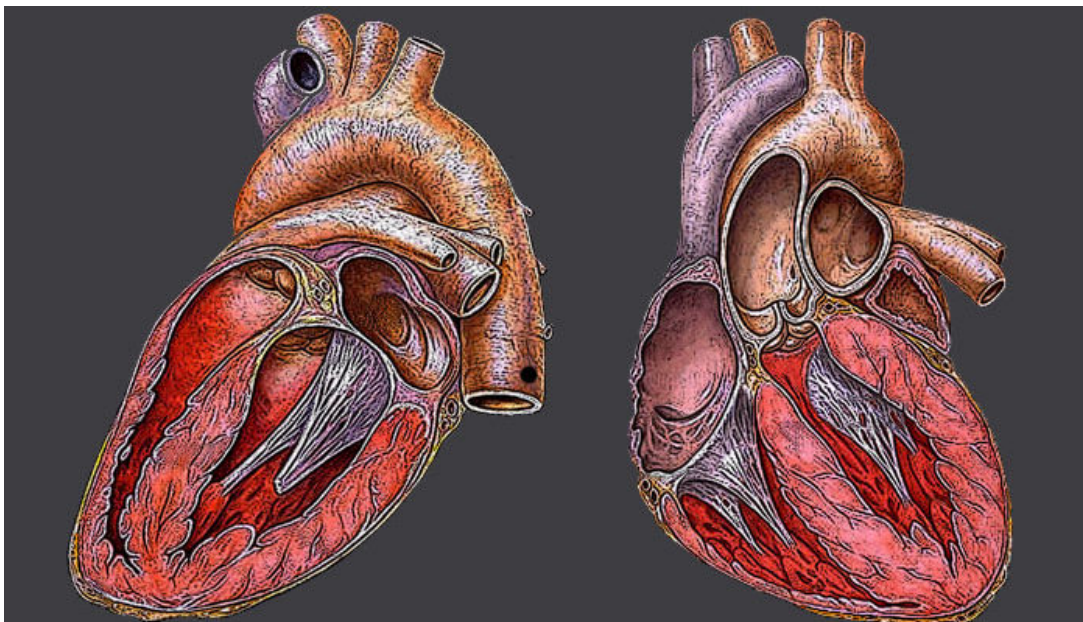


Aortic Sclerosis: Symptoms and Treatment

[See online here](#)

The disease conditions which cause a functional change in the aortic valve include aortic regurgitation and aortic stenosis. Aortic sclerosis represents a thickening of the aortic valve, which could progress to the complete stenosis of the aortic valve if not managed. Aortic sclerosis is also considered as one of the markers for coronary atherosclerosis. The treatment involves conservative management of the risk factor and treatment of mechanical stress, which leads to progress.



Definition and Epidemiology

Aortic valve sclerosis is defined as the **thickening and calcification of the aortic valve without an obstruction of the ventricular flow of blood**. This condition affects the aortic valve and may lead to aortic stenosis.

Aortic sclerosis is increasingly prevalent in older adults, as risk increases as a patient ages. As well, [hypertension](#) and [renal disease](#) are key risk factors for the development of aortic sclerosis. Tobacco smoking and elevated lipoprotein levels are additional risk factors.

Etiology

Three key factors are responsible for the development of aortic sclerosis: **lipid accumulation, inflammation, and calcification**. However, these factors need to be considered alongside the presence of certain risk factors.

Risk Factors

Factors that increase the risk of developing lipid accumulation that leads to [atherosclerosis](#) are also involved in the development of aortic sclerosis (see image). These include:



Image: Aortic valves. By: Anatomist90. License: [CC BY-SA 3.0](#)

- Abnormal level of lipids in the blood ([hyperlipidemia](#))
- An increase in the level of lipoprotein
- Smoking
- [Hypertension](#)
- [Diabetes](#)
- Age: As a person ages, the mobility of the aortic valve is reduced
- Metabolic syndrome (abdominal obesity, high triglyceride levels, low high-density lipoprotein levels, [high blood pressure](#), and high blood glucose levels) is also associated with a risk of aortic sclerosis

Imbalances in the Regulation of Minerals

There is a greater risk of aortic sclerosis in individuals with increased serum calcium, phosphate, parathyroid hormone, and 25-hydroxyvitamin D levels. Although a clear association between these conditions has been shown, a direct causative link has not been determined.

