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ACQUIRED VALVULAR HEART DISEASE IN CHILDREN: OUR EXAMPLES

Fatima Begić

Bihać Cantonal Hospital

Abstract

The most prevalent **congenital heart defects** for paediatric cardiologists to deal with have traditionally been **cardiomyopathy** (primary disease of the heart muscle), **arrhythmia** (irregular heartbeat), and **acquired heart disease**. More recently, most developed countries are also engaged in combatting **fetal echocardiography**.

Rheumatic heart disease and Kawasaki disease are the two most common types of acquired disease in children. Rheumatic heart disease occurs in children aged 5-15 years, and Kawasaki disease is most common in those younger than 5 years. Both diseases represent inflammatory conditions. Kawasaki disease affects the lymph nodes, and blood vessels of the heart. Rheumatic disease affects the connective tissue, and causes inflammation of the joints, and especially inflammation of the heart.

Bacterial infections can affect not only the valves of the heart, but also the heart muscle, and the lining of the heart. Cardiac valves can be affected by systemic diseases, rheumatoid arthritis, and lupus.

Key words: heart valve, rheumatic heart disease, heart muscle, child

Introduction

Heart valves are elements of the cardiac apparatus, which enable it to work as a pump, supplying every cell with blood. When the valves are open, they allow blood to flow, and when they are closed they prevent it from returning. There are four heart valves: aortic, mitral, pulmonary and tricuspid. Heart valves do not work always properly. Congenital valvular disease is mainly evident immediately after birth, whereas more minor defects (such as bicuspid aortic valve disease), which are well-tolerated in childhood, are usually discovered later.

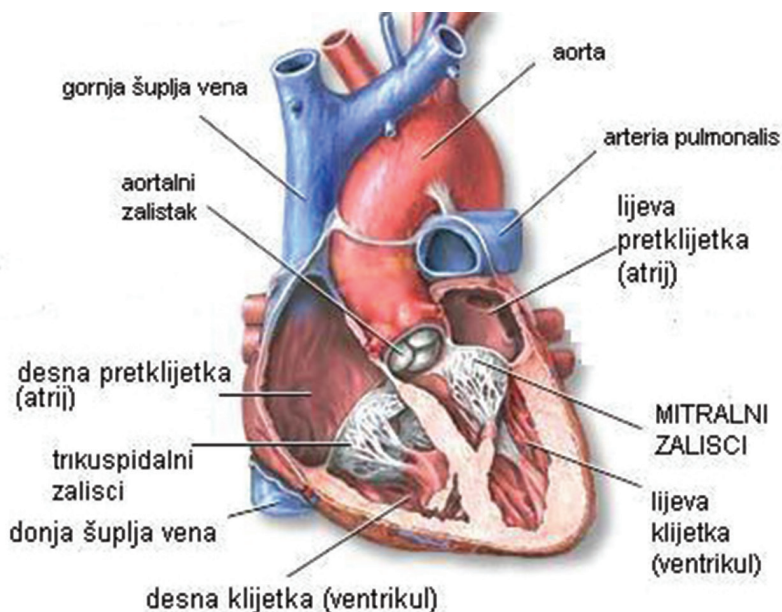


Figure 1. Normal Cardiac Valve

Acquired valvular heart diseases occur later in childhood. Almost all acquired heart diseases are rheumatic. Involvement of the mitral valve occurs in $\frac{3}{4}$, and aortic in $\frac{1}{4}$ of all cases with rheumatic heart disease. Stenosis and regurgitation of the same valve usually occur together. Isolated aortic stenosis of rheumatic genesis is extremely rare. Involvement of the tricuspid valve varies, and that of pulmonary valves almost never occurs.

Mitral regurgitation is the most common valve involvement in children with rheumatic diseases. Children do not usually show symptoms; fatigue and palpitations are rarely present. The main symptom is a systolic murmur at the apex.

Mitral stenosis, although rare in children, is the most common valve involvement in adult rheumatic patients. Most patients have no visible symptoms, but the main symptom is a diastolic murmur over the apex.

Aortic regurgitation is less common than mitral regurgitation, as most patients have a disease associated with the mitral valve. Patients with mild regurgitation have no symptoms. Tolerance to this disease can be reduced with severe regurgitation or heart failure. The patient can be without symptoms for a long time, but if symptoms begin to develop, the condition of most patients will rapidly deteriorate. Anginal pain, premature ventricular contractions, and congestive heart failure are danger signs.

