

# Hypertension and cardiac failure aggravated by dehydration in a case of coarctation of the aorta

*Aort koarktasyonlu bir hastada dehidratasyonun neden olduğu hipertansiyon*

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The coarctation of the aorta is characterized by systemic arterial hypertension above the lesion and normal or decreased blood pressure below it. However we know that severe dehydration is characterized by normo or hypotension. We report a 2-month-old boy with coarctation of the aorta; whose renin-induced hypertension is aggravated by dehydration and controlled by intravenous fluid therapy.

**Key words:** *Coarctation of aorta, dehydration, renin, systemic hypertension.*

Aort koarktasyonu lezyonun yukarisinda sistemik hipertansiyonu ve lezyonun altinda normal veya düşük kan basincıyla karakterizedir. Bununla birlikte ağır dehidratasyonun normo veya hipotansiyonla karakterize olduğunu bilinmektedir. İki aylık aort koarktasyonlu erkek çocukta; reninin uyardığı hipertansiyonun dehidratasyonla belirginleşmesi ve intravenöz sıvıyla tedavisini sunulmaktadır.

**Anahtar kelimeler:** *Aort koarktasyonu, dehidratasyon, renin, sistemik hipertansiyon.*

**C**oarctation of the aorta (CoA) is congenital narrowing of the aorta, most commonly located just distal to the left subclavian artery at the level of the ductus arteriosus insertion (1). Its morphological features include a posterior indentation of the aorta that may be associated with isthmus or transverse aortic arch hypoplasia, along with various forms of intracardiac lesions (1,2). The coarctation is characterized by systemic arterial hypertension above the lesion and normal or decreased blood pressure below it. Systemic hypertension, found both before and after coarctation repair, has been intensively studied for many years and a variety of theories developed to explain its occurrence (1-3).

We reported a 2-month-old boy with coarctation of the aorta. He was admitted to emergency room with aggravation of hypertension and heart failure, which were stimulated by dehydration. We easily controlled his hypertension and associated heart failure with intravenous fluid therapy.

## Case report

A 2-month-old boy had been followed-up with the echocardiographic diagnosis of postductal coarctation of the aorta, small perimembranous ventricular septal defect (VSD), and bicuspid aorta from postnatal 2nd day.

Two weeks ago when he was seen, he was asymptomatic, well-grown baby. Mild systemic hypertension, and pressure difference between upper and lower extremities (right arm blood pressure (BP): 106/58 mmHg, right leg BP: 68/42 mmHg) were found. An electrocardiogram (ECG) showed sinus tachycardia (150/minute), and increased R voltages in left precordial leads. Cardiothoracic index (CTI) was 0.60 at chest X-ray. His cardiac catheterization and angiog-

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raphy demonstrated postductal coarctation of aorta with 30mmHg systolic gradient and perimembranous VSD. Elective surgical repair was planned and he was discharged without therapy.

He was brought to the emergency room, with a generalized tonic-clonic seizure. He was suffering from fever up to 39 °C, diarrhea -5 times a day-, and vomiting -8 times a day-. At the time of his emergency room visit, he was severely dehydrated and, peripheral circulation was poor, he had tachypnea (respiratory rate was 76/minute), dyspnea, and tachycardia (heart rate was 210/minute) with gallop rhythm. He had a harsh pansystolic murmur at the lower left sternal border. Hepatomegaly was found (4 cm below costal margin at midclavicular line). His hypertension was found to be aggravated (right arm blood pressure: 135/105 mmHg). His ECG showed sinus tachycardia (214/minute), and prominent left ventricular hypertrophy. Chest X-ray showed increased dimensions of cardiac shadow (CTI:0.68) and congestion of the lung fields.

His laboratory examinations at admission revealed leukocytosis (21 500/mm<sup>3</sup>), severe metabolic acidosis (pH 7.25, pCO<sub>2</sub> 23.3 mmHg, HCO<sub>3</sub> 10.0 mmol/L, and base deficit -15.5 mmol/L), and normal electrolytes. Renal and hepatic functions were normal. Bacteriologic cultures including blood, urine, stool, and cerebrospinal fluid were all negative. Plasma renin level was 12 ng/dl (0.5-1.9 ng/dl) and plasma aldosterone level was 145 ng/dl (3-35.5 ng/dl). His convulsion was controlled by rectal diazepam. Intravenous fluid resuscitation was immediately started by isotonic saline infusion. Acidosis was controlled by NaHCO<sub>3</sub> infusion.

After the reestablishment of the circulatory volume in the first hospital day, his blood pressure was regressed to the previous levels without any intervention. Tachycardia, tachypnea, dyspnea were all resolved. Increased CTI was returned to original size at 5th day of admission and pulmonary congestion was disappeared. His plasma renin and aldosterone levels re-measured after two weeks and found to be 5.2 ng/dl and 53.9 ng/dl, respectively.

## Discussion

Coarctation of the aorta of varying degrees may occur at any point from the transverse arch to the iliac bifurcation, but 98% occur just below the origin of the left subclavian artery. Patients with coarctation usually present with heart

failure during the first few weeks of life or they are diagnosed during investigation of murmur or hypertension after the neonatal period (1,3,4-6). Although, our patient had no symptoms of heart failure, he was diagnosed too early by echocardiography during the investigation of murmur of small VSD.

Hypertension associated with coarctation is not due to the mechanical obstruction alone, but also involved activated renin-angiotensin-aldosterone system (RAS) (1,2,5-8). An elevated pre-coarctation blood pressure is necessary for the adequate perfusion of kidneys. Thus, increased activation of RAS as a result of severe dehydration to maintain adequate renal blood flow is not an unexpected finding. However, acute additional increment of afterload due to aggravated hypertension led to development of decompensated heart failure in our patient. High levels of renin and aldosterone in this acute setting and decrease of both levels demonstrated the activation of RAS after dehydration therapy. This finding support to the role of RAS in systemic hypertension of the patients with CoA.

In conclusion, we suggest that dehydration may be detrimental in a patient with asymptomatic CoA, causing aggravation of systemic hypertension and decompensated heart failure. It should be remembered that effective intravenous fluid therapy should be encountered a life saving intervention in such a situation.

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