it was imagined that evaluation of the curative effect was difficult, but STS titers were elevated with the treatment.

This phenomenon is probably considered the provocation of latent syphilis with penicillin treatment and seems also to be strong evidence that anginal syndrome is due to syphilitic origin.

Surgical repair is necessary in this case because severe chest pain occurs frequently in spite of resting and internal treatments.

**SUMMARY**

A case of juvenile angina pectoris probably due to congenital syphilis was reported.

Both coronary ostial narrowing could be only presumed in usual physical and laboratory examination. And then aortitis and both coronary ostial stenosis demonstrated in vivo by aortography and selective coronary arteriography.

The progress of this case has been observed at present with internal treatments, but surgical repair will be necessary in the near future.

**REFERENCES**

3. MASTER, M. et al.: The electrocardiogram and