In adult patients, degenerative diseases – such as senile aortic calcification – are the main cause of changes to the valves.

Rheumatic heart disease is a result of idling rheumatic fever, and occurs after untreated or improperly treated infection of the upper respiratory tracts, caused by the bacteria **streptococcus**. The main symptoms are manifested in the joints and the heart, but rarely in the skin and nervous system. This disease can lead to permanent damage of the heart. Since the introduction of penicillin to treat inflammation of the pharynx, there has been a sharp drop in the frequency of occurrence of rheumatic fever.

Rheumatic fever occurs in people who have a congenital tendency to the disease. After contact with bacteria, the immune system reacts not only against the bacteria, but also against the patient's own tissues and organs. The most common sign – **arthritis** – leaves no lasting damage, but carditis (the most serious sign of rheumatic fever) if left untreated can have lasting effects on the heart. Rheumatic disease can affect any heart tissue, manifesting as pericarditis, myocarditis, or endocarditis.

Symptoms that indicate carditis are: tachycardia; quiet tones; cardiac murmur.

The doctor must assess the condition of the heart by listening to the patient's heart-beat with a stethoscope. Discovery of a heart murmur is evidence that a rheumatic inflammation of the heart muscle is present.



Figure 2. Clinical examination of a child's heart

ECG is also used to detect heartbeat irregularities.

A more accurate method for the diagnosis of carditis is **ultrasound scanning**, which shows the structure of the heart, and blood movement. If there is damage to the valves, ultrasound examination helps to detect the damage on time.

These methods are totally painless, and ultrasound scanning is harmless. There is no reason for the child not to be calm.

Rheumatic fever is treated with antibiotics. Streptococcus may remain hidden in the tonsils, and can stimulate the immune system.

In patients who have already had rheumatic fever, the use of the drug Extencillin is required every 3 weeks to prevent a relapse of the disease, which is common in the first 3-5 years. With each relapse the risk of damage to the heart increases. Antibiotics can be used for at least 5 years (or until 18 years of age), but no longer than this by those patients without heart damage. For patients with a damaged heart, it is recommended that antibiotic protection is taken until 40 years of age.

The prognosis of the disease depends on the severity of the clinical picture. If carditis was present at the first attack of the disease, the risk of later damage is increased. The most severe forms of heart failure require surgical intervention to replace the heart valves.

Case Study

Example 1:

A girl born in 1997, and with the initials Č.A., comes to the Department of Paediatrics for the first time at the age of 14. She was referred from the Health Centre with Dg: Cor in obs.

Anamnesis: Denies having frequent sore throats. Present illness began about a month previously, with fatigue and exhaustion. A heart murmur, which was not previously present, is auscultated by a pediatrician.

During a **clinical examination**, an asthenic material (afebrile, eupnoic) was found. The pharynx was calm. In the heart: hyperdynamic ictus; clear sounds; heartbeat accelerated to 120/min; holosystolic murmur over the ictus with an intensity III/6; quiet diastolic murmur over III ICPO left; sat O₂ 97%; RR 110/55 mmHg. Stomach at chest level, soft; liver not palpably enlarged; no aedema; pulse a.radialis et Femoral +.

Laboratory findings: Se 17, CRP 5.6.L:6.9.E: 4.7 Hb: 130, hct 0.39 tromb.299. ASTO 1180.RF neg. Thyroid hormones normal.

RTG pulmo et cor: The heart shadow was not enlarged, with a straightened left contour of the heart.

EKG: Sinus rhythm, frequency 110/min, normogram, PR 0.18 sec, high R tines in left precordial leads, T wave positive from V2-V6.

Heart ECHO: Situs solitus, segmental structure of the heart tidy. Left cardiac chambers enlarged. LVDd 60 mm (up to 49 mm), LVSD 40 mm (up to 37 mm), S and PW 8 mm, FS 34.6%, aortic output normal. The mitral valve morphology had changed, and old post-inflammatory changes were visible, especially on the front cuspid, which slightly protruded with **significant mitral regurgitation v=3.98 m/sec, PG**

63,5 mmHg. Tricuspid regurgitation was not significant. The aorta was trileaflet with a diameter of 27mm, LA enlarged, LA/AO 1.81. **Significant aortic regurgitation v = 3.80 m/sec, PG 57,7 mmHg.** Pericardial separation 2 mm, myocardial contractility tidy. Septum continuous.

