The preoperative assessment of patients with valvular heart disease as a comorbidity

Dejan Marković, Radmilo Janković, Nataša Kovačević-Kostić, Miloš Velinović, Mile Vraneš, Branislava Ivanović

1Clinic for Cardiac surgery, Clinical Center of Serbia, Belgrade
2Faculty of Medicine, Niš University, Serbia
3Faculty of Medicine, Belgrade University, Serbia
4Clinic for Cardiology, Clinical Center of Serbia, Belgrade

INTRODUCTION

With patients that are suffering from valvular heart disease that are being prepared for any type of surgery, preoperative evaluation and preparation are of key importance to the successful outcome of the surgical treatment. Preoperative evaluation includes the anamnesis, medical history review, mandatory physical examination, and additional, primarily cardiological evaluation. If murmur heard during heart auscultation it is an indication of possible valvular heart disease. In such cases it is necessary to perform echocardiographic examination, which is noninvasive and relatively inexpensive procedure. Echocardiographic examination helps us differentiate pathological from physiological murmurs. It is extremely useful in determining the severity of valvular heart disease as well as predicting possible consequences of surgical interventions. In the presence of moderate, and particularly severe valvular disease additional cardiological examination is necessary, after which anesthesiologist and surgeon can determine if cardiological risk is acceptable for the given surgery.

Surgical risk in patients with valvular disease for non cardiac surgery is proportional to the severity of valvular disease and extent of the planned surgery. Risk is higher with major surgery, and especially ones that are followed with loss of blood and possible hypotension, tachycardia and other significant hemodynamic disturbances. In that sense it is important to provide hemodynamic monitoring, which is especially useful in determining whether the hypotension occurred due hypovolemia or heart failure. For minor surgical procedures for hemodynamics monitoring are sufficient to use electrocardiography and noninvasive measuring of systemic blood pressure. For major surgical procedures, it should be use invasive measurement of systemic blood pressure and measuring of central venous pressure. Placement of Swan-Ganz catheter for measuring pressure in the pulmonary circulation can be extremely useful in order to achieve adequate volume replacement and should be considered in patients with pulmonary hypertension and in patients with reduced left ventricular function. Transeosophageal echocardiography may be helpful in these patients too.
Aortic stenosis (AS) is the most common valvular disease in Europe and North America. In most cases it is calcific AS in adults of advanced age (2-7% of the population over 65 years). The second most common occurrence typically found in younger age groups, is congenital, whereas the third, rheumatic AS has become rare. Aortic stenosis disease progresses slowly.

Normal aortic valve has three leaflets, and the normal surface of aortic area is 2.5 to 3.5 cm². In relation to the surface of the aortic area, aortic stenosis can be mild (1.0 to 1.5 cm²), moderate (0.8 to 1.0 cm²) and severe (<0.8 cm²). The degree of aortic stenosis can also be determined based on the mean transvalvular pressure gradient, as mild (PG <20 mmHg), moderate (PG 20-50 mmHg) and severe aortic stenosis (PG >50 mmHg). Due to narrowing of the aortic area compensatory concentric left ventricular hypertrophy occurs. This further leads to increased left ventricular myocardial oxygen demand. Oxygen supply is reduced due to compression of subendocardial blood vessels by increasing pressure in the left ventricle. Therefore, angina pectoris symptoms may develop (although there are no changes in coronary blood vessels). In addition to anginal symptoms there can also be symptoms that suggest left ventricle heart failure. Aortic stenosis is also characterized by the appearance of syncope due to low cardiac output or arrhythmias in the field of left ventricular hypertrophy. The characteristic systolic murmur draws the attention and guides the further diagnostic work. Echocardiography has become the key diagnostic tool.

According to recommendations of the European Society of Cardiology, patients with asymptomatic aortic stenosis (AS) scheduled for small or medium risk elective non cardiac surgery can be safely operated, while asymptomatic patients with AS scheduled for high risk elective non cardiac surgery should be first planned for aortic valve replacement before non cardiac surgery. Patients with symptomatic AS should be first referred to aortic valve surgery even if the low risk non cardiac surgery is planned. If in the patients with AS, the aortic valve surgery is indicated first, and estimated risk of cardiac surgery is unacceptably high, then need for non cardiac surgery should be evaluated once again. In these asymptomatic patients non cardiac surgery should be performed only if really needed, and in symptomatic patients only if absolutely necessary with careful hemodynamic monitoring. Patients with severe AS who require urgent non cardiac surgery can be operated with careful hemodynamic monitoring.