Mechanical Stress

Mechanical stress causes damage to the aortic valve, particularly the endothelium of the aortic valve. This damage leads to lipid accumulation, which, in turn, leads to the oxidation of lipids due to the development of inflammation as a resolving mechanism. Mechanical stress can be due to hypertension, ventricular hypertrophy, and advanced age, all of which are associated with stenosis of the root of the aortic valve.

Familial Association

There is also a **familial association for the occurrence of aortic valve sclerosis, as evidenced by its association with genetic factors** such as single nucleotide polymorphism, rs10455872. This is located in the lipoprotein, a gene in the intron sequence (part of the coding sequence of a gene).

This single nucleotide polymorphism is associated with an **increased level of circulating lipoprotein**, which is a risk factor for the development of atherosclerosis. In addition, LDLc-related genes, vitamin D receptors, and angiotensin-converting enzymes are also linked to the occurrence of aortic sclerosis. There is also a higher occurrence of aortic sclerosis in patients with a familial history of hypercholesterolemia.

The NOTCH1 gene, which plays a key role in intercellular signaling and transcriptional regulation, decides the fate of the osteoblast cells of the aortic valve; thus, it is also involved in the development of aortic sclerosis (especially during mutation of the gene).

The estrogen receptor alpha gene has also been shown to be one of the factors associated with aortic sclerosis in post-menopausal women. In adolescent females, it is linked to abnormal lipid levels.

Classification

According to the [ACC/AHA 2006 valvular heart disease guidelines](#), an aortic valve jet velocity of ≤ 2.5 m/s denotes aortic sclerosis. Further increases in this value constitute the grading system for aortic stenosis.

This system divides aortic stenosis into three categories:

- **Mild:** Aortic valve velocity between 2.6 and 3.9
- **Moderate:** Aortic valve velocity between 3 and 4
- **Severe:** Aortic valve velocity > 4

Other variables that are part of the classification of aortic stenosis include:

- **Mean gradient (in mm Hg):** Divided into mild (< 20), moderate (20–40), and severe (> 40)
- **Aortic stenosis severity (AVA; in cm^2):** Mild (> 1.5), moderate (1–1.5), severe (0.60–0.99), and critical (< 0.60)
- **Indexed AVA (in cm^2/m^2):** Mild (> 0.85), moderate (0.60–0.85), and severe (< 0.6).

Pathophysiology

The pathway by which the development of aortic sclerosis occurs follows the same pathology as that of [atherosclerosis](#). This involves an initial accumulation of lipid, which, on progression, causes destruction of the lining. Inflammation occurs as a restorative and healing mechanism. As this process develops, calcium deposition begins, initiating sclerosis (see image below).

