Aortic regurgitation

Aortic regurgitation is caused by the aortic valve not closing. Aortic valve insufficiency can be caused by **dilation of the aortic root or damage to the valve itself,** *both as a result of acquired and congenital defects.*[1] ***Left ventricular volume overload*** *dominates the pathophysiology of aortic regurgitation.***.**[1][2] ***The diagnosis of this defect is sometimes complicated by the ``long asymptomatic period The diagnosis of this defect is sometimes complicated by the*** .[1][2] *The diagnosis of this defect is sometimes complicated by the ``long asymptomatic period***.**[1] In addition to clinical examination, the basic examination method is echocardiography.[1][3]Both cardiosurgical intervention and catheter replacement of the valve can be used in the treatment.

Etiopathogenesis

The causes of the development of aortic regurgitation can be divided into two main categories - **dilation of the aortic root and involvement of the valve itself'.** *Dilation of the aorta (not only its root) can be the result of genetic syndromes, especially Marfan syndrome and Ehlers-Danlos syndrome. There is also an increased incidence in ankylosing spondyloarthritis.*[1][2] *Another important cause of dilatation of the aortic root is "hypertension" with consequences in the form of increased stiffness of the aortic wall, loss of elasticity and atherosclerotic damage. Aortic regurgitation also often occurs on the basis of infective endocarditis, bicuspid valves, as part of post-rheumatic disease, myxomatous disease, calcification (degenerative) valve disease, chest trauma or aortic dissection. Other less frequent causes are involvement of the aortic valve as part of systemic inflammatory diseases (SLE), infiltrative and persistent diseases or aortitis.*[1][2][3][4] *It also often occurs together with aortic stenosis*. The pathophysiology of aortic regurgitation is dominated by *volume overload of the left ventricle*, which in most cases is chronically progressive.[2][3] Thus, the left ventricle dilates over time and a picture of eccentric hypertrophy develops.[3] In the initial stages, sometimes even for several years, the dilatation may not be accompanied by a decrease in the systolic function of the left ventricle or a significant increase in the filling pressures of the ventricle, and thus by diastolic dysfunction. This defect therefore remains asymptomatic for a very long time, which complicates not only the diagnosis but also the treatment procedure, because there is only a relatively limited period of time for effective intervention before the irreversible impairment of the systolic function of the ventricle.[1] Dilation of the left ventricle leading to increased consumption of the myocardium, its systolic (and later also diastolic) dysfunction together with reduced diastolic blood pressure ultimately lead to hypoxic involvement of the myocardium and "left-sided heart failure".[5] This can be further complicated by secondary mitral regurgitation, which together with reduced ventricular compliance due to progressive dilation leads to increased left atrial pressures.[5]

Clinical picture

Aortic regurgitation is usually asymptomatic for a long time (up to decades). It is manifested by **fatigue**, **exertion dyspnea, loss of performance** and less often *by angina pectoris.*[1][2][3]Characteristic is an **increase in pulse pressure** when the diastolic pressure is reduced while the systolic pressure is simultaneously increased.[1][2][3] atrial fibrillation and extrasystole are poorly tolerated by patients with aortic regurgitation due to increased postextrasystolic ventricular volume.[1]On the contrary, good exercise tolerance is indicated in the development of heart failure, when tachycardia shortens the duration of diastole, and therefore also regurgitation.[6] Patients with acute aortic regurgitation present with pulmonary edema and cardiogenic shock.

Physical Finding

On auscultation, we detect a **blow'''***diastolic decrescendo''* **murmur** above the aortic orifice with a maximum at Erb's point (3.-4 .intercostal parasternal).[1][3] An Austin Flint diastolic mitral murmur reminiscent of mitral stenosis may be heard at the tip, which is caused by premature closure of the anterior leaflet of the mitral valve by the flow of regurgitated blood from the aorta.[1][6] Thereby, the anterior mitral leaflet causes a relative stenosis of the mitral orifice. The already mentioned large range of systolic and diastolic blood pressure is typical, which has its correlate in the nimble "Corrigan's pulse" on the carotids.[6]

Signs of advanced disability are, for example, *Quincke's capillary sign* (the edge of the lunula pulsates when the nail is pressed), *Musset's sign* (shaking of the head with pulsation) and *Müller's sign* (shaking of the uvula with pulse).[2][3][6]

Diagnosis

Transthoracic and esophageal echocardiography is the basic diagnostic tool for aortic stenosis. It is not unusual for aortic regurgitation to be an incidental finding in asymptomatic patients, even in the case of this examination.