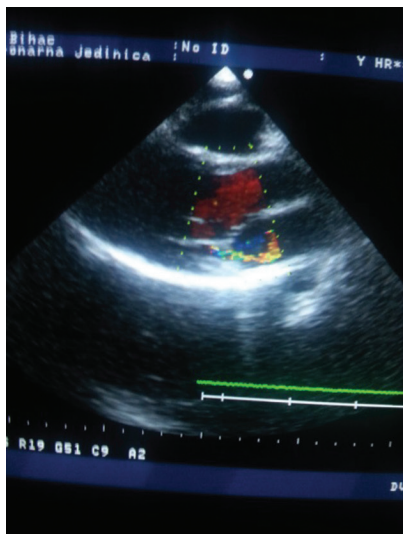


Figure 3. Mitral regurgitation



Figure 4. Aortic regurgitation

Treatment includes Extencillin for 21 days, non-steroidal and anti-rheumatic drugs, and an ACE inhibitor. After a month the non-steroidal anti-rheumatic treatment is excluded, and other therapies are included.

Dg: Vitium cordis aorto – mitrale rheumatica.

Extencillin and ACE inhibitor continued.

The patient now visits a cardiologist every 6 months.

She has recently begun to complain of fatigue, and her needs tend towards referral to a larger clinical centre. The patient has been examined by pediatric and adult cardiologists. In addition to an ultrasound of the heart, a Holter ECG was ordered (there were no rhythm disorders) and ergometry was undertaken, although it was subsequently discontinued due to fatigue.

Dg: Insuffitientio valvulae aortalis et mitralis gradus medii/gravis.

Cor compensatum.

Regular monitoring with ultrasound is recommended, every 6 months.

This patient is on the list for surgery for her heart defect, but it has not yet occurred.

When should surgery on valves be done, and which method should be applied?

The treatment options for valvular heart disease are well-known: medicamentosa; balloon valvuloplasty; surgical replacement therapy; and surgical valve reconstruction.

Balloon valvuloplasty, or percutaneous mitral commissurotomy, is a method for treating mitral stenosis without mitral regurgitation and calcification.

In cases of mitral regurgitation, **surgical correction of the mitral valve** is preferable to its replacement. Anticoagulant therapy is not required, and the mortality rate is less than 1%. Replacing the valve is necessary if the valve is thickened, or significantly deformed.

Another possibility for surgical treatment is to **install an artificial valve**, which may be mechanical, biological or homograft. After installation of the mechanical valve, the patient must receive lifelong anticoagulation treatment, with regular control of coagulation parameters to determine the optimum dose of the drug. After reconstruction of the valve or biological valve installation (i.e. bioprosthesis) such therapy is needed in the first three months after surgery, while maintaining a target value of INR 2.5

The goals in the treatment of valvular heart disease are: a reduction or elimination of the symptoms of the disease; avoiding damage to the left ventricular function; improvement to quality of life; prevention of complications; and mortality reduction.

Example 2:

H. E. came to the department of Paediatrics in 2003 at the age of 7, with Dg: Tachicardia. Carditis susp.

Anamnesis: He had been ill for about a month, with fever, poor appetite, exhaustion, and sweating.

Clinical examination: well-developed for his age, subfebrile, distressed, sweaty, tachypneic.

His pharynx was calm, but breathing was weakened in the left basal lung. Heart action was rhythmic, tones quieter, no sign of a murmur, frequency 124/min, RR 121/71 mmHg. The liver was not palpably enlarged. No aedema.

Laboratory findings: SE 90/125, CRP 118 gr. 72%.L. 11.8. E: 3.95 Hb:196, hct 0,32. tromb.476. ASTO 1382, fibrinogen 7.6. CPK 15, CPK Mb8.Urea 3,7 GUK 6,6 Rf neg,

ECG: Normogram, sinus rhythm, frequency 120/min, voltage QRS of complex tidy, T flattened U II, AVF, a neg from V1 to V6.

RTG pulmo et cor: Significantly increased whole cardiac shadow without infiltrative opacities in the lung parenchyma.

Heart ECHO: Situs solitus, segmental structure of the heart tidy. Heart holes not enlarged. LVDd 38 mm, LVSD 24 mm, S 7 mm, PW 6 mm, FS 36.7%. Pulmonary and aortic flow rates tidy. Minor tricuspid regurgitation. Weaker kinetics of interventricular septum, smaller pericardial effusion.

Abdominal ultrasound tidy.

