Persistent Truncus Arteriosus in a Calf

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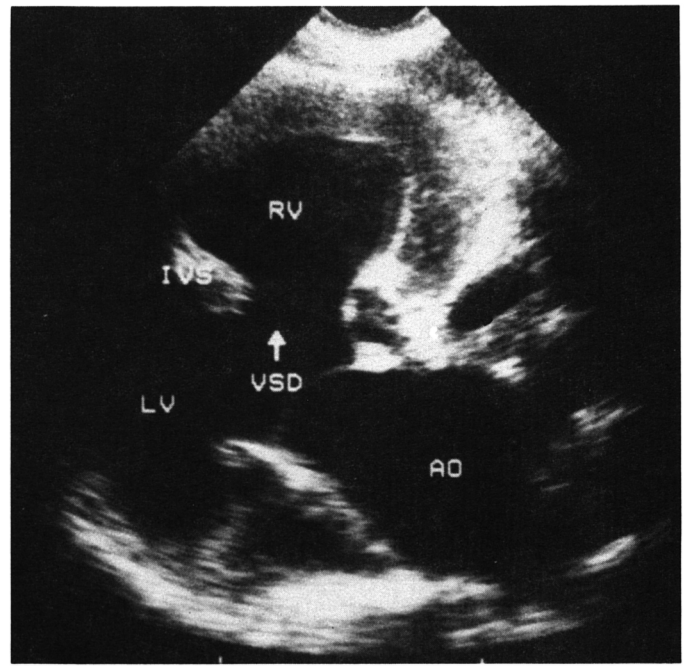
A 5 week old 45 kg Hereford bull calf was referred to the Veterinary Teaching Hospital, U.C. Davis, because of pneumonia unresponsive to antibiotic treatment. It presented with exercise intolerance and fever. The last treatment given before admission included the antibiotic, ceftiofur, and the antipyretic, dipyrone, that morning. The bull was suckling his dam and had access to alfalfa hay.

Initial physical examination revealed the calf to be well muscled, bright, alert, and responsive. The mucous membranes were pink, but the calf became cyanotic when exercised. Auscultation revealed increased inspiratory sounds bilaterally over the lungs and a continuous palpable murmur loudest during systole, and heard equally over the left and right heart bases. Resting respiratory rate was 45 breaths/min, and the heart rate was 126 beats/min.

A hemogram showed a marked leukocytosis (27,500/ul; normal range 4,000-12,000/ul) with a neutrophilia (13,475/ul; normal range 600-4,000/ul), increased bands (550/ul; normal range 0-120/ul), a marked monocytosis (9,075/ul; normal range 25-850/ul), and a markedly elevated plasma fibrinogen (1,700 mg/dl; normal 300-700mg/dl). A chemistry panel revealed the calf to have a metabolic acidosis (total CO2) 10mmol/L; normal 19-34mmol/L), increased chloride (112mmol/L; normal 93-103 mmol/L), increased creatinine (2.9mg/dl; normal .9-1.3mg/dl), and an increased urea nitrogen (46mg/dl; normal 8-23mg/dl). Venous blood pH was low (7.244; normal 7.31-7.53) due to the metabolic acidosis, and arterial PO2 was markedly low (55.1mmHg; normal 88-104mmHg). All other chemistry and hematologic values were normal.

A tentative diagnosis of congenital heart defect with a secondary bacterial infection was made. Radiographs showed an enlarged right heart and general cardiomegaly. Ultrasound was performed which confirmed right ventricular hypertrophy, and showed a ventricular septal defect with an overriding and enlarged aorta (fig. 1). A right-to-left ventricular shunt was demonstrated by contrast echocardiology. An injection of contrast (saline and air bubbles) into the jugular vein showed that blood entering the right ventricle was exiting directly through the aorta, with some mixing in the left ventricle. No contrast was seen leaving via a pulmonary artery. A diagnosis of conotruncal defect, either a tetralogy of Fallot or persistent truncus arteriosus, was then made. After the owner was given a grave prognosis, she donated the calf for teaching purposes and euthanasia.

Figure 1



To better confirm the diagnosis and differentiate between tetralogy of Fallot and persistent truncus arteriosus, an angiographic study was performed. No contrast material entered the pulmonary artery from the heart, but there was a communication between the pulmonary artery and the aorta above the heart base (fig. 2,3). This confirmed the diagnosis of persistent truncus arteriosus.

Due to continuous fever (103-105° F), intermittently depressed attitude, and decreased appetite, sulfadimethoxine was administered to the calf starting on the second day of admission. For the remainder of the stay at the hospital, the calf continued to be febrile even in the face of antimicrobial therapy, and attitude and appetite varied from fair to good.