'*X-ray of the chest* usually reveals an enlargement of the cardiac shadow, dilatation in the area of the ascending aorta, or signs of congestion in the small circulation. **ECG** is also non-specific, usually signs of left ventricular hypertrophy and load, or a tilt of the cardiac axis to the left, are detected. Magnetic resonance imaging or

computed tomography is mainly used to assess dilatation of the aorta or more detailed imaging of other structures. Cardiac catheterization serves to rule out ischemic heart disease.[2][3][6]

The sovereign diagnostic method is transthoracic (TTE) and esophageal (TEE) echocardiography. Examination is able to reveal the etiology of aortic regurgitation (eg, bicuspid valve) to a large extent. The dimensions and function of the heart compartments (hemodynamic impact of the defect) are assessed, as well as the morphology of the valve, and with the help of doppler imaging, it is possible not only to detect but also to quantify various parameters of regurgitation, e.g. regurgitation fraction and volume. Esophageal echocardiography also offers the option of detailed 3D imaging of the valve.[2][3][6][7]

Examination of natriuretic peptides is also appropriate in selected patients.

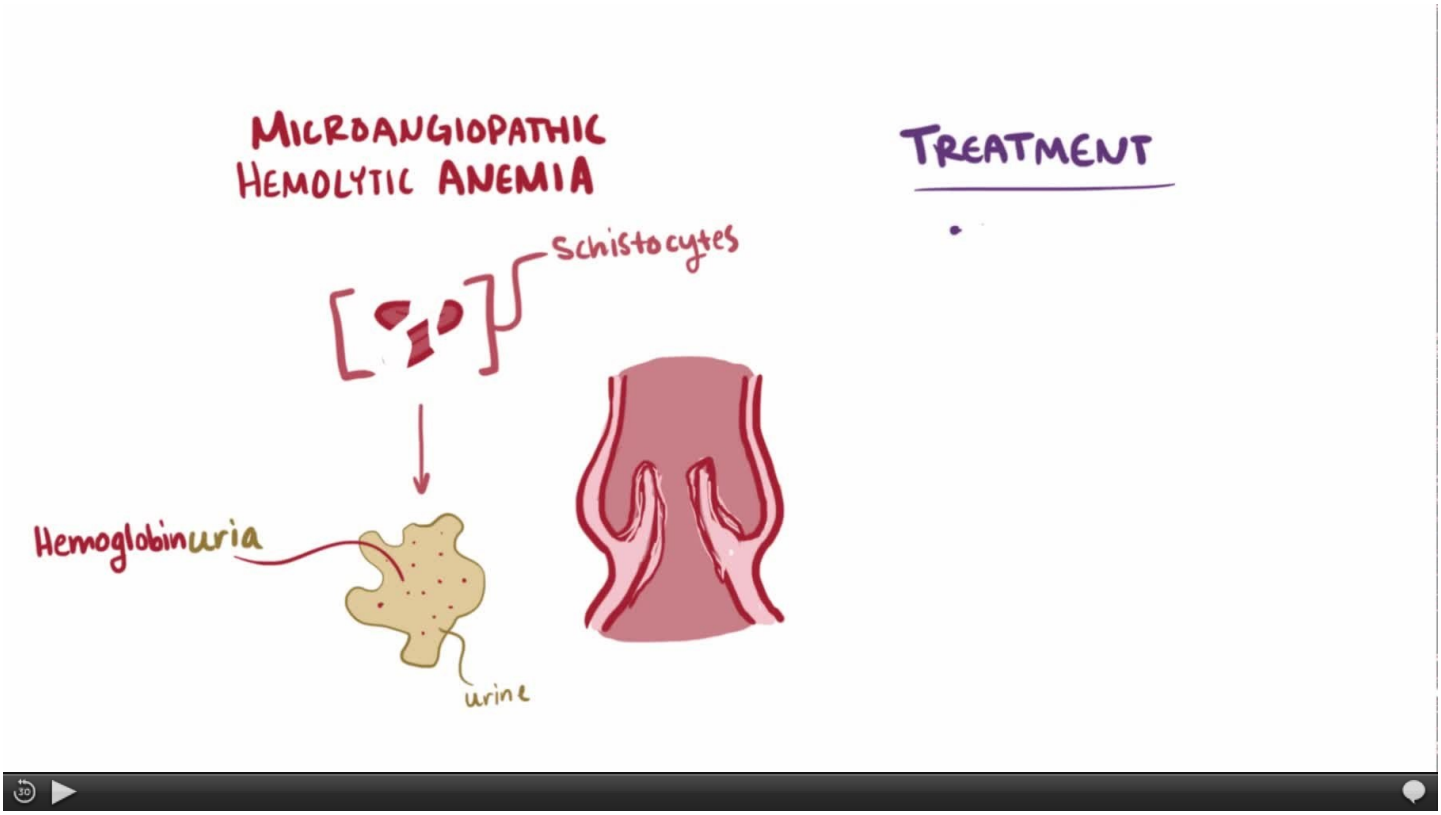
Treatment

Patients with hemodynamically insignificant and asymptomatic aortic regurgitation usually do not require specific therapy.[2]However, this is necessary in case of arterial hypertension. In hypertensive patients and incipient left ventricular dilatation, we try to reduce diastolic hypertension medically by administering ACE inhibitors, which also have a positive effect on remodeling, as well as calcium channel blockers and, in symptomatic patients, diuretics. .[1][2][7] Beta-blockers must be administered with caution due to possible bradycardia, which is poorly tolerated by patients.[2]

Treatment methods for significant aortic regurgitation are cardiosurgical **aortic valve replacement' *or*** *aortic valve and root replacement* (*Bentall operation*) with dilatation of the bulb and ascending aorta. In indicated cases, it is also possible to perform valve-preserving operations, e.g. for dilatation of the aorta with normal morphology of the aortic valve leaflets.[2][6][7] The surgical solution is indicated in ``symptomatic patients *with significant aortic regurgitation, in ``asymptomatic patients with systolic dysfunction or significant dilatation of the left ventricle*, or also in significant dilatation of the ascending aorta. Moderately significant aortic regurgitation can be solved surgically in certain patients with a simultaneous procedure on another valve, on the aorta or aortocoronary bypass.[7] An increasingly developing technique, which is used primarily in high-risk patients, is catheter-based aortic valve replacement (*TAVI* or TAVR).[1]

