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Critical coarctation of the aorta in newborn - A case presentation

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Krytyczna koarktacja aorty u noworodka- opis przypadku

STRESZCZENIE

Wady serca to najczęstsze schorzenia wrodzone spotykane u noworodków. Koarktacja aorty (CoA) stanowi 5-10% wad wrodzonych układu sercowo-naczyniowego. Polega na zwężeniu cieśni aorty między odejściem lewej tętnicy podobojczykowej a przyczepem więzadła tętniczego. W okresie noworodkowym i wczesniemowlęcym w przypadku opóźnionego rozpoznania może prowadzić do ostrej niewydolności serca i zgonu dziecka.

Noworodek płci żeńskiej, urodzony w 37hbd, drogą cięcia cesarskiego, w stanie dobrym został przyjęty na Oddział Noworodkowy. W wywiadzie ciążowym obecne było podejrzenie CoAo u płodu. W badaniu echo potwierdzono zwężenie w miejscu cieśni, do leczenia włączono Prostin. W 2. dobie życia nastąpiło przeniesienie na oddział kardiologiczny. Próba czterokończynowego pomiaru ciśnienia tętniczego wykazała wyższe wartości na kończynach górnych niż dolnych. W badaniu echokardiograficznym stwierdzono hipoplazję łuku aorty na odcinku poniżej odejścia drugiego naczynia dogłowego z koarktacją w miejscu cieśni. Badania laboratoryjne wykazały wysoki poziom pro-BNP. W trakcie hospitalizacji dziecko przebywało w inkubatorze, stale monitorowane, bez spadków saturacji. Kontrolne echo serca wykazało anatomie wady jak poprzednio, drożny szeroki przewód tętniczy z dominującym przepływem prawo-lewym, przepływ w aorcie zstępującej o szybkości 2,7m/s z koartacyjnym spektrum przepływu. Dziecko zostało przetransportowane do Śląskiego Centrum Chorób Serca w Zabrze celem leczenia operacyjnego.

W przypadku noworodków i niemowląt z krytyczną postacią zwężenia cieśni aorty należy zapewnić drożność przewodu tętniczego przez ciągły, dożylny wlew prostaglandyny E1. Obecnie podstawową metodą w diagnostyce zwężenia cieśni aorty jest badanie echokardiograficzne.

Abstract

Background

Heart defects are the most popular congenital disease noticed among infants. Aortic Coarctation (CoA) forms 5 - 10 % of congenital heart diseases. The heart defect consist on aortic coarctation between inlet of the left subclavian artery and origin of the arterial ligament. In the during neonatal and early infantile period, in case of delayed diagnosis, it may lead to acute heart failure and the death of the child.

Case report

A female infant born at 37 weeks of gestation, by caesarean section, in good condition, was admitted to the Neonatal Department. There was a suspicion of CoA in the fetus in the pregnancy history. The Echocardiography showed a narrowing of the carpal tunel, prostin was included in the treatment. In the second day of life, the child was transferred to the

Cardiology Department. Four-extremity blood pressures test showed higher values in the upper limbs than in the lower limbs. Echocardiography revealed hypoplasia of the aortic arch in the section below the exit of the second cephalic vessel with coarctation at the isthmus site. Laboratory tests showed a high level of pro-BNP. The control echocardiography showed the anatomy of the defect as before, an unobstructed wide arterial duct with a dominant R-L flow, a flow in the descending aorta of 2.7 m/s, with a coartable flow spectrum. The child was transported to the Department of Cardiac Surgery, Heart Transplantation and Mechanical Circulatory Support for Children in Zabrze for surgical treatment.

Conclusions

In neonates and infants with critical aortic stenosis, ensure the patency of the ductus arteriosus with continuous intravenous infusion of prostaglandin E1. Currently, echocardiography is the basic method in the diagnosis of aortic stenosis.