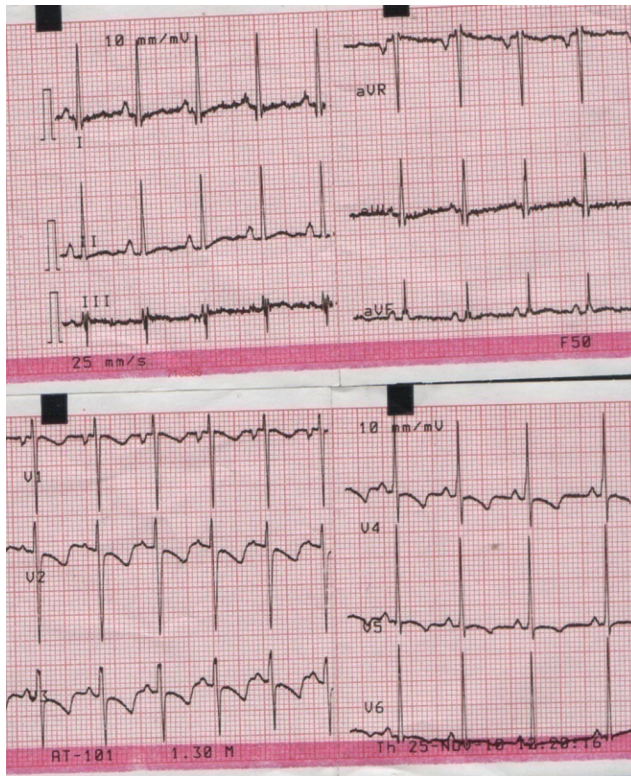


Figure 5. ECG

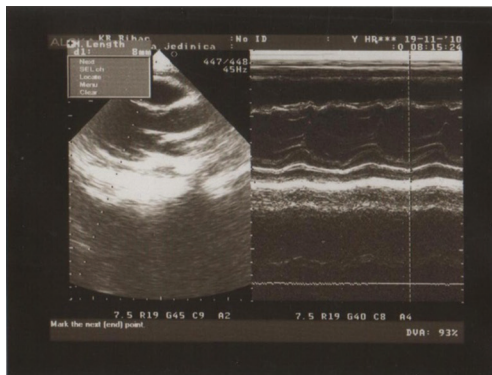


Figure 6. B and M Mod pericardial effusion



Figure 7. B Mod pericardial effusion

Upon receipt of results, the treatment included: penicillin, a non-steroidal antirheumatic, a diuretic, and corticosteroids.

The general condition of the patient is gradually improving, with less dyspnoea. Tachycardia is reduced. On the fourth day, he showed a systolic murmur and/6 auscultated with quieter tones. Pericardial effusion is gradually reducing.

Control findings: SE 24/38, CRP 38,6 ASTO 1023, fibrinogen 2,3. L. 20,28 (corticosteroid therapy).

On the tenth day, penicillin was excluded, and Extencilin included. Corticosteroids are being reduced, as are diuretic doses.

Heart tones are clearer, their frequency decreased. In the ultrasound, pericardial effusion has retreated, mobility of interventricular septum is better, and there are no changes to the heart valves, FS 38.1%. Control RTG of lungs and heart: cardiac shadow is smaller.

Discussion

Thanks to the primary and secondary prophylaxis of streptococcal infections, rheumatic heart disease is rare today.

If a child has a mild streptococcal infection, usually inflammation of the pharynx, and it is not treated, there is a chance of 1:1000 that rheumatic fever will develop. This figure increases to 3:100 if the infection is stronger.

Keeping in mind that a very small number of children suffer from rheumatic diseases, it is believed that other factors, such as a weakened immune system, participate in the development of the disease.

Clinical picture: Clinical symptoms and signs vary depending on the organ or system affected by the disease.

Some general symptoms are typical for an inflammatory process in the organism, and these are: fever, rapid heartbeat, fatigue and general malaise, loss of appetite accompanied by weight loss, sweating, headaches, bleeding from the nose, and vomiting.

Localised rheumatic symptoms include: migratory polyarthritis, rheumatoid carditis, chorea minor, subcutaneous rheumatoid nodules, and erythema marginatum, or anulare.

Diagnosis is based on the anamnesis of the idled streptococcal infection, clinical examination, laboratory findings, raised SE and CRP values, fibrinogen, and ASTO titre.

According to the Jones criteria, if there are two major or one major and two minor diagnostic criteria present, it can be assumed with high probability that the patient has acute rheumatic fever. If these criteria are accompanied with any of the above-mentioned symptoms, it is likely that an existing streptococcal infection preceded the appearance of the clinical picture of acute rheumatic fever, but without signs of carditis.

