Patent Ductus Arteriosus

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Summary

A 10 month old female mixed breed dog was presented to the ISU Veterinary Clinic for an ovariohysterectomy. A patent ductus arteriosus was diagnosed and surgically corrected. Aortic pressures and angiocardiographic studies were also performed.

Introduction

Patent ductus arteriosus (PDA) is the most common congenital cardiac defect seen in the canine. It has been determined that approximately 0.6% of the dogs seen in veterinary school clinics will present some form of congenital cardiac disease and more than 30% of these will be PDA's. The defect is twice as common in females as males and is most often seen in purebred dogs, especially in Poodles, Collies, Pomeranians, and Shetland Sheepdogs.[4]

Recognition of PDA is important for several reasons. Since it is usually detected in purebred pups, newly acquired animals could presumably be returned. Early detection is important to ascertain the extent of cardiac compensation for the defect and the feasibility of surgical correction. From the surgical standpoint, the earliest possible detection involves the least risk and the best prognosis. Since PDA is an inheritable defect in some breeds, perspective breeders should be made aware of its existence.[2]

PDA is a persistent terminal portion of the sixth aortic arch. Prenatally the ductus arteriosus is a short arterial connection bypassing blood from the pulmonary artery to the distal aorta and systemic circulation, thereby circumventing the nonfunctional lungs. Normally at birth with the inflation of the lungs, the resistance (in the pulmonary arteries) drops. Simultaneously the left ventricle assumes the role of the major pumping chamber surpassing the right ventricular pressure. As a consequence to these changes, the fetal circulation assumes adult characteristics. The blood flow across the ductus arteriosus in the normal heart will stop at birth or shortly after, and the ligamentum arteriosus will form. If the ductus fails to close for some reason, it becomes a patent ductus arteriosus (Fig. 1).

With a PDA the rising postnatal presure in the aorta surpasses the pulmonary artery pressure, reversing the flow of blood across the ductus. This reversal causes an arterial-venous (A-V) shunting in both systole and diastole, decreasing the total output in systemic circulation. This type of A-V shunt or fistula is the most common in dogs. Compensatory increases in blood volume and increases in left ventricular output are required to maintain normal systemic cardiac output. Typically on auscultation an accentuated late systolic and early diastolic murmur is heard and termed a continuous or machinery murmur. Pulmonary arterial pressure should always be greater than right ventricular pressure in this condition. In severely compensated hearts, pulmonary hypertension can develop, and only a systolic murmur occurs since the left-to-right shunt is limited to the systolic phase. When the pulmonary arterial pressure is equal to or exceeds aortic pressure, reverse (right to left) shunting or bidirectional shunting occurs. In this case there may be no murmur heard at all, but a split second heart sound may be recognized.[2]

The defective closure of the ductus is a graded phenomenon. A partial closing, called a ductus diverticulum, can be recognized only with angiocardiography. In this condition there is no shunting nor auscultatable

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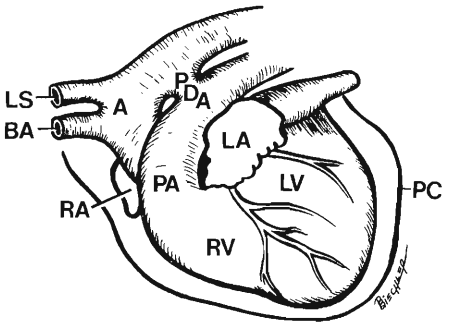

Fig. 1: Diagramatic representation of Patent Ductus Arteriosum. Legend: LS, Left Subclavian Artery; BA, Brachiocephalic Artery; A, Aorta; PDA, Patent Ductus Arteriosum; PA, Pulmonary Artery; RV, Right Ventricle; RA, Right Atrium; LA, Left Atrium; LV, Left Ventricle; PC, Pericardium.

murmurs. The patency may vary from a small channel to the size of the aorta. Usually the larger the PDA shunt the more severe will be the clinical manifestations. It has been determined that, whether the dog has a clinically or nonclinically apparent PDA, the defect will present itself in equal inheritance in the offspring.[4]

Case Report

On February 9, 1978, a 15 lb., ten-month-old female mixed breed dog was presented to the ISU Veterinary Teaching Hospital for an ovariohysterectomy (OHE). The owner was worried that the dog may have been bred two weeks before. No further pertinent history was given. During the admitting examination, ascultation revealed an abnormally loud first heart sound and a systolic murmur over the mitral area. Moving cranially over the pulmonic area, a diastolic murmur blended with the systolic murmur, producing a continuous or machinery murmur. The dog was hyperactive and with minimal excitement the murmur became easily identifiable over the entire heart field.