Introduction:

Heart defects are the most popular congenital disease noticed among infants. Approximately 8 out of 1000 alive born babies has heart defect determinate as significant. To add more, among 30 % of them need surgical procedure during first twelve months of live [1]. Aortic Coarctation (CoA) forms 5 - 10 % of congenital heart diseases. Approximately twice as often appears among boys [2]. CoA often is noticed in group of patients who suffer from Turner Syndrome [3]. The heart defect consist on aortic coarctation between inlet of the left subclavian artery and origin of the arterial ligament. Coarctation could take various forms of intensity. We can pick out infant type CoA, when coarctation appears on long section. There is also adult type CoA, when ring-shaped coarctation appears. CoA could be diagnosed in the first trimester, but very often diagnosis could be false-positive as well as false-negative. Many cases remain undetected up to the labour [4,5]. Correct diagnosis of CoA during neonatal and early infantile period also pose a significant clinic problem. When the coarctation is minor clinical symptoms may not appear. Subject to the speed of closing of the arterial duct, symptoms of cordial failure may appear after few hours, days or weeks. The most popular symptoms which appears among infants are: decrease blood saturation in lower appendages, impairment of appetite, heart murmur alongside left edge of sternum with beam to the interscapular area. Among older children very often appears headaches, epistaxis, decrease of pulse wave on femoral artery. In the case of late diagnosis acute decompensated heart failure and death of the baby may occur [2]. Treatment consist in surgical removal of stenosis. In order to keep patency of the ducts to the moment of surgical procedure, there is necessary to secure constant intravenous infusion of prostaglandin E1 [6]. After surgical or interventional treatment Aortic Coarctation could occur. That may lead to arterial hypertension [3].

Case report:

A female infant born at 37 weeks of gestation, by caesarean section, in good condition, Apgar 8-9-9-10 points was admitted to the Neonatal Department. There was a suspicion of CoA in the fetus in the pregnancy history. The Echocardiography showed a narrowing of the carpal tunnel. Prostin was included in the treatment at a dose of 0.01 $\mu\text{g}/\text{kg}/\text{min}$. In the second day of life, the child was transferred to the Cardiology Department. The girl did not show signs of overt CHF (chronic heart failure). Four-extremity blood pressures test showed higher values in the upper limbs than in the lower limbs. The values of blood pressure and saturation on 4 limbs were: right upper limb 73/34, 95%, left upper limb 71/33, 94%, right lower limb 69/34, 92%, left lower limb 66/32, 94%. The skin was properly warmed, moist, and the bruising covered the peripheral parts of the limbs. Lung auscultation revealed normal symmetrical vesicular sound. There were two normal, correctly accented heart sounds heard above the heart. Echocardiography revealed hypoplasia of the aortic arch in the section below the exit of the second cephalic vessel with a diameter of about 3 mm and a length of about 1-1.2 cm, with coarctation in the place of the isthmus with a diameter of about 2.4 mm. In CT (Computed tomography) angiography, the aortic arch appeared to be constricted between the exit of the left subclavian artery and the Botalla arterial duct, to 2.8 mm by about 10 mm. Ascending aorta approx. 7 mm width, aortic arch approx. 6 mm width. The assessment of the ascending aorta and the aortic arch was difficult due to the lack of proper contrasting of the above-mentioned aortic sections (Fig. 1).