Often the clinical picture of rheumatic diseases pass asymptomatic, without arthritis. Carditis symptoms are often mild and can be anticipated, and the child is not treated.

The first symptom of idled rheumatic diseases can be a heart murmur that had not been present earlier. At this point, changes to the valves are already permanent, and over time they will require surgery.

When to operate on valves is a contentious question among cardiologists and cardiovascular surgeons

Many cardiologists believe that a need to think about surgery exists at the moment when symptoms of heart failure develop. Cardiologists and cardiovascular surgeons calculate whether and when to intervene. This is crucial for a successful surgery and further RCA work. Rarely is a patient sent to surgery immediately after the discovery of a defect. Sometimes two or more years will pass until the intervention, depending on the individual case.

Another issue for cardiovascular surgeons is which surgical approach to apply. There are typically two methods to choose from. One is the so-called “plastic surgery of heart surgery”, where the valve is repaired rather than replaced, as is usually the case with mitral valves. This is the right method for a significant percentage of diseases, although all surgical interventions are very demanding. However, valve replacement surgery has the advantage of the patient not requiring long-term post-operative anticoagulant therapy. **The second option is replacement of the valve.** This is done with two types of artificial prosthesis. One type is a **mechanical valve**, made from titanium, which effectively lasts for a lifetime (at least 20-30 years). Another type, **biological prosthesis**, is made from porcine valves or bovine pericardium. These valves have the advantage of not requiring anticoagulant therapy, but there is also a drawback – they have a shorter lifetime, of only about ten years.

If the patient is treated surgically at the right time, he will have a normal life expectancy, provided he undergoes permanent anticoagulation treatment, and takes regular prothrombic time tests.

In another case, a patient is in the acute phase of heart disease, with symptoms of myocarditis and pericarditis.

Treatment can begin immediately, using antibiotics, a non-steroidal antirheumatic, and corticosteroids. Clinical symptoms of carditis will quickly retreat, and changes to the valves did not develop.

It is important to establish the aetiology of carditis. Is there arthritis? Is the disease infectious? Bacterial? Rheumatic? Idiopathic?

In long-term monitoring the prognosis is good, without congenital changes to the valves.

Conclusion

Although heart defects in paediatric cardiac medicine are predominantly congenital, paediatric cardiologists are also faced with cases of acquired heart disease. Almost all acquired heart disease is rheumatic. Thanks to the primary and secondary

prophylaxis of streptococcal infections, rheumatic heart diseases are becoming less frequent. The disease is often asymptomatic, and as a result is not treated. In this situation, heart disease can develop many years later.

When a heart defect forms, it becomes a question of when is the best time to perform surgery, and which method to use: valve replacement or valve correction.

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THE IMPORTANCE OF PHARMACOLOGICAL STRESS IN MYOCARDIAL PERFUSION SCINTIGRAPHY

Šejla Cerić, Elma Kučukalić-Selimović

Clinic of Nuclear Medicine, University Clinical Centre of Sarajevo

Introduction

Myocardial perfusion scintigraphy is a non-invasive diagnostic procedure for assessing coronary flow in the myocardium. It is used for the detection and assessment of the extent of ischaemia and necrosis, the assessment of myocardial viability, and prognosis and risk stratification of coronary disease. The radiopharmaceuticals that are routinely used for this procedure are technetium-labelled monovalent cation complex Tc-sestamibi and Tc-tetraphosphine for SPECT (Single photon emission computed tomography), and Nitrogen-13 ammonium and Rubidium 82 for PET (Positron emission computed tomography).

After the radiopharmaceutical is administered intravenously, it accumulates in myocytes in proportion to the flow through the coronary artery. The two most important factors for uptake in the myocardium are the size of coronary flow and integrity of myocytes. In a healthy coronary artery, myocardium steadily accumulates the radiopharmaceutical, while myocardial segments that supply the stenosed or occluded coronary arteries display reduced or absent uptake. This difference increases significantly during stress to the heart.(1) Myocardial perfusion scintigraphy consists of two parts: stress imaging (applications of radiopharmaceuticals after stress), and rest imaging (applications of radiopharmaceuticals when the heart is at rest).