The calf was euthanatized and necropsy revealed bronchopneumonia and multifocal lesions in the kidney suggestive of septic nephritis. Culture yielded *E. coli* from the kidney, with no bacterial growth from the lung. Persistent truncus arteriosus was confirmed post mortem.

The pattern of congenital cardiac disease varies with

Figure 2

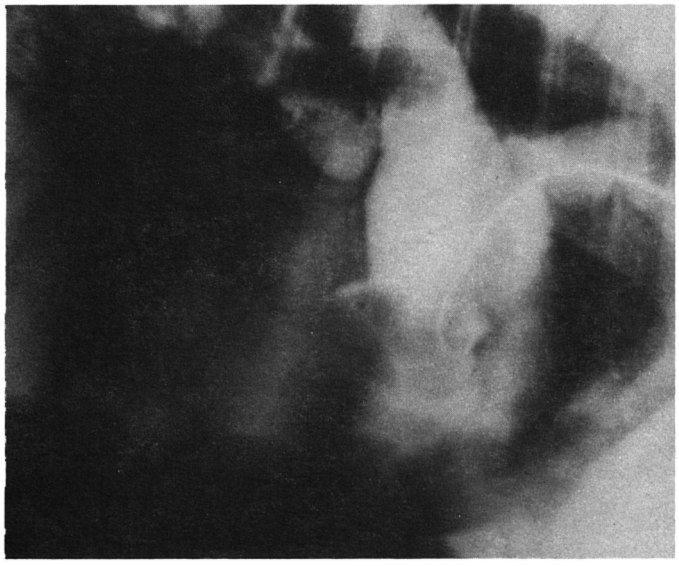
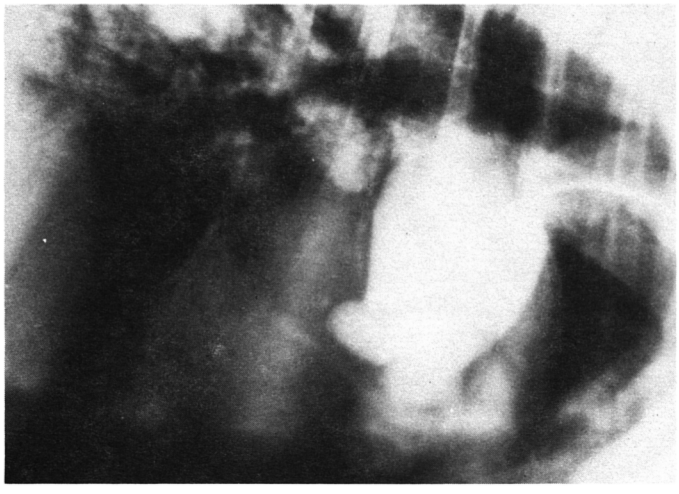


Figure 3



different species. In dogs, patent ductus arteriosus, pulmonic stenosis, subaortic stenosis and persistent right aortic arch are the most common findings (1). In cats, congenital mitral insufficiency and endocardial cushion defects are more frequently seen (1). Congenital cardiac anomalies are relatively rare in the horse (1). In cattle, atrial and ventricular septal defects and transpositions of the main vessels are the most common congenital cardiac defects (1,2,3). This calf demonstrated a persistent truncus arteriosus, which is relatively rare, especially in cattle (2,3).

Persistent truncus arteriosus is a congenital defect which involves failure of the embryonic truncus arteriosus (the single primitive outflow tract of the heart) to fully divide into an aorta and a pulmonary artery. The ventricular septum also fails to fully develop and does not complete close to separate the left and right ventricles. This results in both ventricles opening into a common trunk straddling the septal defect which leads into an enlarged aorta. The pulmonary arteries branch directly from the aorta distal to the heart. Persistent truncus arteriosus differs from the relatively more common tetralogy of Fallot. Tetralogy also has right ventricular hypertrophy, dextro-position of the aorta, and ventricular septal defect, but the aorta and pulmonary vessel do separate, and the pulmonary artery is anatomically distinct, yet often quite stenotic.

Tissue oxygenation is often compromised in patients with congenital heart defects such as persistent truncus arteriosus. In this case, the majority of venous blood is returned to the systemic circulation, with only a small portion going to the lungs via the narrow pulmonary branches off the common aortic trunk. Due to mixing of arterial and venous blood through the inrtraventricular septum, along with inadequate oxygenation of the blood, efficiency of circulation is reduced to the point that any oxygen stress can result in clinical signs of cyanosis and/or exercise intolerance. Because the right ventricle is now pumping blood directly into the systemic circulation, hypertrophy of the muscular walls develop which was observed in this case. Although not observed in this case, polycythemia (increased number of red blood cells) often develops in these types of anomalies in an attempt to compensate for decreased oxygenation of tissues.

References

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Editor's Note: Hans Konrad was awarded $50 for his student clinical paper.