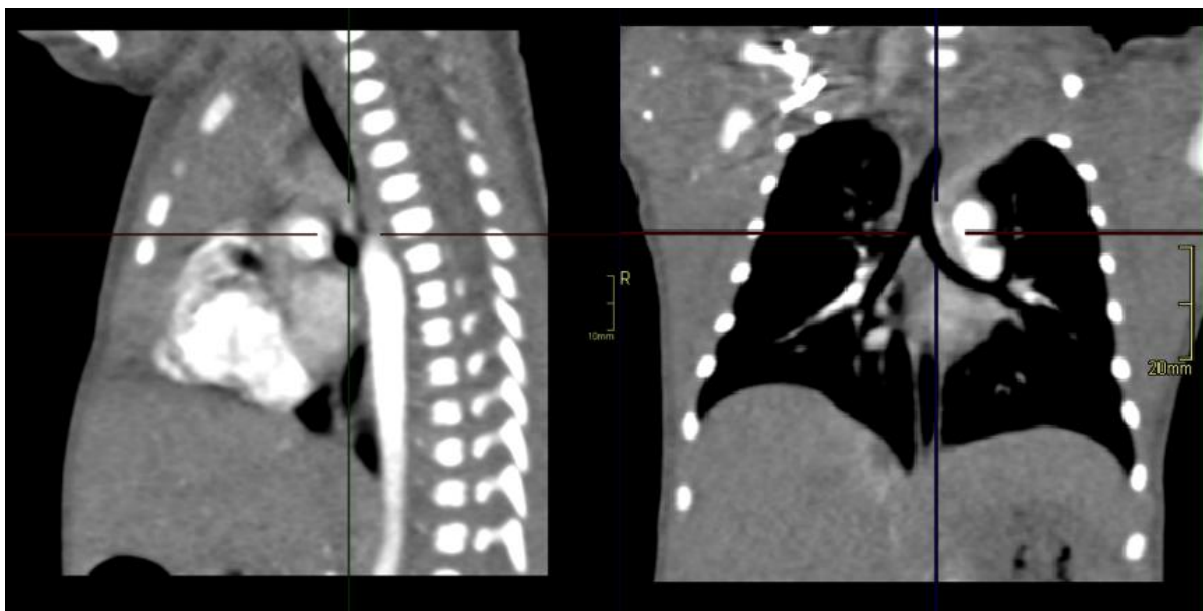


Figure 1: CT aortography- Optimal contrast of the descending aorta has been achieved. The ascending aorta, the aortic arch, and the large vessels extending from it were not normal contrasting. Botalla ductus arteriosus: 6 mm, aorta directly behind the arterial duct: 7 mm, pulmonary trunk: 11 mm. The aortic arch appears to be narrowed between the left artery exit point subclavian and Botalla's arterial duct, up to 2.8 mm in length about 10 mm; aorta 7 mm wide ascending aorta, 6 mm wide aortic arch.

Laboratory tests showed a high level of pro-BNP (14 127.00 pg/ml - the day after admission, 25 109.00 pg/ml - 4 days after admission and 32 925.00 pg/ml - on the 8th day of hospitalization). In blog gas test, pH - 7.305, blood lactate 1.80mmol/l. During hospitalization, the child was in the incubator, constantly monitored. There were no decreases in saturation.

Prostin was infused at a dose of 0.03 µg/kg/min. The girl ate 30-40 ml of mother's milk. There was a slight tightening of the diaphragm attachments. Due to the increased value of total bilirubin on the 8th day of hospitalization (14.09 mg/dl reference range 0-2), continuous phototherapy was used for 2 days. The control echocardiography showed the anatomy of the defect as before, an unobstructed wide arterial duct with a dominant R-L flow, a flow in the descending aorta of 2.7 m/s, with a coartable flow spectrum. Gradient by taper was 30 mmHg. Moderate mitral and tricuspid valve regurgitation, elevated right ventricular systolic pressure (RVSP) and dominant right heart chambers were present. The child was transported to the Department of Cardiac Surgery, Heart Transplantation and Mechanical Circulatory Support for Children in Zabrze.

Conclusions:

In neonates and infants with critical aortic stenosis, ensure the patency of the ductus arteriosus with continuous intravenous infusion of prostaglandin E₁. Currently, echocardiography is the basic method in the diagnosis of aortic stenosis. In newborns and infants, careful assessment of blood pressure, presence of pulse and oxygen saturation on the lower limbs is also essential. Older children should have their blood pressure measured during medical check-ups. Patients after surgical procedures must be observed throughout their lives. This way, you can minimize the risk of developing life-threatening late complications.

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