Anesthesia approach to these patients involves prevention of hypotension and any hemodynamic changes leading to reduced cardiac output. Maintenance of sinus rhythm is essential because the loss of atrial contraction can dramatically reduce systemic blood pressure and cardiac output. Slower heart rate is preferred to allow time for left ventricle to get filled with the appropriate amount of blood, myocardium to overcome resistance through the narrowed aortic valve and in the aorta ejection sufficient stroke volume, and to maintain adequate coronary perfusion. Tachycardia should be promptly corrected intravenously by administering beta blockers. Hypotension should be treated with phenylephrine (without increasing heart rate) or ephedrine (with an increase in heart rate), and bradycardia should be treated with atropine or gikopirolat. General anesthesia is better choice than regional anesthesia techniques (spinal and epidural anesthesia sympathetic blockade may lead to significant hypotension) for these patients. For induction of anesthesia, it is recommended to use hipnomidate or cautiously propofol, while ketamine should be avoided because of possible tachycardia. For maintenance of anesthesia propofol, benzodiazepines and opioid analgesics are recommended, while inhaled anesthetics are used with great caution due to reduced peripheral vascular resistance. Pancuronium should be avoided because of possible tachycardia. Aortic stenosis is often accompanied by reduced levels of Von Wilebrand coagulation factor and platelet dysfunction that can lead to significant perioperative bleeding in these patients (level of coagulation disordered correlated with the degree of aortic stenosis). Perioperative fluid replacement should be based on the volume loss estimated by measuring the values of central venous or pulmonary artery pressure. And in the postoperative period, maintenance of stable hemodynamics is the main priority. It is necessary to avoid tachycardia, extreme bradycardia, hypertension and hypotension. Adequate fluid replacement, regular analgesic therapy, correction of anemia and hemodynamic monitoring are preconditions for a good postoperative treatment of patients with aortic stenosis.

In the preoperative preparation of patients with aortic valve stenosis, as well as intra-and postoperatively, it is necessary to achieve these hemodynamic goals:
- Low normal heart rate 60-70/min
- Maintenance of sinus rhythm.
- Adequate preload.
- High normal systemic vascular resistance.

Aortic regurgitation (AR) occurs when the blood returns during diastole phase through the incompetent aortic valve into the left ventricle. It can be congenital or acquired (due to valvular damage or aortic disease), chronic or acute. Acute aortic insufficiency is the result of aortic dissection, trauma, or endocarditis, and this is emergency state that is manifested by heart failure and pulmonary edema. Causes of chronic aortic insufficiency are numerous: idiopathic aortic dilatation, rheumatic disease, systemic hypertension, myxomatous degeneration, Marfan syndrome, congenital bicuspid aortic valve, sifilitic aortitis. Chronic aortic insufficiency is a slowly progressing disease that be present for many years without symptoms. Left ventricle volume overload leads to eccentric hypertrophy of the left ventricular wall. With the progression of the disease, left ventricle dilatation can occur. AR is diagnosed by the presence of a diastolic murmur. Characteristic of this disorder is that systolic and diastolic blood pressure have divergent values (diastolic pressure is significantly lower than normal). With echocardiography, us-
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Mitral stenosis usually occurs as a consequence of rheumatic fever. Leflets the mitral valve can be so rigidly modified to ineffectively close the mitral orifice and in addition to the initial stenosis, in about 40% of patients there is also a coexisting mitral regurgitation. Mitral stenosis is more common in women than in men, and this relationship is 2:1.

In developed countries the disease progress is slow and it remains for many years without symptoms (20-40 years from the onset of rheumatic fever). The normal mitral valve area is 4-5 cm². Patients are usually asymptomatic until this area is reduced to less than 2.5 cm².

Degree of mitral stenosis is determined based on the surface of mitral valve opening, the pressure gradient on the mitral valve or on the value of pulmonary artery pressure. Mitral stenosis can be mild (area >1.5 cm², mean gradient <5 mmHg or systolic pulmonary arterial pressure <30 mmHg), moderate (area 1.0 to 1.5 cm², mean gradient 50-100 mmHg, or systolic pulmonary pressure 30-50 mmHg), or severe (area <1.0 cm², mean gradient >10 mm Hg or systolic pulmonary ateuriski pressure >50 mmHg).

