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CONGENITAL HEART DISEASE IN THE NEWBORN: A LITERATURE REVIEW AND CASE REPORT

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Abstract: Critical congenital heart diseases are important causes of mortality and morbidity in the neonatal period. Even after diagnosis, its management can be seriously compromised by unplanned delivery in hospitals with limited experience and resources. The newborn can spend days or weeks before transport, placing a significant burden on the neonatal team to deal with these challenging illnesses. In this review, the management principles of each individual pathology are discussed prior to transport to cardiac centers. Until transport can be accomplished in a matter of hours, neonatologists and pediatricians need to master the essential principles of managing these conditions in the pre-transport setting. Although understanding the hemodynamics in several critical congenital heart diseases is a difficult task and requires a trained professional, the practical concepts in its management before surgery are simple and can be mastered by the neonatologist and the pediatrician with minimal effort.
Keywords: Congenital heart defects; congenital malformations; Newborn; congenital heart surgery; pediatrics.

INTRODUCTION

Congenital heart defects (CHDs) are the most common congenital anomaly, occurring in approximately 6 to 10 children per 1,000 live births. Approximately 25% of children born with CHD will require immediate heart surgery or other interventions to survive. CDs account for the highest percentage of hospitalizations (34%), have higher costs and require longer hospital stays than any other birth defects in neonates. Newborns with CHD require neonatal intensive care and intensive care. Therefore, newborns undergoing congenital heart surgery require highly specialized and resource-intensive perioperative care.

Congenital malformations are important causes of mortality in children under one year old (CATARINO et al., 2017), with heart disease being the most common isolated congenital anomalies (BELO et al., 2016). The suspicion and early diagnosis of congenital heart diseases is recommended (CARVALHO et al., 2006) from the prenatal period, through the morphological examination of the fetus; after birth, still in the nursery; and in childcare consultations, to which the child will be submitted throughout its development (AGUILHERA et al., 2006).

Furthermore, the transfer of newborns with critical congenital heart disease (CCHD) is challenging in many countries with limited resources and bed availability. Newborns with CCHD may spend several days or weeks in a neonatal intensive care unit (NICU) with limited support from pediatric cardiology and cardiac surgery services. Fortunately, most CCHD can be kept alive without additional morbidities for weeks with the appropriate use of prostaglandin E1 (PGE1) and respiratory support that can be provided in almost all neonatal care units. In this article, we will review important CCHD management strategies in the NICU before the baby is transferred to a cardiac center. In addition, the objective of this literature review and case report is also to describe the diagnosis of a newborn with congenital heart disease identified in the immediate postpartum period, paying attention to the systemic manifestations that may be indicative of congenital heart diseases in newborns, emphasizing the importance of good clinical evaluation by the generalist pediatrician, with the use of timely additional diagnostic tests, aiming at clinical suspicion and early diagnosis of critical congenital heart disease (CCC).