A lateral radiograph revealed generalized cardiomegaly with rounded right and left borders. The cardiac shadow was spread over four intercostal spaces. Elevation of the trachea was seen throughout its entire thoracic course. In a dorsoventral radiograph an aneurysmic bulge on the aortic shadow and slight bulging of the left atrial border were seen. From both views it was determined that the pulmonary arteries were somewhat enlarged, but that no alveolar changes in the lung field were evident.

An EKG revealed high amplitude R waves and deep Q waves. The heightened R wave (3½ mv) was present with a normal mean electrical axis, which indicated left ventricular enlargement. The deep Q wave suggested right ventricular enlargement.

Radiographic and electrocardiographic studies were compatible and confirmative of the initial PDA diagnosis. Because the dog presented no clinical signs indicative of heart disease and the compensated heart showed no arrhythmic activity, it was determined that the dog would be a fair to good surgical risk. The stresses of an OHE or of parturition would probably be too great for the dog to survive without corrective cardiac surgery. Without any surgery the life expectancy was estimated to be a maximum of 3 years. The owner elected to donate the dog to ISU.

On February 13, 1978, angiocardiography and cardiac surgery were performed. Aortic pressure and pulse were recorded throughout both procedures. The dog was not given any premedication prior to anesthesia. Atropine, it was felt, would inactivate the possibility of cardiac response in the event of rapidly increasing blood pressure. Barbiturates were avoided because they often produce ventricular arrhythmias in normal dogs. The dog was masked down with halothane and nitrous oxide and intubated.

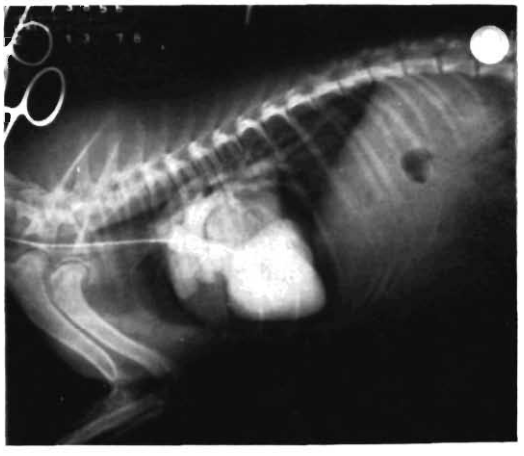

Fig. 2: Angiogram showing shunting of contrast media from the aorta to the pulmonary artery.

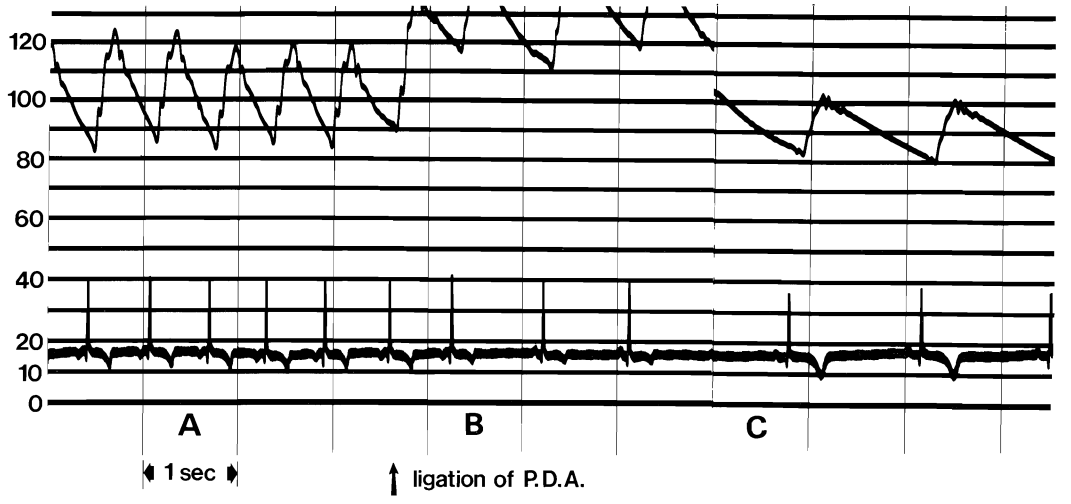

Fig. 3: Aortic blood pressure and Lead II Electrocardiogram before (A), immediately after (B) and 30 minutes following (C) ligation of PDA.