As the mitral orifice narrows the pressure gradient between the left atrium and the left ventricle must increase to maintain adequate flow across the stenotic MV causing left atrium dilatation, while the left ventricle is small, poorly filled with the normal function. Increasing the pressure in the left atrium leads to increased pressure in the pulmonary veins, which may lead to transudation and appearance of pulmonary edema. The initial increase of the left atrium maintains a low pulmonary pressure. With progression of disease, pulmonary arterial pressure increases, which can lead to pulmonary hypertension. Right ventricular hypertrophy occurs as a response to pulmonary hypertension and high pressure in the left atrium. With time tricuspid valve regurgitation can occur. Thromboembolic complications are common in patients with mitral stenosis, especially in those with atrial fibrillation and enlarged left atrium. Spontaneous echo contrast or thrombus in the left atrium, can also be seen.

According to the recommendations of the European Society of Cardiology, in patients with no significant mitral stenosis (mitral valve area >1.5 cm²) non-cardiac surgery can be performed with low risk, while in asymptomatic patients with severe stenosis and pressure less than 50 mmHg, the surgery can be performed with low risk, but the risk is significantly increased if atrial fibrillation exists. Symptomatic patients and patients with pulmonary arterial pressure greater than 50 mmHg, where non-cardiac surgery is planned, should be sent to the mitral valve surgery before non cardiac surgery (it is necessary if planned high risk surgery). In the clinical picture of patients with mitral stenosis most common are palpitations, fatigue, dyspnea, hemoptyis, recurrent bronchitis. Physical examination may reveal facies mitralis, peripheral cyanosis and signs of left ventricular failure, accentuated first tone, diastolic drip, and in severe cases the signs of right heart failure (hepatomegaly, ascites, peripheral edema). Electrocardiography often registers atrial fibrillation, and pathological p-wave (p-mitrale) if the patient is in sinus rhythm. Chest X-ray will show mitral valve calcification, left atrium enlargement, while echocardiography provides precise information on the degree of narrowing of the mitral valve. Filling the left ventricle is optimized at a slower heart rate. Faster heart rate, especially atrial fibrillation, reduces diastolic filling time and significantly reduced cardiac output. General anesthesia is preferred over the regional anesthesia techniques (spinal and epidural anesthesia), and intraoperative treatment of these patients is similar to patients with aortic stenosis: maintenance of sinus rhythm, adequate volume replacement, slower heart rate 50-70/min, systemic vascular resistance closer to the higher normal limits, avoiding hypercarbia, hypoxia and acidosis and other factors which
Mitral regurgitation (MR) is one of the most common valvular disease, since at least mild MR can be seen in 1 out of 5 adults.\(^9,10,11\) Mitral regurgitation results from leaflet (myxomatous degeneration, rheumatic fever, endocarditis), chordal (chordae rupture, endocarditis) or papillary muscle abnormalities (ischemic papillary muscle), or is a secondary result from left ventricular dysfunction. If the changes are on the mitral valve leaflets this is organic MR, but if the leaflets are normal and changes are on the other parts of the mitral valve apparatus, it is a functional MR. MR can be acute or chronic. Through the incompetent mitral valve a certain amount of blood in each systole returns back to the left atrium. When MR is acute there is no sufficient time for compensatory increase in the left atrium size so that leads to the left atrium overload. Increased pressure in the left atrium leads to increased pressure in the pulmonary veins, which cause transudation and pulmonary edema. Acute MR is an urgent condition and must be corrected prior to any other surgical procedure. Chronic mitral regurgitation develops gradually, leading to structural changes of the left ventricular (volume caused left ventricular eccentric hypertrophy) and enlargement of the left atrium. These patients have long periods without symptoms, and when the amount of regurgitated blood volume exceeds 60% of stroke heart volume, the symptoms occur (dyspnea, fatigue, weakness). Pulmonary hypertension can occur as a result of a long-lasting significant mitral regurgitation. The degree of mitral regurgitation is determined by the size of regurgitation fraction (a part of left ventricular stroke volume which backs through mitral valve to left atrium during systole). Mitral regurgitation can be mild (regurgitation fraction 20-30%), moderate (RF 30-50%) and severe (RF >55%). The degree of MR can be determined based on the surface area of jet regurgitation: mild (area <3cm\(^2\)), moderate (area 3.0 to 6.0 cm\(^2\)) and severe (area >6cm\(^2\)). Clinical examination of patients reveals a systolic murmur at the top of the heart that spreads in axilla. On ECG often reveals atrial fibrillation, left ventricular hypertrophy and two-phase wave - p mitral if the patient is in sinus rhythm. Chest X-rays can show enlarged heart, while echocardiography can obtain a precise information about degree of mitral regurgitation and left ventricular function. Echocardiographically determined ejection fraction (EF) in patients with MR is not a good indicator of heart function because it is usually overestimated. If measured value is EF <50% then the function of the left ventricle is significantly decreased in these patients. Asymptomatic patients with mild to moderate MR can handle non cardiac surgery well. Patients with severe MR, especially symptomatic patients must be further evaluated. Assessment of cardiac risk depends not only on the degree of mitral regurgitation, but also to the etiology of its origin (coronary heart disease, myocardial infarction and ischemic cardiomyopathy significantly increase the risk of perioperative adverse events). In patients with organic MR, mitral valve surgery is rarely required before planned non cardiac surgery, while patients with functional MR (resulting from ischemic heart disease) coronary artery bypass graft surgery and mitral valve surgery must be considered before non cardiac surgery.\(^14\) Patients with MR can benefit from reduction of systemic vascular resistance (decreased regurgitaciona fraction) and faster heart rate (shortening the duration of diastole and reduces the amount of regurgitation volume). Hypotension in the perioperative period should be treated with adequate amounts of fluids and beta agonists (ephedrine, dobutamine, epinephrine), while alpha agonists (phenylephrine, norepinephrine) should be avoided. Bradyhycardia should be avoided, and should be treated using atropine or glikopirrolat.