In normal myocardial perfusion study, there is homogenous radiotracer distribution in both stress and rest images (Figure 1 and 2). If information on the scintigram is defective (or lacking) after stress repairs compared to the scintigram at rest, a reversible defect, or ischemia, is indicated. If the defect on the scintigram after stress persists on the scintigram at rest, this indicates an irreversible defect, occurring as a result of myocardial infarction and caused by the absence of myocytes. (Figure 3 and 4)

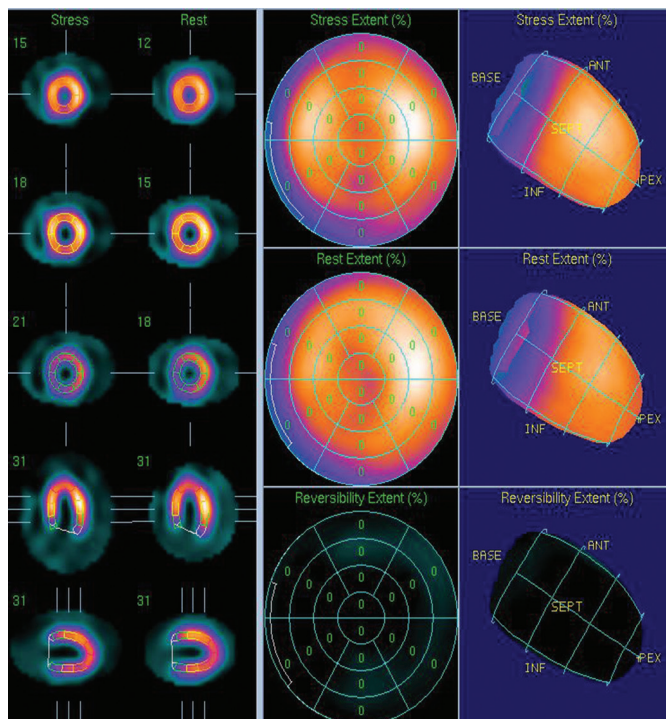
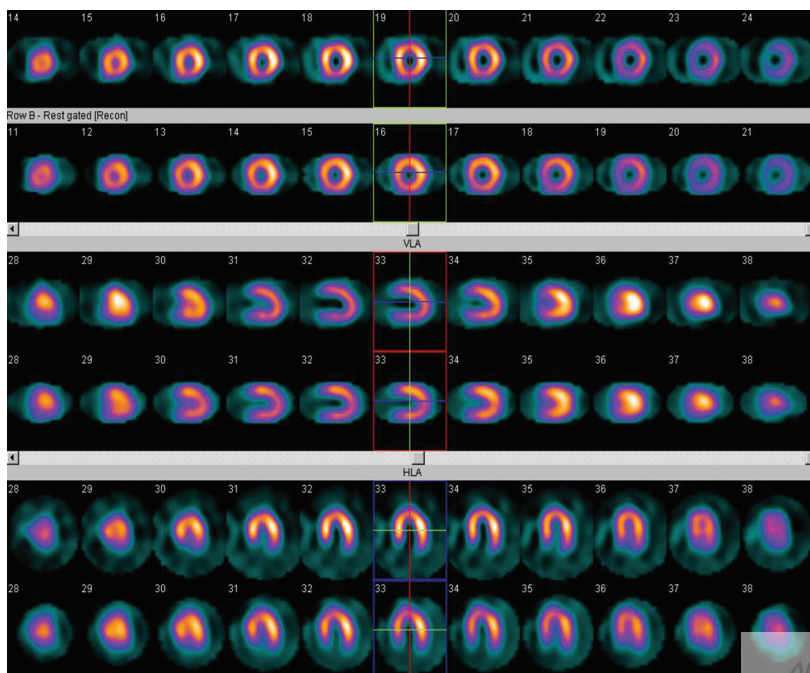


Figure 1 and 2. Normal myocardial perfusion SPECT – homogenous radiotracer distribution in both stress and rest images