CASE REPORT

Term newborn (39 weeks and 5 days), male, born by cesarean delivery, prenatal

care with 9 consultations, uneventful, Apgar 8/9 at birth, weighing 3,680g. On physical examination: newborn at birth showing good vitality, strong crying, active, reactive, ruddy, hydrated, anicteric, good peripheral capillary perfusion, normotensive fontanelle, present peripheral pulses, full and symmetrical, present reflexes. cardiovascular system with regular heart rhythm in 2 beats, normophonetic sounds, unidentified heart murmur. Respiratory system with universally audible breath sounds, no respiratory effort, no tachypnea. Globose abdomen, painless, without palpable visceromegaly. Limbs without edema, with good perfusion and pulses present and wide. Evolved from 24h of life to the presence of a holosystolic (3+/6+) pancardial murmur, but more intense in the upper left sternal border, radiating to the back, without cyanosis, without tachydyspnea, without palpable visceromegaly, with normal little heart test (MSD 99% | MID 99%) and the rest of the physical examination was unchanged. At 48 hours of life, assessed by the pediatric cardiology service, presenting dynamic precordium, propulsive ictus, systolic murmur 3+/6+ across the entire left sternal border radiating to the right and back, present and wide pulses, other physical examination suitable for the age group. An echocardiogram was performed which showed: situs solitus | absence of right atrioventricular connection (tricuspid atresia); concordant arterial ventricle connection; pulmonary artery emerging from the right ventricle and aorta emerging from the left ventricle | interatrial septum with ostium secundum type communication (ASD) measuring around 13mm | wide interventricular communication (IVC), measuring around 9mm, with bidirectional shunt | left heart chambers with significant dilation; left ventricle with preserved systolic function; EF 68%; hypoplastic right ventricle | atretic tricuspid valve; stenotic pulmonary valve, measuring the valve annulus at around 6mm, with a gradient of 25mmHg (mild stenosis); other valve apparatuses with normal morphology and dynamics | normal caliber aortic arch | Previous ductus arteriosus, around 3 mm, with a large bidirectional shunt. It was concluded: Type IB tricuspid atresia (with pulmonary stenosis and VSD); CIA ostium secundum broad; Patent ductus arteriosus. Suggested conduct: correction of metabolic disorders; target saturation > 75% (around 85%); transfer to a reference unit for palliative surgical treatment. Newborn transferred to the Neonatal Intensive Care Unit.

LITERATURE REVIEW

GENERAL CONSIDERATIONS

Surveillance of the newborn with CCC begins with reviewing the maternal medical history for risk factors for congenital heart disease and reviewing the fetal ultrasound or fetal echocardiogram. Physical examination is critical on initial admission to the nursery and prior to discharge with emphasis on cyanosis, tachypnea, bruit, and palpable femoral pulse. Physical examination of newborns with CCHD may not reveal a clear abnormality due to the transition of the fetal circulation with high pulmonary vascular resistance (PVR) and the presence of patent ductus arteriosus (PDA). Neonatal pulse oximetry screening for CCHD must be performed under ideal conditions and interpreted with care. When in doubt about the presence of CCC, the pediatric cardiologist must be consulted before the newborn is discharged. Newborns who appear normal in the nursery can present very critical conditions within two weeks with potentially long-term sequelae.

Although prostaglandin E1 (PGE1) infusion is the main intervention in most CCHD conditions, it does not help or may

not be sufficient in some of these conditions. In other conditions where the circulations are balanced, the use of PGE1 is potentially harmful and will prolong the hospitalization of the stable and potentially disposable newborn. Likewise, oxygen therapy may not be necessary in balanced circulation with mixed lesions. The lowest target oxygen saturation that is appropriate for the physiology is the optimal approach. Blinded oxygen therapy to achieve saturation above 90% in an excessive pulmonary blood flow (PSF) physiology will unnecessarily prolong respiratory support and hospital stay. Finally, unless ventricular dysfunction is documented by echocardiography, inotropic infusions must not be used for the sole purpose of improving cardiac function, especially if blood pressure is acceptable. Excessive and inappropriate use of inotropic infusions alters circulatory balance and compromises systemic blood flow with possible serious consequences on intestinal and renal perfusion.

SHUNT FROM LEFT TO RIGHT

Large ventricular septal defect (VSD), single ventricle, aortopulmonary window, and large patent ductus arteriosus (PDA) are the most common causes of significant left-to-right shunt in newborns. They cause significant pulmonary congestion and systemic hypoperfusion starting a few days after birth in many cases. Classically, they have normal oxygen saturation due to excessive pulmonary blood flow (PFF); however, they may have lower oxygen saturation due to elevated pulmonary vascular resistance or pulmonary edema. Oxygen is a potent pulmonary vasodilator and must be avoided in these cases unless there is significant desaturation. An acceptable 88% saturation may be required to avoid excessive oxygen use and allow discharge from the NICU. Diuretic therapy, high-calorie eating, and fluid restrictions are the mainstays of treatment. The newborn is expected to have some residual tachypnea and slow weight gain that can be followed and managed in the outpatient setting. In a few cases, the newborn cannot be discharged and will need to be transferred for surgical or catheter intervention.