Atherosclerosis

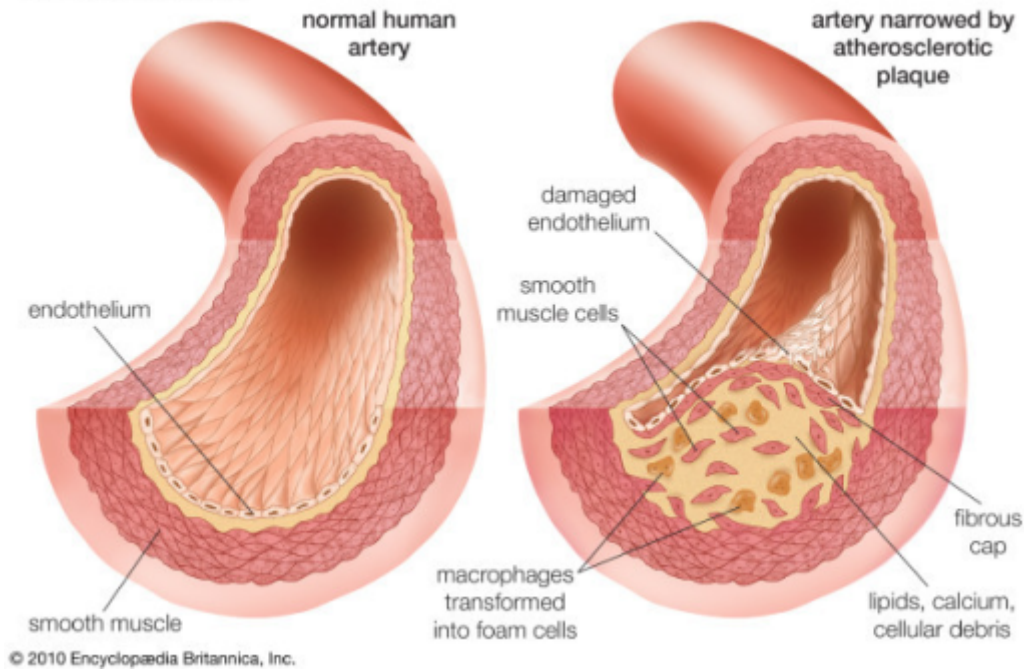


Image: Coronary atherosclerosis. By: Openi. License: [CC BY 2.0](https://creativecommons.org/licenses/by/2.0/)

Lipid Oxidation, Inflammation, and Calcification

Apolipoprotein B and E accumulate in the lining of the aortic valve, where they undergo oxidation. This oxidation leads to the destruction of the lining of the cells of the aortic valve, which, in turn, infiltrates T lymphocytes and macrophages, leading to inflammation, as evidenced by histopathology of the specimen.

The process of inflammation of the aortic valve leads to the deposition of calcium, causing calcification of the aortic valve. Inflammation also leads to fibrosis of the valve. Fibrosis and calcification are responsible for the thickened nature of the aortic valve—that is, aortic sclerosis.

This process, when left unchecked and with the persistence of risk factors such as hypertension, leads to further disease progression, ultimately leading to aortic sclerosis.

Clinical Manifestations

Because the aortic valve is not completely blocked in many patients, these **individuals present without characteristic symptoms and are asymptomatic**. Cardiovascular examination usually detects systolic ejection murmur (during the mid-systolic phase), which is a vital clue in the diagnosis of aortic sclerosis.

In some patients, however, [cardiovascular examination](#) will not detect a murmur; thus, the condition must be diagnosed through radiological techniques such as echocardiography. In patients who are asymptomatic, routine follow-ups with electrocardiograms are essential.

Progression

As noted, patients whose condition is not adequately controlled will progress to aortic stenosis. The severity of the aortic valve stenosis is one of the predicting factors in deciding the severity of the condition. As well, when an asymptomatic patient moves to

the symptomatic stage also constitutes the level of disease progression of aortic sclerosis.

Diagnosis

Transthoracic echocardiography is used to aid diagnosis in asymptomatic patients. Conditions such as aortic calcification can also be **diagnosed using a CT scan to measure the thickness of the aortic valve**.

It is important to distinguish between aortic sclerosis and aortic stenosis on echocardiography. This can be done by assessing the mobility of the valve leaflets and the blood velocities across the chambers using Doppler ultrasound.

The **surface and center of the valve will also show irregularities and an increase in signals (echogenicity)**. The velocity and mobility of the valve remain unaffected during aortic sclerosis. The more the aortic valve is impeded, however, the higher the velocity seen during echocardiography.

Differential Diagnosis

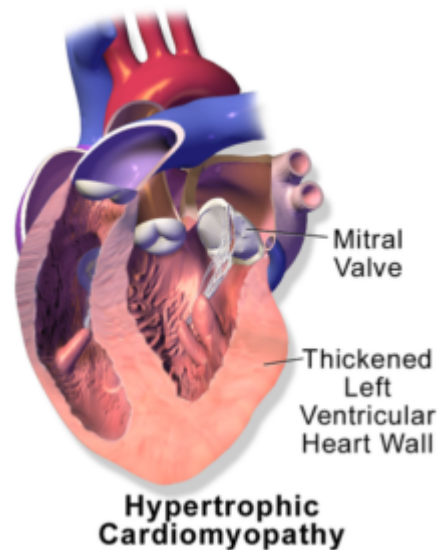


Image: Hypertrophic cardiomyopathy. By: Blausen Medical Communications. License: [CC BY 3.0](#)

Differential diagnosis includes the conditions in which systolic murmur occurs.