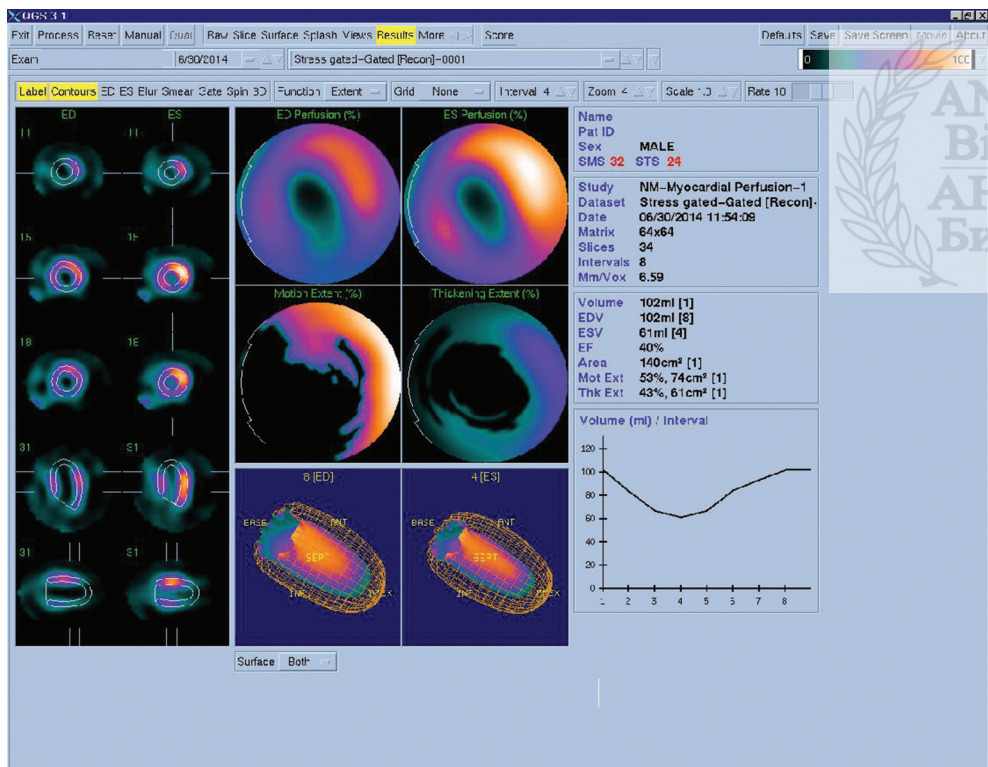
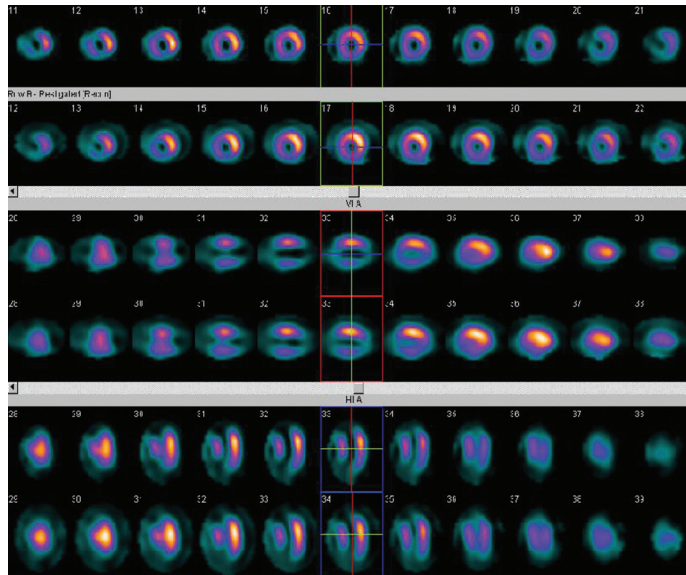


Figure 3. and 4. Abnormal myocardial perfusion SPECT. Stress images show an absence of perfusion in the apex and apical segments of the inferior wall and septum. In the at rest images, defects persist but with a slight improvement to perfusion in the inferior wall and septum. This indicates scarring with surrounding ischaemia

Single photon emission computed tomography

Single photon emission computed tomography produces images of the heart in three planes: two parallel and one perpendicular to the long axis of the left ventricle. This procedure is performed using a tomographic gamma camera with one or more detectors. The patient lies on his back while the gamma camera rotates around him. To assess the systolic function of the left ventricle, the SPECT recording is synchronised with the patient's ECG.(2)

Indications for myocardial perfusion scintigraphy

Myocardial perfusion scintigraphy is indicated in: patients with atypical pain in the precordium; asymptomatic patients with risk factors; asymptomatic patients with positive ergometric findings; patients with symptoms but with negative ergometric findings; and patients with existing coronary disease. Before revascularisation, it is essential to: evaluate the extent of ischaemia; distinguish any consequent scarring; evaluate the functional significance of stenosis determined by coronary angiography; and monitor patients after coronary artery bypass or stent implantation. It is indicated in the prognosis of coronary artery disease and assessment of viable myocardium.