TOTAL ANOMALOUS PULMONARY VENOUS DRAINAGE

Total anomalous pulmonary venous drainage (TAPVD) is usually unobstructed in the cardiac type, where the pulmonary venous confluence (PVC) is connecting to the coronary sinus, and almost always obstructed in the infracardiac type, where the PVC connects via a vertical vein to the systemic veins below the diaphragm. In the supracardiac type, where the PVC connects via a vertical vein to the innominate vein, and in the mixed type, the presence of obstruction needs to be carefully evaluated by echocardiography. In the presence of obstruction, the newborn will develop progressive pulmonary venous edema and pulmonary hypertension. The veins need to be surgically repaired after the blockage is discovered. As pulmonary blood flow through the pulmonary arteries is normal, the ductus arteriosus is not needed and prostaglandin can increase pulmonary congestion. In the rare cases where the obstruction is at the level of the ductus venosus, prostaglandin can improve the obstruction; however, it must not be considered a standard short-term therapy, as the standard treatment for obstructed TAPVD is surgical repair. This is one of the few pathologies where most of the temporary stabilization measures applied in other conditions do not work and the newborn must be immediately transferred to a facility where surgery can be performed. For unobstructed cardiac or supracardiac types, the newborn can be safely discharged from the hospital with an acceptable saturation of

75% or greater and follow-up with pediatric cardiology in 2 to 4 weeks.

CRITICAL PULMONARY VALVE STENOSIS AND PULMONARY ATRESIA

In critical pulmonary stenosis, the newborn cannot maintain acceptable saturation without PDA. In both conditions, PGE1 infusion must be continued until a more stable source of PBF is established via pulmonary valve dilation/ perforation, PDA stent, or BT shunt. PGE must be started as soon as the diagnosis is suspected at a dose of 0.03 to 0.05 μm/kg/min in an intravenous line separate from other infusions and must be evaluated regularly for patency and adequate flow. The dose can be increased to 0.1 μm/kg/min if 75% saturation cannot be achieved. Care must be taken to watch for apnea that may require mechanical breathing support. On rare occasions when the PCA could not be opened, we used PGE1 boluses of 3 to 5 μm/kg over 10 min in a small number of cases with success. PGE1 infusion can lower blood pressure and the newborn may need inotropic support with dopamine or epinephrine. The newborn may develop pulmonary hypercirculation due to a large PDA suggested by congested lung on chest X-ray and/or development of metabolic acidosis. In this case, PGE1 must be lowered to the lowest possible dose of 0.01 to 0.005 μm/ kg/min as long as the saturation is maintained above 75%. In transporting such cases, care must be taken not to interrupt the flow of PGE1, as this can have fatal consequences in an already limited setting.

TRICUSPID ATRESIA

In tricuspid atresia (TA), there is an obligatory right-to-left shunt at the atrial level. Rarely, the atrial septal defect is restrictive and the newborn will require a balloon atrial septostomy. The connection of the great arteries and the presence of pulmonary stenosis are determinants of the newborn's hemodynamics. If pulmonary atresia or critical pulmonary stenosis is present, the PDA must be left open to provide pulmonary blood flow. If the aorta arises from the right ventricle (RV), systemic flow is dependent on the size of the VSD. The VSD in TA is usually large but can become restrictive over time in the first few months. If the aorta arises from the LV and there is no pulmonary stenosis, the physiology is similar to a large VSD with a left-to-right shunt beyond the first week or two. In the presence of pulmonary stenosis, it is recommended to keep the newborn hospitalized until the PDA is closed or becomes very small and saturation is observed. If the saturation is above 75%, the baby can be safely discharged.