1. One important differential diagnosis for aortic sclerosis is aortic stenosis. Aortic stenosis can be distinguished from aortic sclerosis by the following:
 - The flow in aortic stenosis is obstructed (as assessed by an increase in velocity, as assessed by echocardiography)
 - The mobility of the valves is reduced
2. Hypertrophic [cardiomyopathy](#) can be distinguished in echocardiography by the presence of hypertrophy along the left ventricular wall. This occurs alongside outflow obstruction, which is absent in aortic sclerosis.
3. Mitral regurgitation can be distinguished by the presence of a mitral regurgitation jet, as assessed by echocardiography (see image).
4. The presence of aortic stenosis at the supra- and sub-valvular level is also a

differential diagnosis for aortic sclerosis.

Treatment

Risk Factor Modifications

Factors related to the development of aortic sclerosis and its subsequent progression, such as **smoking**, **hypertension**, **ventricular hypertrophy**, **hyperlipidemia**, and **diabetes**, need to be controlled and treated appropriately.

Other than risk factor modification, there is no active treatment required for aortic valve sclerosis, as blood flow across the valve is not affected by this condition.

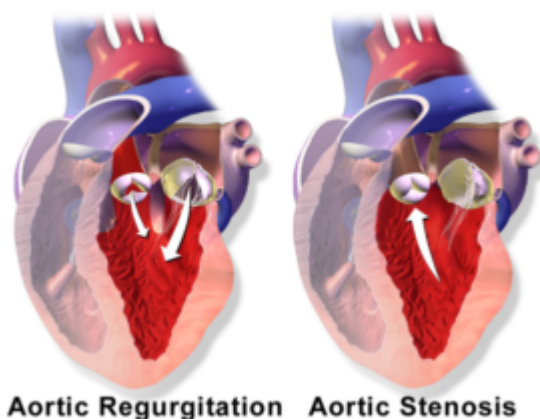
Additional Treatments

Lipid-lowering therapies, such as HMG-CoA reductase inhibitors, have not shown any marked difference in decreasing the rate of progression of the aortic valve sclerosis.

In conditions involving the chambers of the heart, stasis of the blood is thought to play an important role in the development of thrombus leading to **embolism**, which, in turn, leads to **stroke**. However, antithrombotic therapy has no role in the management of aortic valve sclerosis.

Additionally, although an imbalance in calcium-regulating hormones leads to the calcification of the aortic valve, there is no evidence to suggest that the excessive supplementation of calcium in patients suffering from **osteoporosis** leads to the development of aortic sclerosis. The use of calcium supplements, if required, is indicated in patients with aortic sclerosis.

Complications



[Image](#): Aortic valve regurgitation vs. aortic valve stenosis. By BruceBlaus. License: [CC BY 3.0](#)

One of the important reasons for ensuring the early diagnosis of aortic sclerosis is the patient's risk of progressing to aortic stenosis. Aortic sclerosis is also a risk factor for other cardiovascular diseases.

The damage to the aortic valve also leads to the development of other conditions of aortic dysfunction, such as **aortic regurgitation** (see image) and its related complications.

Prevention

Patients with the risk factors of smoking, hypertension, diabetes, and chronic renal failure need to be managed appropriately at an early stage in order to inhibit the development of aortic valve sclerosis. Patients with a family history of aortic sclerosis need to have regular follow-ups to prevent the development of the disease.

References

Prasad Y, Bhalodkar NC. Aortic sclerosis – a marker of coronary atherosclerosis. Clin Cardiol. 2004 Dec;27(12):671-3.

[Genetic Associations with Valvular Calcification and Aortic Stenosis](#) via nih.gov

Nordstrom P, Glader CA, Dahlen G, Birgander LS, Lorentzon R, Waldenstrom A, et al. Oestrogen receptor alpha gene polymorphism is related to aortic valve sclerosis in postmenopausal women. J Intern Med. 2003 Aug;254(2):140-6.

[Aortic sclerosis: not an innocent murmur but a marker of increased cardiovascular risk](#) via nih.gov

Gharacholou SM, Karon BL, Shub C, Pellikka PA. Aortic valve sclerosis and clinical outcomes: moving toward a definition. Am J Med. 2011 Feb;124(2):103-10.

Drolet M-C, Couet J, Arsenault M. Development of aortic valve sclerosis or stenosis in rabbits: role of cholesterol and calcium. J Heart Valve Dis. 2008 Jul;17(4):381-7.

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