Cardiac stress tests

Stress during myocardial perfusion scintigraphy may be dynamic-ergometric or pharmacological stress.(3)

Exercise testing is a physiological test and most accurately marks the state of the increased metabolic needs of the heart. It can constitute running on a treadmill, or riding a specially provided bike. For patients who cannot adequately perform ergometric stress, pharmacological stress is applied.(4,5) (Table 1.)

Table 1. Contraindications for adequate ergometric stress

Conditions that prevent the proper execution of the acute exercise stress of myocard
Peripheral vascular disease
Arthritis
cerebrovascular accident
Chronic obstructive pulmonary disease
Poor motivation for dynamic test
Application of β -blockers, long-acting nitrates, calcium antagonists (alleviate ischemia induced by load)
Left bundle branch block
The early post-infarction period

Contraindications for ergometric stress are: uncontrolled unstable angina pectoris; **heart failure**; life-threatening or untreated arrhythmias; uncontrolled hypertension (BP >200/115 mmHg); acute myocardial infarction; myocarditis; pericarditis; acute respiratory infections; severe pulmonary hypertension; pulmonary embolism; and severe aortic stenosis.

Pharmacological stress test

Pharmacological stress testing involves the use of vasodilators, such as: dipyridamole; adenosine; regadenoson; or the inotropic agent, dobutamine. Dipyridamole and adenosine cause vasodilatation, which occurs in healthy coronary arteries, at an increased flow of 3-5 times compared with that at rest. In a stenosed coronary artery, vasodilatation does not occur after stenosis.

Dobutamine is a β -adrenergic agonist with both positive chronotropic and inotropic effects, resulting in coronary artery dilatation secondary to an increase in myocardial oxygen demand. It is used in patients for whom the use of vasodilators is contraindicated.(6)

Regadenoson is a selective coronary vasodilator (selective A_{2A} adenosine receptor agonist) and is used as a pharmacological stress agent in perfusion scintigraphy. Activation of the A_{2A} adenosine receptor causes coronary vasodilatation, and a sudden but short-lived increase in coronary blood flow. It is used only for diagnostic purposes. The dose of regadenoson is fixed at 400g (5ml), and administered in a peripheral vein. There is no need to adjust the dose according to body weight, and it is not dependent on renal and hepatic function. Regadenoson is applied in the form of a rapid intravenous injection, making the procedure quicker and easier.(7) Before using regadenoson, patients should avoid the consumption of products containing methylxanthines (e.g. caffeine), as well as any medicine containing theophylline for at least 12 hours before the procedure. If it is possible, dipyridamole should not be taken for at least two days prior to regadenoson administration. Regadenoson causes a sudden increase in heart rate after injection, so patients should remain sitting or lying down and should be monitored until ECG parameters, heart rate and blood pressure readings return to their pre-dosing values. Cardiac frequency returns to baseline within 10 minutes.

Contraindications for use of regadenoson are:

- Hypersensitivity to the active ingredient, or any other components
- Atrioventricular (AV) block, second or third degree, or dysfunctional sinoatrial node, unless the patient does not have a functioning pacemaker (pacemaker)
- Unstable angina that has not been stabilised by drugs
- Severe hypotension
- Heart failure

Side effects

The most commonly reported side effects during the clinical development of regadenoson were: dyspnoea (29%), headache (27%), redness and heating of the skin (23%), chest pain (19%), changes in the ST segment in the electrocardiogram (18%), discomfort in the gastrointestinal tract (15%), and dizziness (11%). Aminophylline may be used to relieve severe and/or persistent side effects of regadenoson.

Results of the Brinkert, Reyes et al. study demonstrate that regadenoson is well-tolerated, and can be considered safe, with no deaths or severe cardiac complications. Its side effects are slight, and similar to those of adenosine (chest discomfort and dyspnoea), but less expressed, although regadenoson use has more frequent gastrointestinal symptoms.(8) Regadenoson is safe for use in patients with chronic obstructive pulmonary disease, and asthma.(9)

Jager PL et al. performed their research to compare the severity and duration of side effects of regadenoson and adenosine as a medicament for pharmacological cardiac stress. They have shown that regadenoson use has significantly fewer side effects, with shorter durations.(10)

Conclusion

Myocardial perfusion scintigraphy as a non-invasive diagnostic procedure has a significant place in the algorithm of diagnostic tests in ischaemic heart disease.

Using pharmacological stress agents, this procedure can be performed on patients with a contraindication for ergometry.

With the development of new pharmacologic agents such as regadenoson, these diagnostic procedures have become more efficient, safer, and have fewer side effects.

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