TETRALOGY OF FALLOT

Tetralogy of Fallot (TOF) in the newborn is a spectrum of limited pulmonary blood flow (PBF); from normal flow with little or no obstruction to total atresia. It is an abnormality of the branches of the pulmonary arteries, as well as of the heart with varying degrees of hypoplasia. The management of the newborn depends on the degree of pulmonary stenosis and the source of pulmonary blood flow in case of pulmonary atresia. If mild to moderate pulmonary stenosis is present, the newborn can be discharged with follow-up in 2 to 4 weeks to reassess the degree of pulmonary stenosis after the decrease in pulmonary vascular resistance. If pulmonary stenosis is severe, the newborn must be kept in the hospital and saturation observed until the PDA is closed. If saturation is maintained at or above 75%, the newborn can be discharged, otherwise prostaglandin E1 must be started in preparation for a more reliable source of PBF with Blalock-Taussing shunt (BT shunt), PDA stent, right ventricular outflow stent

(RVOT). There must be no delay in starting prostaglandin if saturation is not adequate, because the longer the delay, the less likely the PCA will respond to treatment. Other circulatory support measures while waiting for the emergency BT shunt are fluid boluses and inotropes to raise systemic vascular resistance and increase PBF. In this context, diuretic therapy must be avoided, as it reduces the preload necessary to overcome RVOT obstruction. In pulmonary atresia or if one of the branches of the pulmonary arteries is discontinuous and supplied by a PCA, it is unnecessary to emphasize the importance of prostaglandin E1 until a reliable BPF is established for both lungs. In case of pulmonary atresia and large aortopulmonary collaterals for both lungs, the newborn can be safely discharged if the saturation is greater than 75%. Time and effort must be devoted to carefully excluding the possibility that the PDA supplies one of the branches of the pulmonary arteries, as this will lead to loss of that branch if the PDA closes.

TOF with absent pulmonary valve is a rare condition in which the valve annulus is small but the leaflets have not formed causing significant stenosis and regurgitation. In late pregnancy, the fetus will have large pulmonary arteries causing significant bronchomalacia and airway compression. If they are born with severe airway obstruction, their management is challenging and the prognosis is not favorable. They must undergo angiotomography to assess the size of the pulmonary artery and the degree of bronchial obstruction. The decision to discharge versus transport depends on the degree of respiratory distress caused by the airway obstruction.

EBSTEIN ANOMALY

In mild or moderate Ebstein anomaly of the tricuspid valve, the newborn is likely to be asymptomatic; however, in severe Ebstein, right atrial (RA) pressure is elevated and can cause systemic congestion with edema, ascites, and hepatomegaly. The abnormal valve can cause functional pulmonary atresia, necessitating the use of prostaglandin to maintain adequate saturation of ≥ 75%. Diuretic therapy is required to improve edema but must be used with caution as pulmonary flow can be load dependent.

DOUBLE OUTLET RIGHT VENTRICLE

Dual outlet right ventricle (DORV) physiology is similar to TOF physiology if severe pulmonic stenosis is present and similar to large VSD physiology if the VSD is subaortic without pulmonic stenosis, both discussed above. A third entity is similar to the physiology of transposition of the great artery if the VSD is subpulmonary (Taussing-Bing Anomaly) and is discussed in the following sections. The TGA type may be associated with posterior deviation of the conal septum causing obstruction of the left ventricular outflow tract and commonly associated with hypoplastic aortic arch or coarctation of the aorta.

