

Waist Circumference Measures Predict the Cardiovascular Risk Parameter

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Description

A Congenital Heart Defect (CHD), also known as a congenital heart anomaly and congenital heart disease, is a defect in the structure of the heart or great vessels that is present at birth. A congenital heart defect is classed as a cardiovascular disease. Signs and symptoms depend on the specific type of defect. Symptoms can vary from none to life-threatening. When present, symptoms may include rapid breathing, bluish skin (cyanosis), poor weight gain, and feeling tired. CHD does not cause chest pain. Most congenital heart defects are not associated with other diseases. A complication of CHD is heart failure. The cause of a congenital heart defect is often unknown. Risk factors include certain infections during pregnancy such as rubella, use of certain medications or drugs such as alcohol or tobacco, parents being closely related, or poor nutritional status or obesity in the mother. Having a parent with a congenital heart defect is also a risk factor. A number of genetic conditions are associated with heart defects, including Down syndrome, Turner syndrome, and Marfan syndrome. Congenital heart defects are divided into two main groups: cyanotic heart defects and non-cyanotic heart defects, depending on whether the child has the potential to turn bluish in color. The defects may involve the interior walls of the heart, the heart valves, or the large blood vessels that lead to and from the heart.

Heart Substructure

Congenital heart defects are partly preventable through rubella vaccination, the adding of iodine to salt, and the adding of folic acid to certain food products. Some defects do not need treatment. Others may be effectively treated with catheter based procedures or heart surgery. Occasionally a number of operations may be needed, or a heart transplant may be required. With appropriate treatment, outcomes are generally good, even with complex problems. Congenital heart defects are the most common birth defect.

In 2015, they were present in 48.9 million people globally. They affect between 4 and 75 per 1,000 live births, depending upon how they are diagnosed. In about 6 to 19 per 1,000 they cause a moderate to severe degree of problems. Congenital heart defects are the leading cause of birth defect-related deaths: in 2015, they resulted in 303,300 deaths, down from 366,000 deaths in 1990.

Molecular Pathways

The genes regulating the complex developmental sequence have only been partly elucidated. Some genes are associated with specific defects. A number of genes have been associated with cardiac manifestations. Mutations of a heart muscle protein, α -myosin heavy chain (MYH6) are associated with atrial septal defects. Several proteins that interact with MYH6 are also associated with cardiac defects. The transcription factor GATA4 forms a complex with the TBX5 which interacts with MYH6. Another factor, the homeobox (developmental) gene, NKX2-5 also interacts with MYH6. Mutations of all these proteins are associated with both atrial and ventricular septal defects; In addition, NKX2-5 is associated with defects in the electrical conduction of the heart and TBX5 is related to the Holt-Oram syndrome which includes electrical conduction defects and abnormalities of the upper limb. The Wnt signaling co-factors BCL9, BCL9L and PYGO might be part of this molecular pathways, as when their genes are mutated, this causes phenotypes similar to the features present in Holt-Oram syndrome. Another T-box gene, TBX1, is involved in velo-cardio-facial syndrome Diverge syndrome, the most common deletion which has extensive symptoms including defects of the cardiac outflow tract including tetralogy of Fallot. The notch signaling pathway, a regulatory mechanism for cell growth and differentiation, plays broad roles in several aspects of cardiac development. Notch elements are involved in determination of the right and left sides of the body plan, so the directional folding of the heart tube can be impacted. Notch signaling is involved early in the formation of the endocardial cushions and continues to be active as the develop into the septa and valves. It is also involved in the development of the ventricular wall and the connection of the outflow tract to the great vessels. Mutations in the gene for one of the notch ligands, Jagged1, are identified in the majority of examined cases of arteriohepatic dysplasia (Alagille syndrome), characterized by defects of the great vessels (pulmonary artery stenosis), heart (tetralogy of Fallot in 13% of cases), liver, eyes, face, and bones. Though less than 1% of all cases, where no defects are found in the Jagged1 gene, defects are found in Notch2 gene. In 10% of cases, no mutation is found in either gene. For another member of the gene family, mutations in the Notch1 gene are associated with bicuspid aortic valve, a valve with two leaflets instead of three. Notch1 is also associated with calcification of the aortic valve, the third most common cause of heart disease in adults.

Changes at birth

The ductus arteriosus stays open because of circulating factors including prostaglandins. The foramen ovale stays open because of the flow of blood from the right atrium to the left atrium. As the lungs expand, blood flows easily through the lungs and the membranous portion of the foramen ovale (the septum primum) flops over the muscular portion (the septum secundum). If the closure is incomplete, the result is a patent foramen ovale. The two flaps may fuse, but many adults have a foramen ovale that stays closed only because of the pressure

difference between the atria. Rokitansky (1875) explained congenital heart defects as breaks in heart development at various ontogenesis stages. Spitzer (1923) treats them as returns to one of the phylogenesis stages. Krimski (1963), synthesizing two previous points of view, considered congenital heart diseases as a stop of development at the certain stage of ontogenesis, corresponding to this or that stage of the phylogenesis. Hence these theories can explain feminine and neutral types of defects only.