Systemic vascular resistance should be kept at the lower limit of normal values using vasodilatators (urapidil, sodi um nitroprusside). In the postoperative period patients require adequate analgesia to reduce sympathic activity which may cause increased systemic vascular resistance and exacerbate mitral regurgitation. Postoperatively, careful monitoring of hemodynamics is necessary because of risk of overloading fluids and development of pulmonary edema.

In the preoperative preparation of patients with mitral regurgitation, as well as intra-and postoperatively, it is necessary to achieve these hemodynamic goals:
- High normal heart rate 80-90/minute
- Adequate preload
- Low systemic vascular resistance
- Low pulmonary vascular resistance

MITRAL VALVE PROLAPSE

Mitral valve prolapse (MVP), with or without mitral regurgitation, occurs in 1-3% of the population, and more common in younger women.\(^12,13\) MVP is defined as thickening of the mitral valve leaflets (>5 mm) with prolapse (>2-4 mm) above the mitral valve annulus in the left atrium during systole. Cause thickening of mitral valve leaflets is myxomatous degeneration caused by dysregulation of synthesis mitral matrix protein and degradation of collagen. Mitral valve prolapse may be asymptomatic and symptomatic when in clinical picture dominated palpitations, chest pain, fatigue, dyspnea, and orthostatic hypoten-
sion. It is usually a benign condition, but may be complicated by infective endocarditis, cerebral embolization, arrhythmias - atrial and ventricular, mitral regurgitation and sudden cardiac death (more common in older men).

Diagnosis is made by echocardiography. Although, similar pathophysiology as well as mitral regurgitation, there is a significant difference between these two entities that affect different anesthesia management. In the mitral valve prolapse, mitral regurgitation is smaller if left ventricle volume is larger, and vice versa, if the volume of the left ventricle decreases regurgitation is greater. Therefore, reduction in peripheral vascular resistance and a faster heart rate, preferred in mitral insufficiency, are unfavorable in patients with mitral valve prolapse because they reduce the left ventricle volume and increase regurgitation. Adequate fluid resuscitation and avoidance of hypovolemia increase the volume of the left ventricle, while maintenance of systemic vascular resistance at the upper limit of normal values using vasoconstrictors reduces the left ventricle emptying with consequent reduction of regurgitation in patients with mitral valve prolapse. Patients with mitral valve prolapse usually have a preserved left ventricular function and regional anesthesia techniques can equally be used as general anesthesia. Reduction in peripheral vascular resistance during spinal and epidural anesthesia should be anticipated with adequate volume administration. Hypotension should be treated with adequate amounts of fluids, and if needed alpha agonists (eg phentolamine). Ventricular arrhythmias are common in these patients, especially when surgery is performed in sitting or reverse Trendelenburgh position. For the treatment of these arrhythmias, lidocaine or beta antagonists can be used.