TRANSPOSITION OF THE GREAT ARTERIES

Transposition of the great arteries (TGA) has had very good long-term results after successful arterial-switch surgery at the present time. Unfortunately, it has one of the highest mortalities in the neonatal period if the diagnosis is not promptly established or if hemodynamic inadequacies are not addressed. Newborns with TGA and large atrial septal defects (ASD) usually have an acceptable saturation of more than 75% and can be operated on in the first weeks of life. It is advisable to keep them in PGE until they are transported to a facility that has experience in managing such cases. Newborns with TGA

who do not saturate well after starting PGE have restrictive patent foramen ovale (PFO) or pulmonary hypertension (PHTN). Balloon atrial septostomy is the ideal procedure to establish mixing at the atrial level if the PFO is small and must be performed, if possible, in the peripheral hospital or the baby transported within the first hours after establishing the diagnosis. Those with desaturation despite an acceptable ASD size and an open PDA likely have PHTN. They need to be treated with adequate sedation, paralysis, and mechanical ventilation to achieve normal arterial blood carbon dioxide (CO 2) levels (35 to 45 mmHg) and normal bicarbonate (HCO 3). They need to be started with nitric oxide (NO) and a maximum concentration of oxygen. Milrinone must be used for its pulmonary vasodilator effect and other inotropes (dopamine or epinephrine) must be titrated to achieve normal blood pressure. The sooner these measurements are used in combination with careful attention to avoiding high CO 2 levels and metabolic acidosis, the more likely the baby will survive without major sequelae. These measures need to be continued until the baby is stable enough for surgery and may be needed for a few days after surgery. Newborns with a large VSD are likely to have adequate mixing; however, this needs to be verified before discharge, observing the baby's saturation until the closure of the PDA.

CRITICAL AORTIC STENOSIS

In critical aortic stenosis (AS), flow through the aortic valve is inadequate to support the systemic circulation. They have very high LV pressure and low aortic pressure, causing severe coronary hypoperfusion. The newborn will need the PDA to maintain perfusion and blood pressure. LV obstruction causes varying degrees of left ventricular hypertrophy, dilation, dysfunction, or hypoplasia. The newborn is at risk of ventricular arrhythmia, which increases the serious risk of these newborns. The lung is congested due to elevated left atrial (LA) pressure and will get worse over time if the obstruction is not relieved. Prostaglandin infusion to keep the PDA open is essential, but it does not improve elevated LA pressure. Associated pulmonary hypertension must never be treated with pulmonary vasodilators such as nitric oxide, as this will cause increased flow into an already obstructed venous drainage. Adequately sized DSA improves pulmonary edema; however, atrial septostomy is not a standard procedure in this condition, as the same effect can be achieved by balloon dilation of the aortic valve. The newborn may need a small dose of inotrope to increase blood pressure and counteract the sedative effect.

In severe aortic stenosis, a milder degree of the same hemodynamics is usually manifested, but the baby does not need the PCA to maintain systemic perfusion. They are at risk for pulmonary edema, ventricular dysfunction, and arrhythmia caused by coronary hypoperfusion. After a period of observation, some of them may be stable enough to be discharged and evaluated in a cardiac center in 1 to 2 weeks with balloon dilation as soon as possible in the first month of life. A careful assessment in collaboration with the acceptance center must be considered before discharging the newborn.

HYPOPLASTIC LEFT HEART SYNDROME

The hemodynamics of hypoplastic left heart syndrome (HLHS) is the most challenging of all CCHD. Systemic circulation is dependent on PDA. The ratio of blood flow to the pulmonary and systemic circulation (Qp:Qs) is dependent on pulmonary vascular resistance that decreases over days to weeks accompanied by increased PBF and systemic hypoperfusion. Oxygen saturation of 75

to 80% is a simple but rough measure of relatively balanced flows. Higher saturation means lower systemic perfusion and must be avoided. The lower the oxygen saturation toward 75 to 80%, the more balanced the Qp:Qs are, and oxygen therapy must be avoided if these goals are met. If the atrial communication is small, the newborn will develop pulmonary venous congestion, but will have less pulmonary hypercirculation. These changes are dynamic and are constantly changing by factors that manipulate pulmonary and systemic resistance such as oxygen, CO2, PH, mechanical ventilation, inotropes and temperature. Most newborns will develop a progressively increasing need for diuretics due to lung congestion. As perfusion of the gastrointestinal tract and kidney may be compromised, feeding must be carefully monitored and efforts made to prevent kidney injury. Trophic feeding is advantageous before surgery. The ethics of the do-not-resuscitate decision (DNR) is an important consideration in this condition before and after birth and must be discussed with the attending cardiologist. The outcome of Norwood surgery for this condition is less favorable with prematurity, genetic syndrome, severe tricuspid valve regurgitation, and right ventricular dysfunction. These factors must be considered before committing the family to a transfer to another city or province for a potentially prolonged hospital stay. If Norwood is considered, serious effort needs to be made to transport the newborn as soon as possible, because delaying Norwood surgery beyond three weeks increases the risk of operative mortality and significant morbidity.

