Spontaneously Disappearing Murmur of Patent Ductus Arteriosus

A Case Report

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In 1903, Wagener reported two cases of patent ductus arteriosus with a perforated valve in the lumen of the ductus. Taussig has seen two cases of patent ductus arteriosus with such a valve in the lumen of the ductus. The disappearance of a murmur in patients who previously had an audible bruit has been mentioned by Levine and Harvey and is known also to occur in a number of conditions. Levine and Harvey in their book on auscultation, stated, "In some cases, the continuous murmur is intermittent and its presence or disappearance suggests fluctuation of the level of pulmonary hypertension," but they do not give any additional information about their experience with this type of case. Shapiro et al. recently reported a case of intermittent disappearance of a murmur of patent ductus arteriosus, and they explained its disappearance by mediastinal shifting. Their patient had normal pressures in the pulmonary circuit so that, obviously, changes in the pressure gradient across the ductus could not explain the unusual auscultatory findings.

We present a case of a girl with a murmur of patent ductus arteriosus, discovered at age 10, with no evidence of pulmonary hypertension and in whom the murmur disappeared intermittently for no apparent reason. At surgery, the ductus was seen to contain a valve proximal to the pulmonary artery end, and this valve was thought to be responsible for the unusual auscultatory findings of this case.

S.Z., a 10-year-old white girl had a normal birth and development. Growth was somewhat slow but she was thoroughly normal otherwise. Incidence of respiratory infections was not high. Six months prior to admission, a heart murmur was discovered in the course of a routine physical examination. The patient was completely asymptomatic both before and after the detection of the murmur.

On physical examination, the patient was found to be somewhat underdeveloped. The positive findings were limited to the thorax. The heart rate was 100 and regular. The apical impulse was located at the fifth interspace in the midclavicular line. The mitral first sound was loud, and the aortic second sound was normal. At the pulmonary area, a continuous, machinery type of murmur, grade III to IV, was heard that obliterated the second heart sound. Both the systolic and the diastolic components of the murmur were well transmitted to the left side of the neck and with diminishing intensity to the right side; it was also transmitted to the apex. The phonocardiogram was typical of the condition (fig. 1). The blood pressure was 110/60 mm. Hg in the right arm and 114/64 in the left arm. Femoral and pedal pulses were palpable.

Roentgenograms demonstrated one- to two-plus prominence of the pulmonary conus segment, with slight elongation of the left border on the postero-anterior view of the chest. The electrocardiogram was interpreted as showing "a horizontal position, and suggestive of left ventricular preponderance without evidence of hypertrophy or strain," (fig. 2). Right heart catheterization was recommended but was refused.

The patient was admitted to the Passaic General Hospital for operation. Examination on admission, however, to the amazement of all, revealed no murmur! Several observers were present, one of whom had examined the patient when the murmur was audible. The patient was exercised, placed in various positions, made to cough,
and to perform a Valsalva maneuver, but no murmur was audible. She was immediately sent in a wheelchair for a phonocardiogram, but by the time she arrived in the cardiac station, the murmur was audible again!

At this point, we tried all conceivable maneuvers to make the murmur disappear, but these also were unsuccessful. The patient was carefully observed but nothing happened until the next morning when the murmur again disappeared. Again we were unable to document with a phonocardiographic tracing the disappearance of the murmur, for it was very audible after 5 minutes of silence.

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Permission for right heart catheterization was again refused, and the referring physician insisted on going ahead with the operation.

On April 17, 1957, the patient was operated upon by Dr. Maxwell Chamberlain. A patent ductus arteriosus, 2 cm. in length and 1 cm. in diameter, which extended from the aorta to the bifurcation of the pulmonary artery, was found just below the subclavian artery. After the ductus was divided, it was seen to contain a valve, or veil-like structure, at the pulmonary artery end of the ductus. It was thought that this valve could account for the unusual auscultatory features of the patient, for it was easy to occlude the whole ductal lumen with the valve.

Discussion

In an extensive review of the literature we have been unsuccessful in finding a report of a similar case. The murmurs of patent ductus arteriosus are usually typical but the small percentage that are atypical usually occur in infants and children and are thought due to pulmonary hypertension, low systemic pressure, very small diameter of ductus, spontaneous closure of ductus, and shifting of mediastinal structure with occlusion of ductus. Clinically, our findings were similar to those of shifting mediastinum but the operative findings were entirely different. We consider it worthwhile to bring to the attention of the medical profession a case in which a murmur, loud and typical of a clinical condition (patent ductus arteriosus), became suddenly inaudible. In our patient the murmur was first heard at the age of 10 years and by the same pediatrician who had seen her after birth and who had taken care of her all her minor illnesses of childhood. One may theorize that the ductus was closed by the valve found at surgery until the age of 10, at which time the valve split open. Only after the patient had been operated upon and the unusual pathologic structure of the ductus was known were we able to offer a satisfactory explanation for the auscultatory findings.

We have reason to believe that the pressures in the pulmonary circuit were intermittently elevated in this case, which might explain closure of the valve. There was no evidence of pulmonary hypertension in the auscultatory, roentgenologic, or electrocardiographic findings.

This, no doubt, was a case of ductus arteriosus with a left-to-right shunt exclusively, and this shunt at times completely disappeared, probably as a result of the sudden closure of the ductus by the valve. Unfortunately, no catheterization was possible in this patient. It was thought, however, that the case presented enough clinical interest to warrant a report of our findings. It is true that Taussig wrote about two patients with patent ductus arteriosus in which a valve was present at the pulmonary artery side of the ductus, and in this respect our case is similar to theirs. The spontaneous disappearance and later reappearance of an otherwise typical, loud murmur is in itself interesting. In this case the murmur, a so-called machinery murmur, was typical of patent ductus arteriosus; so were the other findings. In the two cases of Taussig, we are not told whether these patients had any unusual auscultatory features, although it is speculated there that certain cases of sudden appearance of a typical murmur of patent ductus arteriosus could be due to similar pathology.

Summary

A 10-year-old girl with patent ductus arteriosus had most unusual auscultatory changes, but the typical roentgenologic and electrocardiographic findings. There was an intermittent, spontaneous disappearance and reappearance of the entire machinery murmur. At surgery, a valve, or veil-like structure, was found inside the ductus at its entrance into the pulmonary artery. This valve apparently accounted for the "spontaneous" intermittent disappearance of the murmur, which was first discovered when the child was 10 years old.

References


The physician without physiology and chemistry practices a sort of popgun pharmacy, hitting now the malady and again the patient, he himself not knowing which.—Sir William Osler. Aphorisms From His Bedside Teachings and Writings. Edited by William Bennett Bean, M.D. New York, Henry Schuman, Inc., 1950, p. 49.
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