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Case Reports

Enlarged and Echogenic Kidneys While on a Pediatric Ventricular Assist Device

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Abstract: Pediatric ventricular assist devices (VAD) specially designed for small children have been increasingly implanted in North America. We present unexpected renal findings developing in three children receiving circulatory support using VAD (EXCOR Pediatric Ventricular Assist Device; Berlin Heart Inc., The Woodlands, TX). The mechanisms of heart failure in these children included: 1) hypoplastic ventricle with aortic stenosis; 2) anomalous coronary artery arising from the pulmonary artery; and 3) idiopathic progressive heart failure. During circulatory support with VAD, each child had coagulation times maintained at 1.5–2.5 times normal. Each child had normal or mildly reduced renal function. Each of these children had imaging studies (ultrasound or computed tomography) while receiving VAD support, which demonstrated enlarged echogenic kidneys. These children had coagulation parameters within recommended ranges. These children subsequently died and autopsy studies of two children showed multiple bilateral kidney infracts, parenchymal congestion, and arterial thrombosis. Our case series shows that enlarged and echogenic kidneys may be associated with thromboembolism of the renal artery in pediatric patients on VAD. We were not able to identify a specific cause-and-effect relationship. **Keywords:** ventricular assist device; enlarged kidneys; echogenic kidneys. *JECT. 2013;45:248–250*

In the last decade, there has been an increase in use of pediatric mechanical circulatory support using ventricular assist devices (VAD). Mechanical circulatory support has been used for two primary purposes: 1) bridge-to-transplant and 2) bridge-to-recovery (1,2). Pediatric ventricular assist devices (EXCOR Pediatric Ventricular Assist Device; Berlin Heart Inc., The Woodlands, TX), specially designed for small children, have been increasingly implanted in North America (3). Prospective trials have shown pediatric VAD improves the survival of patients on heart transplant lists and has superior survival outcome compared with extracorporeal membrane oxygenation (ECMO) (4,5). Although this therapy improves the survival and quality of life among pediatric patients with heart failure, the device also carries the risk of several complications. We present previously unreported renal findings in three patients who were receiving pediatric VAD support using the EXCOR Pediatric Ventricular Assist Device.

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CASE HISTORY

Patient A

A baby boy born at 38 weeks of gestation was diagnosed prenatally with aortic stenosis and a hypoplastic left heart. A postnatal echocardiogram showed critical aortic valve stenosis, bicommissural aortic valve, and a hypoplastic left ventricle with severe left ventricle dysfunction. On Day 11 of life, the baby underwent aortic coarctectomy, reconstruction of the aortic arch, aortic valvotomy, and closure of an atrial septal defect. He had severe left ventricular diastolic dysfunction and left atrial hypertension contributing to worsening right ventricular failure refractory to surgical correction. He underwent VAD implantation on Day 58 of life.

The Pediatric Nephrology Service was consulted at Day 68 of life, Day 10 on VAD because of enlarged echogenic

kidneys. Doppler showed normal blood flow to both kidneys. A baseline renal ultrasound (US) at Day 4 of life showed a right kidney size 4.2 × 1.7 × 2.4 cm, a left kidney size 4.7 × 2.5 × 2.4 cm, and normal echogenicity and blood flow. Renal function was normal with serum creatinine .27 mg/dL. The patient was receiving heparin anticoagulation with prothrombin time PT in seconds of 13.5, partial thromboplastin time (PTT) in seconds of 68, international normalized ratio (INR) of 1.1, and fibrin degradation products (d-dimer) in micrograms per liter of 5.5. Summary of kidney size for Patient A is shown Table 1. The infant died at Day 138 of life, Day 84 on VAD. The cause of death at autopsy was attributed to hypoplastic left heart syndrome, systemic aortic stenosis, left ventricular dysfunction, and severe end-stage pulmonary hypertension. Autopsy gross examination of the kidneys showed lobular cortex with indistinct corticomedullary borders. Microscopic examination showed congested renal parenchyma with acute tubular necrosis, dilated renal tubules, and atrophic glomeruli. There was no evidence of thromboemboli in kidney vessels.

Patient B

A baby girl born at 38 weeks of gestation was admitted to the hospital on Day 6 of life as a result of respiratory distress. She was found to have an anomalous left coronary artery arising from the pulmonary artery. She underwent surgical repair at 3 weeks of life. She presented at Day 110 of life as a result of decompensated heart failure and respiratory failure. She underwent VAD implantation on Day 118 of life. The pediatric nephrology service was consulted at Day 128 of life, Day 10 of VAD, because of rising serum creatinine levels (from .3 mg/dL to 1.39 mg/dL over 3 days) and abnormal kidney perfusion on the computed tomogram scan of the abdomen and hypertension. Baseline renal US at Day 11 on VAD showed the right kidney 6 × 3.2 × 2.3 cm and the left kidney 6 × 2.9 × 2.6 cm. The patient was receiving heparin anticoagulation with PT 21.5, PTT 122, INR 1.1, and d-dimer 5.2. A summary of kidney size for Patient B is shown in Table 1.

Patient B died at Day 155 of life, Day 37 on VAD. The cause of death was attributed to the result of severe left ventricular dysfunction, bacteremia with overwhelming sepsis, and pneumonia. Autopsy findings showed kidneys with multiple bilateral infarcts with large right renal artery thrombosis and focal hemorrhage of remaining viable tissue.