COARCTATION OF THE AORTA AND INTERRUPTION OF THE AORTIC ARCH

In severe coarctation (COA) or interrupted arch (IAA), the blood supply to the parts of the body that are supplied by branches distal to the COA or IAA depends on the PDA. Classically, the lower body has less saturation but adequate perfusion if the PDA size is large enough. If caught early, the newborn will function normally and will not need more than a prostaglandin infusion. Newborns who present at an advanced stage may develop severe left ventricular (LV) dysfunction and need more support with ventilation and inotropes.

FINAL CONSIDERATIONS

The health and survival of newborns with CCHD are vulnerable in the period prior to establishing the diagnosis and during hospitalization in facilities not equipped to deal with their conditions. Until transport can be accomplished in a matter of hours, neonatologists and pediatricians need to master the essential principles of managing these conditions in the pre-transport setting. Although understanding the hemodynamics in several CCCs is a difficult task and requires a trained professional, the practical concepts in its management before surgery are simple and can be mastered by the neonatologist and the pediatrician with minimal effort.

The early diagnosis of congenital heart diseases even in the neonatal period allows a timely and more accurate assessment regarding the necessary interventions, thus confirming the importance of early diagnosis for adequate treatment, prevention of aggravation and sequelae, providing opportunities for improved prognosis and allowing the prolonged survival of the child's health. Thus, it is necessary for the assistant pediatrician to understand the systemic manifestations of congenital heart diseases. Knowing how to evaluate alterations during the clinical examination, being precise in the proper management of complementary diagnostic tests, as well as the opportune

moment of their requests, becomes relevant for the planning of actions that involve the prevention and control of injuries, offering the opportunity for actions proactive actions and development of appropriate care strategies, thus improving the prognosis and quality of life of children with congenital heart diseases.

REFERENCES

AGUILERAS, RODRÍGUEZJ. G,ENRÍQUEZG.Cardiopatíascongénitascondiagnósticoprenatal:seguimiento. RevChilObstetGinecol.2006;71(5):320-6.http://dx.doi.org/10.4067/S0717-75262006000500005.

AMARAL F, MANSO P. H, GRANZOTTI J. A, VICENTE W.V.A, SCHMIDT A. Cardiopatia congênita noadulto: perfil clínico ambulatorial no Hospital das Clínicas de Ribeirão Preto. Arq BrasCardiol.2010;94(6):707-13.http://dx.doi.org/10.1590/S0066-782X2010005000053.

ANSHULAT,SENGUPTAS,SHUKLAV,DANDAS.Riskfactorsforcongenital heart disease (CHD)inVellore,India. CurrResJ.2010;2(4):253–8.

ARAÚJOJ.S.S,RÉGISC. T,GOMESR.G.S,SILVAC. S,ABATHC.M.B,MOURATOF.A,etal.Cardiopatiacongênita noNordestebrasileiro: 10anos consecutivos registrados no EstadodaParaíba,Brasil.RevBrasCardiol.2014;27(1):13-9.

BASTOSL. F,ARAÚJOT.M,FROTANM.CAETANOJÁ.Perfilclínicoepidemiológicodecriançascom cardiopatiascongênitas submetidas àcirurgiacardíaca.Rev Enferm UFPEOnLine.2013;7(8):5298-304.10.5205/reuol.3452-28790-4-ED.0708201330.