The prognosis of asymptomatic aortic regurgitation without left ventricular systolic dysfunction is good. In symptomatic patients with the development of heart failure or significant dilatation of the left ventricle, the prognosis is worse, without surgery the mortality rate is approximately 20% per year.[6] It is therefore essential to *correctly time the interventional solution*, ideally before the development of symptoms and significant systolic dysfunction, when the patient's prognosis is best.

Summary Video and Listening Finding



Links

Related Articles

- Congenital heart defects • Acquired heart defects
- Aorta abdominalis • Aorta thoracica
- Aortic Stenosis • Abdominal Aortic Bulge
- Elastic artery (histological preparation)

External links

- Aortální regurgitace - Šelest - Audio nahrávky (TECHmED) (https://www.techmed.sk/ejekcny-systolicky-selest/)
- Šelesty pri Aortální regurgitaci - Audio nahrávky (TECHmED) (https://www.techmed.sk/selesty-pri-aortalnej-regurgitacii/)
- Pracovní text z Interní propedeutiky: Aortální regurgitace (http://int-prop.lf2.cuni.cz/zof/vysetreni/srdceva_n.htm#ar)

Resources

- ČEŠKA, Richard, et al. *Interna.* 3. edition. Triton, 2020. ISBN 978-80-7553-782-9.
- MANN, Douglas L, et al. *Braunwald´s Heart Disease : A Textbook of Cardiovascular Medicine.* 10. edition. 2015. ISBN 978-0-323-29429-4.
- SILBERNAGL, Stefan – LANG, Florian. *Atlas patofyziologie.* 2.. edition. 2012. ISBN 978-80-247-3555-9.
- OTTO, Catherine M. *Textbook of Clinical Echocardiography.* 6. edition. Elsevier, 2018. ISBN 978-0-323-48048-2.
- HLUBOCKÁ, Zuzana. *Chlopenní vady* [lecture for subject Kardiovaskulární medicína, specialization Všeobecné lékařství, 1. LF UK]. Praha. -. Avaliable from <https://int2.lf1.cuni.cz/1LFIK-96-version1-chlopenni_vady_web.pdf>.
- BAUMGARTNER, Helmut – FALK, Volkmar – BAX, Jeroen J. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. *European Heart Journal.* 2017, y. 36, vol. 38, p. 2739-2791, ISSN 0195-668X. DOI: 10.1093/eurheartj/ehx391 (http://dx.doi.org/10.1093%2Feurheartj%2Fehx391).
- STANĚK, Vladimír. *Kardiologie v praxi.* 1. edition. Axonite CZ. 2014. ISBN 978-80-904899-7-4.

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10.1093/eurheartj/ehx391 (http://dx.doi.org/10.1093%2Feurheartj%2Fehx391).
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