CASE REPORTS

Aorto—Left Atrial Fistula

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Published reports of aorto—left atrial fistula are exceedingly rare, and it is hoped that a report of this interesting case will draw further attention to a condition which has been treated successfully by surgical repair (Beck, Schrire, and Barnard, 1964). Rupture of the sinus of Valsalva into the left atrium has also been reported once (Kay et al., 1959).

Case Report

A Chinese man, aged 55 years, was admitted to hospital in January 1964, complaining of increasing shortness of breath on exertion, of about two months' duration. He had had some retrosternal chest discomfort but this was never severe, though it increased in severity following exertion. His face was swollen in the mornings and this swelling decreased towards the end of the day. In the past 10 years he had been prone to asthmatic attacks but he had been free from this complaint for five years before admission. He admitted exposure to venereal disease “when young” but had not been aware of any lesions following exposure.

On examination he was found to be orthopnoeic, with a regular pulse rate of 88 a minute and a blood pressure of 110/60 mm. Hg. His jugular veins were very engorged, weakly pulsatile, and the upper level was not discernible. The veins in the front of the chest and over the forehead were dilated. The eyelids were puffy. There was no anaemia or jaundice.

Over the praecordium, no thrills or abnormal pulsations were felt. The apex beat was only faintly palpable and was located in the fifth left intercostal space just outside the left mid-clavicular line. Both heart sounds were heard in all areas and the pulmonary second sound was split. Over the aortic area an ejection systolic murmur (grade 2/6) was heard, and a soft early diastolic murmur was heard to the left of the edge of the sternum. The lower border of the liver was four fingerbreadths below the costal margin. There was pitting oedema over the dorsa of both feet and in the pretibial areas. No other abnormalities were present.

On investigation the blood Kahn and VDRL tests were positive. The ESR was raised to 27 mm. (Westergren) in the first hour. The x-ray film of the chest showed some enlargement of the heart and a generally dilated ascending aorta. The radiologist who screened the chest reported that the left atrium was slightly enlarged but not sufficient to displace the barium-filled oesophagus. The cardiographic tracing showed early left ventricular preponderance.

The patient showed improvement with bed-rest, diuretics, and a course of penicillin injections. He was discharged and followed up in the clinic. In April 1964, he complained of having coughed up blood and also of increasing dyspnea, and was readmitted. He was in heart failure, and on auscultation a continuous murmur was heard over the back of the left chest.

Radiological examinations, however, did not yield added information. Cardiac catheterization was not done, because of the lack of such facilities at the time. He remained in hospital until his death eight months later. Three days before his death he complained of sudden abdominal pain after the evening meal. No diagnostic signs were found on examining the abdomen. The following morning there was abdominal distension, with continuous abdominal pain. Mesenteric infarction was suspected, but surgical intervention was not attempted because of his poor condition. He died on the following day.

At necropsy the heart (640 g.) showed enlargement, mainly of the left side. Immediately above the aortic cusps, but not involving the sinus of Valsalva or the coronary orifices, there was a saccular aneurysm 5-5 cm. in diameter, abutting both atria. The superior vena cava was also compressed. The aortic opening of the aneurysm measured 3-5 cm. in average diameter (Fig. 1). The aneurysm was bulging into the left atrium and communicated with the left atrial chamber through an orifice 0-5 cm. in average diameter, the edges of which were smooth and rounded. On the lateral wall of the left atrium opposite this aperture there was an area of endocardial thickening 3-3 cm. in diameter—a “jet stream endocarditis” (Fig. 2). The aortic and mitral valves
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Orifice of aneurysm communicating with ascending aorta

**FIG. 1.—**Left ventricle and ascending aorta showing the orifice of the aneurysm.

"Jet-stream endocarditis"

Orifice in aneurysm leading to left atrium

**FIG. 2.—**Left side of heart showing a probe through the aneurysm of the aorta into the left atrium, and a pointer to the area of "jet-stream endocarditis".
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were normal. The aortic intima was “wrinkled” in patches.

The entire length of the gastro-intestinal tract was grossly edematous and congested, but no occlusion was demonstrated in the gastric and mesenteric blood vessels. The spleen, too, showed areas of recent infarction.

Comment
This patient showed several interesting features. Since the rupture of the aneurysm did not cause pain, it probably occurred gradually. The adherent aortic and left atrial walls could have been eroded through, as opposed to a sudden rupture as in the case of Beck et al. (1964). These authors suggested that a “fistulous” murmur heard at the back of the chest was a diagnostic feature of this rare lesion, and the present case confirms this. Antemortem diagnosis was not possible in this case, because the aneurysm was small and concealed by both atrial chambers, and hence not demonstrated radiologically. In addition, the significance of the continuous murmur at the back of the chest was not appreciated. He lived for nearly a year with the shunt.

The patient died of massive infarction of the gastro-intestinal tract without any occlusive vascular lesions. This mode of death has been recorded earlier in association with aortic valvular insufficiency (Hoffman, Zimmerman, and Cardwell, 1960). In this patient, since the diastolic pressure was never lower than 60 mm. Hg, the aorto-atrial shunt is the significant factor. In the absence of occlusive vascular disease the gastro-intestinal infarction is the result of prolonged mesenteric vascular insufficiency (he had severe, generalized, abdominal pain for over 48 hours before death). Corday et al. (1962) have demonstrated experimentally an angiospasm of the arteriolar bed of the gastro-intestinal tract, when the blood pressure or cardiac output is reduced, to explain such pathological changes.

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References
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