BELO, WANESSA ALVES; OSELAME, GLEIDSON BRANDÃO; NEVES, EDUARDO BORBA. Perfil clínico-hospitalar de crianças com cardiopatia congênita. Cad. saúde colet., Rio de Janeiro , v. 24, n. 2, p. 216-220, Junho 2016 .

BOCCHIE.A,BRAGAF.G.M,FERREIRAS.M.A,ROHDEL.E,OLIVEIRAW.A,ALMEIDAD.R,etal. IIIDiretrizdaSociedadeBrasileiradeCardiologiaparaGravideznaMulherPortadoradeCardiopatia. ArqBrasCardiol2009;93(1Supl1):110-78.http://dx.doi.org/10.1590/S0066-782X2009002000001

BRASIL. Lei no. 9.610, de 19 de fevereiro de 1998. Altera, atualiza e consolida a legislaçãosobre direitos autorais e dá outras providências. Diário Oficial daUnião, Brasília,DF; 20fev.1998[citadoem12fev.2015].Disponívelemhttp://www.dou.gov.br/materias/do1/do1legleg19980220180939-001.htm.

BRUNSR.F, MORONA.F, MURTAC.G.V,GONÇALVESL.F.A,ZAMITHM.M.Opapeldatranslucêncianucalno rastreamentode cardiopatias congênitas.ArqBrasCardiol.2006;87(3):307-14. http://dx.doi.org/10.1590/S0066-782X2006001600013.

CATARINO, CF ET AL. Registos de cardiopatia congênita nos sistemas de informações em saúde. Epidemiol. Serv. Saúde, Brasília, 26(3): 535-543, julho-setembro 2017.

CARVALHOS.E.M,MENDESM.C,CAVALLIR.C,MACHADOJ.C,DUARTEG,BEREZOWSKIA. Rastreamentoediagnósticoecocardiográficodasarritmiasecardiopatiascongênitasfetais.RevBrasGinecolObstet.2006;28(5):304-9. http://dx.doi.org/10.1590/S0100-72032006000500007.

CHEWC,HALLIDAYJ.L,RILEYM.M,PENYD.J.Population-basedstudyofantenataldetectionofcongenitalheartdiseasebyultrasoundexamination.UltrasoundObstetGynecol.2007;29(6):619-24.http://dx.doi.org/10.1002/uog.4023.

CriticalAppraisalSkillsProgramme(CASP).Makingsenseofevidence.MiltonKeynesPrimaryCareTrust,2006.

FRIAS, L ET AL Diagnóstico precoce das cardiopatias congênitas: Uma revisão integrativa. JMPHC | Journal of Management & Primary Health Care | ISSN 2179-6750, v. 9, 19 dezembro 2018.

FROTAM. A,SILVAI.A,SANTOSZ.M.S.A,SILVAC.A.B.S,FERNANDESA.F.C.Perfilsociode mográficofamiliareclínico de crianças comcardiopatiacongênitaatendidasemumainstituiçãohospitalar.RevBrasPromocSaude.2014,27(2):239-46.http://dx.doi.org/1010.5020/18061230.2014.p239.

HISHINUMA, GILBERTO. Rastreamento de cardiopatias congênitas críticas em recém-nascidos assintomáticos de um hospital de ensino. 2017. 34 f. Dissertação (mestrado em Ciências da Saúde) --Universidade Estadual de Maringá, 2017, Maringá, PR.

HUBERJ,PERESV.C,SANTOST.J,BELTRÃOL.F,BAUMONTA.C,CANEDOA.D,etal. Cardiopatiascongênitasemumserviçodereferência:evoluçãoclínicaedoençasassociadas.ArqBrasCardiol.2010;94(3):333-38. http://dx.doi.org/10.1590/S0066-782X2010000300009.

