Cardiovascular Diseases in Paediatrics

Commentary Article

Cardiovascular problems used to be more of a worry in the adult population than in the paediatric population. The increased prevalence of paediatric obesity, as well as the shrinking knowledge gap about the link between adult obesity and cardiovascular difficulties, has raised public awareness that non-congenital cardiovascular disorders can affect children.

Adult cardiovascular disorders, such as hypertension and peripheral vascular disease, have their origins in the foetal and childhood environment, and it is becoming obvious that childhood obesity increases cardiovascular risk in adulthood [1]. The cardiovascular system appears to be prone to harm from maternal factors and dietary habits from conception until puberty [2]. According to one study, there is a link between cardiovascular disease risk factors and obesity as early as age nine. Approximately 30% of birth defects are heart related [3].

Four out of every 1,000 live births is affected [4]. One of the most common causes of newborn death due to a birth defect is heart problems [3,5]. CHDs are structural issues caused by faulty heart or major blood artery development.

They are divided into 15 different categories. Ventricular septal defects (14% to 16%), tetralogy of Fallot (9% to 14%), transposition of the great arteries (10% to 11%), atrioventricular septal defects (4% to 10%), coarctation of the aorta (8% to 11%), patent ductus arteriosus (PDA; 5% to 10%), and hypoplastic left heart syndrome (5% to 10%) are the most common (4% to 8%) [3].

In the year 2000, more than 130,000 newborns and children with CHDs were admitted to hospitals, costing $6.5 billion in medical bills. From 1993 to 2003, CHD-related mortality decreased by 25%, but the actual number of deaths decreased by 26%. The mortality rate varies based on the newborn’s weight and the type of abnormality [3].

CHDs are more likely in kids born with a low birth weight (less than 2,500 g). In the United States, 36 thousand (nine per 1,000) malformations are projected each year, with 9,200 (2.3 per 1,000) live babies requiring invasive treatment or resulting in death within the first year of life [3].

Although invasive surgery is still required in a large proportion of cases, many CHDs are tiny and close spontaneously during a child’s first year of life. [3] For example, up to 5% of newborns (about 200,000 each year) are born with microscopic muscular ventricular septal defects that repair spontaneously and do not require emergency surgery [3].

PDA, where the defect is in the vascular structure connecting the proximal descending aorta to the roof of the pulmonary artery, is another example of spontaneous closure. [4,6] Small PDAs, sometimes known as silent PDAs, may close spontaneously or remain open for the first year of life, but the patient is asymptomatic.

Larger PDAs can lead to problems like respiratory discomfort, hypotension, and pulmonary hypertension if left untreated. Indomethacin, [6,7] ibuprofen, [8,9] or surgical ligation are used to close PDAs [9] PDAs ranging in size from medium to big might reduce lung compliance and increase breathing strain. Secondary pulmonary hypertension can also be caused by large PDAs.
References


