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Short Communication

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Atherosclerosis from the fetus to the adult

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Atherosclerosis (AS) is a focal lesion that mainly affects large and medium arteries such as the aorta, carotid, coronary, iliac, femoral, etc. It is characterized by the formation of atheromatous plaques. These lesions primarily develop in the intima, although the tunica media is also involved. The typical plaque consists of a central area with an abundant fatty substance (atheroma: pulp, papilla) and is surrounded by a dense collagenous tissue (sclerosis: hardening). This multifactorial process involves genetic, environmental, and social factors. Furthermore, it is currently considered a biological process due to the formation of atheroma, involving cell aggregation, matrix formation, and lipid accumulation. Recent research confirms that it is a chronic inflammatory disorder.[1,2]

Atherosclerosis (AS) is a progressive disease. Today, we can affirm that it can begin in fetal life as well as in early childhood in an asymptomatic form. It develops silently during the first decades of life and later leads to significant morbidity and mortality in mature and older adults. The progression of AS begins with the proliferation of the intima due to the action of multiple known and unknown causal factors, both genetic and acquired. These factors are ordered in terms of importance according to the affected vascular bed.[3,4] There is evidence that precursor cell changes in arterial walls occur in all children after the age of one year. Additionally, approximately a quarter of young people aged between 15 and 20 years are affected by non-obstructive and silent plaques.[5,6,7]

In the course of the last hundred years, cardiovascular diseases and atheromatous lesions of the coronary arteries, aorta, brain, and others have become the main public health problem throughout the world. Myocardial infarction and cerebrovascular accidents have been the leading cause of mortality in Western countries, as well as in our country, for many years.[8]

There are several theories that try to explain the origin of this disease, among them we can cite Virchow's insudation hypothesis, Rokitansky's theory of small thrombi incrustation and the response to injury, in recent years it has also been associated with infection. Especially herpesvirus, cytomegalovirus, mycoplasma pneumoniae, helicobacter pylori and Chlamydia pneumoniae. However, its origin has not yet been clearly established.[9,10]

Cardiovascular diseases are the first cause of general mortality in Chile according to the INE with 27.1% in 2016. Government agencies dedicated to health continue to indicate that, despite all the advances in the different disciplines, atherosclerosis, through its classic symptomatic expressions, continues to be the leading cause of death for humanity. Enos et al.,[11] in the 1950s found lesions in the coronaries of young American soldiers killed in the Korean War.[11]

Schwartz, in 1967, found fatty streaks in the aorta in 43% of infants under one year of age and in almost all those over the first year of life.

The earliest AS lesion is usually present in infants and young children as a lesion called a fatty streak. Studies have shown it in around 45% of children at one year of life and around 6-10 years of life they all present it.

An anatomopathological study carried out by us in the 1990s in aortas of children under one year of age found 30.1% AS lesions. Of these, lipid deposits (fatty streak) were found in 92.3% and 7.7% of the lesions presented thickening of the subintima and variable degrees of fibrosis (fibrous plaque).[12]

An autopsy study carried out in a pediatric hospital, analyzing the coronary arteries and aorta in Mexican children, found 3% fatty streaks in the abdominal aorta. The affected cases had a family history of myocardial infarction, but their nutritional status was adequate.[13]

Another pathology study of ours found AS lesions in 46% of the aorta of fetuses and newborns who died from different causes, the mothers had no associated pathology or risk factors, the most frequent location was in the abdominal aorta with 64%.[14]

Lesions found in the aortic arch in children under 3 years of age whose mothers were hypercholesterolemic were 64% smaller than those previously found in fetuses, suggesting that AS plaques may decrease after birth. The lesions progress linearly with age, but more so in children of hypercholesterolemic mothers.[15]

The risk factors are directly associated to both children and mothers with the size of the lesion, but not with the greater or lesser progression of AS.

Traditional risk factors such as diabetes, dyslipidemia, and smoking play a significant role. However, it's noteworthy that approximately half of the patients who experience a myocardial infarction do not exhibit any of these risk factors. This observation has revitalized the investigation into mechanisms that elucidate the formation and progression of atherosclerosis (AS) plaques. The nutritional status and dietary habits of pregnant mothers and young children hold pivotal importance, not only in the development of individuals but also in the prevention of chronic diseases. This is particularly relevant for atherosclerosis, the foremost cause of global morbidity

and mortality in adulthood. Atherosclerosis lesions in their initial uncomplicated stages have demonstrated the potential for regression. Numerous published studies have explored primary and secondary prevention methods related to AS regression. Prolonged use of lipid-lowering drugs and implementation of control measures targeting factors associated with its onset have shown promise in promoting regression.[4,16,17]

In addition, it is important to instill in our population to maintain healthy lifestyles, a balanced and varied diet that allows us to maintain cholesterol and triglyceride levels within normal ranges. In addition, physical activity for at least 30 minutes every day of the week helps prevent heart attacks and strokes. It is even more important to avoid classic modifiable risk factors such as obesity, sedentary lifestyles, and smoking.[1]

We think that the background and existing studies in the last 50 years allow us to propose a new scheme of the natural history of AS.[18] The initial lesions of AS are present in fetuses, children, and adolescents. We postulate that this biological process is already present in fetal life and that the preliminary lesions of AS can involute, reaching a normal endothelium, as well as in childhood and adolescence, to later progress silently over the years and become an irreversible lesion that will cause pathology and mortality in adult life. (Figure 1)

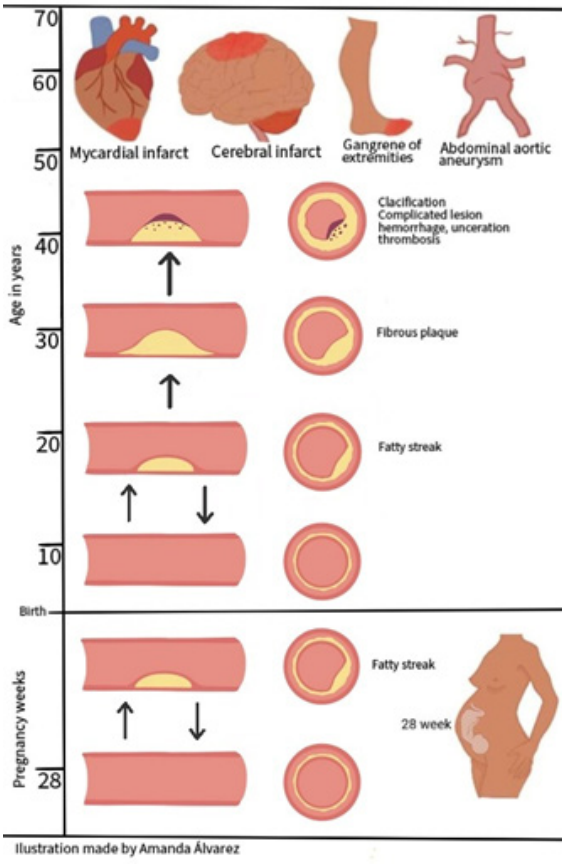

Figure 1 Natural history of human atherosclerosis[20] modified as postulated from A. Alvarez J in Concepción, Chile.

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None.

Conflicts of interest

The authors declare no conflict of interest.

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