Patient C

A baby girl born at 25 weeks of gestation was admitted at 22 months of life as a result of increasing respiratory distress and progressive heart failure. The cause of heart failure was not known. She required ECMO support for 10 days. She underwent VAD implantation at 23 months of life. The pediatric nephrology service was consulted at 24 months of age, Day 45 on VAD, because a computed tomography scan of the abdomen showed patchy areas of decreased contrast uptake in bilaterally enlarged kidneys. Renal US showed decreased flow in the intrarenal vessels corresponding to hypoechogenic regions seen in both kidneys and patent in both the main renal artery and vein. Renal function was normal with serum creatinine was .22 mg/dL. The patient was receiving heparin anticoagulation. PT was 13.9, INR 1.1, PTT 102, fibrinogen 412, and d-dimer 3.14.

Baseline renal US at 22 months of life showed the right kidney 8.9 × 5.1 × 4.7 cm and left kidney 9.2 × 4.6 × 4.2 cm. A summary of kidney size for Patient C is shown in Table 1. She subsequently underwent orthotopic heart transplantation at 2 years 3 months of age after 5 months on VAD. She died at 4 years of age as a result of a massive thromboembolic stroke resulting in cardiorespiratory failure. Autopsy findings showed the right kidney was atrophic with the upper pole replaced with a large scar. Bilateral renal artery branches and venous tributaries

Table 1. Summary of kidney size in Patients A, B, and C.

Summary of kidney size, Patient A			
Days on VAD	Imaging	Right kidney size (cm)	Left kidney size (cm)
Day 4 of life before VAD	US	4.2 × 2.5 × 2.4	4.7 × 2.5 × 2.4
Day 14 VAD	US	7 × 3.5 × 2.9	6.3 × 3.7
Day 63 VAD	US	7 × 2.8 × 3.4	8.8 × 4.5 × 4.5
Day 69 VAD	US	7.3 × 4.2 × 3.6	8.8 × 4.5 × 4.5
Summary of kidney size, Patient B			
Day 10 VAD	CT	Hypoperfusion of two-thirds of right kidney	Hypoperfusion of whole left kidney
Day 11 VAD	US	6 × 3.2 × 2.3	6 × 2.9 × 2.6
Day 15 VAD	US	6.3 × 3.1 × 3.9	6.2 × 3.7 × 2.8
Day 28 VAD	US	6.5 × 3.7 × 3	7 × 3.8 × 3.5
Summary of kidney size, Patient C			
Before VAD	US	8.9 × 5.1 × 4.7	9.2 × 4.6 × 4.2
Day 44 VAD	CT	Kidneys enlarged multiple peripheral wedge-shaped hypodensities	
Day 45 VAD	US	9.5 × 4.2 × 4.2	11.1 × 5.1 × 3.9
S/P heart transplant	US	8.7	9.7

VAD, ventricular assist device; S/P, status post; US, ultrasound; CT, computed tomography.

were unremarkable. Microscopic examination of the kidney revealed diffuse glomerulosclerosis, mild tubular atrophy and chronic interstitial nephritis, right superior pole of the kidney with fibrosis, and parenchyma replaced by scar.

A Berlin Heart EXCOR Pediatric Edmonton anticoagulation protocol was used in all three patients (6). Patients A and B were younger than 12 months age; unfractionated heparin was started 24 hours after surgery with an initial dose of 15 units/kg/h and increased to 28 units/kg/h after 6 hours. Patient C was started on an unfractionated heparin initial dose of 10 units/kg/h and increased to 20 units/kg/h after 6 hours. PTT, INR, platelet count, fibrinogen, thromboelastography (TEG), and antithrombin III were checked every 24 hours. Primary target for heparin anticoagulation was 1.5–2.5 times baseline PTT (normal PTT 28–38 seconds) and secondary target TEG Rk 8–15 minutes. Dipyridamole was initiated 48 hours after VAD implantation at 4 mg/kg/day peroral/pernasogastric-tube divided in four doses in all three patients. Aspirin was initiated 96 hours after VAD implantation at 1 mg/kg/day divided into two doses in all three patients. All three patient anticoagulation parameters were on target at the time when imaging studies, either computed tomography or US of the abdomen, was obtained, which showed enlarged and echogenic kidneys.

DISCUSSION

To our knowledge this is a first time kidney findings have been reported in patients implanted with a pediatric VAD. In neonates up to 2–3 months age, kidneys are sonographically more echogenic than those of older infants and children. After 2–3 months, the renal cortex assumes the adult pattern and becomes less echogenic than the liver. It has been suggested that the increase in echogenicity in neonates and young infants is the result of more glomerular density in the renal cortex (7). This resulted in an increased number of acoustical interfaces and increased echogenicity. Although nonspecific, if echogenicity of the kidney is greater than that of the liver, one should suspect an underlying renal parenchymal disease. The differential diagnosis of enlarged and echogenic kidneys in infants and young children includes adult and infantile polycystic kidney disease, Pearlman syndrome and Beckwith-Wiedemann syndrome, trisomy 13, cystic dysplasia, and normal variant (8).

An interesting finding in our patient was rapid enlargement of the kidney a few days after VAD placement. In Patients B and C, evidence of renal artery thromboembolism was demonstrated by imaging (computed tomography/US) and confirmed on autopsy finding. Renal artery thromboembolism, rapid enlargement, and increased echogenicity of the kidneys coexisted in Patients B and C. However, in Patient A, there was no evidence of renal artery thrombosis both on imaging studies and autopsy findings.

Our case series shows that enlarged and echogenic kidneys may be associated with thromboembolism of the renal artery in pediatric patients on VAD. A specific cause-and-effect relationship is not clear.

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