A #5 French multihole radiopaque catheter was introduced into the left carotid artery after the area had been prepared. Using image intensification the catheter was retrograded through the brachiocephalic artery and the aorta into the left ventricle. During this procedure aortic and ventricular pressures were monitored on a VR-6 recorder (Electronics for Medicine). The pressures were 78/42 mm Hg (normal 107/86 mm Hg) and 80/10 mm Hg (normal 122/41 mm Hg) respectively. The left ventricular end diastolic pressure was recorded at 30 mm Hg, which was considered high and approaching left heart failure.

With the catheter placed in the left ventricle, contrast medium (Hypaque M-75,® Winthrop) was rapidly injected with a CO_2 powered syringe. Immediately after the contrast medium was injected, six radiographs were taken with a rapid cassette changer at an exposure rate of two per second. After initial rapid accumulation of the contrast in the left ventricle, a marked reflux was noted into the left atrium. This regurgitation was indicative of mitral insufficiency. The left atrium and left ventricle were markedly dilated. At the level of the aortic aneurysm the PDA was identified and estimated to be ½ cm. in diameter (Fig. 2). With passage of the contrast medium across the PDA, the main pulmonary artery was seen to be markedly dilated, with the pulmonary segment being approximately twice the size of the aorta.

During surgery the catheter was left in the aorta and pressures and pulses were continually monitored. After surgical preparation, the left thorax was incised from the wing of the scapula to the costochondral junction. The thorax was entered through the left fourth intercostal space. After adequate positive pressure ventilation was attained, aortic pulse pressure was recorded to be 35 mm Hg. The approach to the pericardium was made by reflecting the lung lobes caudally. At this point the PDA was localized both visually and by palpation. The pericardium was carefully incised between the left vagus and phrenic nerves. These two nerves were retracted from the surgical field with 3-0 silk traction sutures. After dissecting around the caudal and ventral aspects of the PDA, four strands of 3-0 silk were placed under the PDA, carefully avoiding the left recurrent laryngeal nerve. Immediately after closure of the first strand of silk, the aortic pressure rose abruptly to 150/110 and the heart rate dropped from 110 to 50 beats per minute (Fig. 3). After the remaining three strands were securely ligated, a Foley catheter was placed in the chest for post-operative drainage. The nerves were released and the lung lobes were returned to their normal positions and inflated. The thoracotomy incision was closed routinely. Before the aortic catheter was removed, about 60 minutes after the PDA ligation, the pressure had dropped to 100/80 mm Hg and the pulse pressure was 20 mm Hg.[1]

Postsurgical auscultation of the heart revealed a slight systolic murmur over the mitral valve. It was felt that this would disappear with time as the left atrioventricular annulus reduced in size. The Foley catheter was removed 12 hours postoperatively, having removed 50 cc. of fluid over this time. The dog was given a good prognosis for a normal life span.

Conclusion

The necessity for a thorough presurgical physical examination was particularly emphasized in this case. Physical examination, combined with radiography and electrocardiography, is usually sufficient to confirm a PDA and establish its surgical potential. Although angiocardiography is not necessary to PDA diagnosis, it would be necessary to diagnose non-clinical cases, those without machinery murmurs or with partial closing (ductus diverticulum). Angiocardiography can also be useful in determining associated congenital or secondary effects of a PDA, such as mitral insufficiency.

Since PDA is the most common congenital heart disease seen in young dogs, its potential presence should not be overlooked. In most cases it is surgically treatable with a high degree of success. More importantly it guarantees the affected dog a chance of a full life span. Since surgical correction is feasible in many cardiovascular cases, case referrals to ISU are requested. When such cases arise Dr. D. H. Riedesel or Dr. D. W. DeYoung can be contacted at (515) 294-4900.

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