JEGATHEESANP,DONGLIS,CATHYA,KAMAKSHID,GOVINDASWAMIB. Oxygensaturationnomograminnewbornsscreenedforcriticalcongenitalheartdisease.Pediatrics.2013;131(6): e1803-10.https://doi.org/10.1542/peds.2012-3320.

KEMPERA.R,MAHLEW.T,MARTING.R,COOLEYC,KUMARP,MORROWR,etal.Strategiesforimplementing screeningforcriticalcongenitalheartdisease.Pediatrics.2011;128(5):e1259-67.https://doi.org/10.1542/peds.2011-1317.

MELLANDERM, SUNNEGARDH J. Failure todiagnose criticalheart malformationsinnewborns before dischargeanincreasingproblem?ActaPaediatric.2006;95(4):407-13.http://dx.doi.org/10.1080/08035250500541910.

MENDESK. D.S,SILVEIRAR.C.C.P,GALVÃOC. M.Revisãointegrativa: métododepesquisaparaaincorporação deevidências nasaúdeenaenfermagem.TextoContextoEnferm.2008;17(4):758-64.http://dx.doi.org/10.1590/S0104-07072008000400018.

MENDESM.J.F.L,ALVESJ.G.B,ALVESA.V,SiqueiraPP,FreireEF.Associaçãodefatoresderiscoparadoenças cardiovasculares em adolescentes eseus pais.Rev Bras SaudeMaternInfant.2006;6(1):549-54.

MINISTÉRIO DA SAÚDE (MS). Teste do coraçãozinho (oximetria de pulso) na triagem neonatal. Departamento de Gestão e Incorporação de Tecnologias em Saúde da Secretaria de Ciência, Tecnologia e Insumos Estratégicos – DGITS/SCTIE. Comissão Nacional de Incorporação de Tecnologias no SUS (CONITEC) - Relatório nº 115. 2014.

MOSERL.R.D.N,DIÓGENEST.C.P, SOUZAV.O.P,OLIVEIRAA.R.F,MOURATOF.A,MATTOSS.S.Novomodelodeteletriagem dascardiopatiascongênitas.JBras Tele.2014;3(1):229-31.https://doi.org/10.12957/jbrastele.2014.10236.

NORDOND.G,PRIGENZIM.L.Cardiopatiacongenital:difícildiagnósticodiferencialeconduçãodotratamento. RevFacCiencMedSorocaba.2012;14(1):24-6.

PIKEJ.I,KRISHNANA,DONOFRIOM.T.Earlyfetalechocardiography:congenitalheartdiseasedetectionanddiagnosticaccuracyin the hands of anexperiencedfetalcardiologyprogram.Prenat Diagn. 2014;34(8): 790-6. https://doi.org/10.1002/pd.4372.

PINTOJr,VALDESTERC.Cardiopatiascongênitas:rededeatençãoàsaúde.Fortaleza:Ediçãodoautor;2015.152p.

SANTOSA.D.S,MENEZESG.A,SOUSAD.S.Perfildosrecém-nascidoscomcardiopatiacongênitaemumamaternidadedealtoriscdomunicípiodeAracaju.CadGraduac.2013;1(17):59-70.

TELLLESJ.A.B,SCÜLER-FACCINIL.S.DefeitoscongênitosnoRiodeJaneiro:diagnósticoultrassonográficopeloestudomorfológico fetal.RevAMRJ.2010;54(4):421-26.

URAKAWAI.T,KOBAYASHIR.M. Identificaçãodoperfildiagnósticodeenfermagemdoneonato comcardiopatia congênita. RevPesq Cuid Fundam.2012;4(4):3118-24.10.9789/2175-5361. 2012.v4i4.3118-3124.

VOLPEP,ROBERTISV,CAMPOBASSOG,TEMPESTAA,VOLPEG,REMBOUSKOSG.Diagnosisofcongenital heartdiseasebyearlyandsecond-trimester fetal echocardiography. JUltrasoundMed.2012;31(4):563-8.https://doi.org/10.7863/jum.2012.31.4.563.