In the preoperative preparation of patients with mitral valve prolapse, as well as intra-and postoperatively, it is necessary to achieve these hemodynamic goals:

- a) Low normal heart rate 60-70/min
- b) Adequate preload
- c) High normal systemic vascular resistance

BACTERIAL ENDOCARDITIS PROFILAXYS

Bacterial endocarditis is a rare but difficult disease. It occurs after bacteria entering into bloodstream either after everyday activities (brushing teeth, shaving ...) or after some of the invasive procedures (dental or other interventions). Recommendations of the European Society of Cardiology defined patients and procedures that requires bacterial endocarditis prophylaxis. 19

Patients who are at increased risk of bacterial endocarditis:

- Patients with prosthetic cardiac valve or prosthetic material used for cardiac valve repair.
- Patients with previous infective endocarditis
- Patients with congenital heart disease:
  a. Unrepaired cyanotic CHD, including palliative shunts and conduits
  b. Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure.
  c. Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device.

Medical procedures with increased risk for bacterial endocarditis:

- All dental procedure that involves manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa.
- Cystoscopy or other urogenital procedure where the urinary tract is infected with Enterococcus species.
- Drainage of abscess, empyema, phlegmona when probable or proven pathogens are Staphylococcus aureus, Streptococcus or Enterococcus.

For many other invasive intervention antibiotic prophylaxis of bacterial endocarditis is not recommended, but it is understood that they are performed in strict aseptic conditions (eg venous cannulation).

In the bacterial endocarditis prophylaxis drugs of choice are penicillins and cephalosporins according to the following scheme:

- Single dose of amoxicillin or ampicillin 2 g orally or intravenously for adults and 50mg/kg orally or intravenously for children, 30 - 60 minutes before the procedure (an alternative is intravenous cephalaxin 2gr for adults or 50mg/kg for children, or intravenous cefazolin or ceftriaxone 1 g for adults and 50 mg / kg for children).
- For patients allergic to penicillins and cephalosporins it is recommended a single dose of Clindamycin 600 mg orally or intravenously for adults and 20mg/kg orally or intravenously for children, 30 - 60 minutes before the procedure.

When performing surgery in patients with prosthetic valve antibiotic prophylaxis of wound infection (eg cephalosporins) also represents the prevention of bacterial endocarditis. In patients allergic to penicillins and cephalosporins alternative medications are vankomycin or clindamycin.

PATIENTS WITH ARTIFICIAL VALVE AND SURGERY

There are two basic types of artificial heart valve: biological and mechanical valves. Patients with artificial valve without valvulal or ventricular dysfunction are not under increased risk of non cardiac surgery. After valve replacement, and before the planned intervention it should be prevent bacterial endocarditis as the previous described.

When a patient with an artificial aortic valve and no risk factors for thromboembolic complications (atrial fibrillation, procoagulant state, previous thrombosis, EF less than 30%, more than one mechanical valve), is scheduled for elective surgery it is necessary to stopped oral anticoagulant therapy (OAT) 48 - 72 hours before the planned surgery (INR values should be less than 1.5). Anticoagulant therapy should continue within 24 hours after surgery. If the patient with artificial mitral valve, or with an prosthetic aortic valve and risk factors for thromboembolic complications, is preparing for elective surgery, oral antico-
agulant therapy should be stopped 48-72 hours before surgery with the introduction of therapeutic doses of low molecular weight heparin (when INR values fall below 2.0). In these patients low-molecular-weight heparin should be given at least 4-6 hours before the planned surgery and should be continued as soon as possible after surgery until effective anticoagulation is achieved with oral therapy. When patients have therapeutic or supratherapeutic INR and required emergency surgery, it should be immediately INR corrected to reference range (INR<1.5). In this situation it is recommended an infusion of fresh frozen plasma, rather than intravenous infusion vitamin K. Eventually only low-dose of vitamin K (1mg) can be given intravenously. After fresh frozen plasma infusion and/or vitamin K administration it is necessary checked INR and introduced low molecular weight heparin if indicated.

CONCLUSION

Valvular heart disease, particularly if complicated by heart failure and atrial fibrillation significantly increased the risk of perioperative adverse events. Type of valvular disease and preoperative evaluation of the degree of valvular disease affect the decision for surgery and the choice of anesthetic technique, while adequate perioperative hemodynamics monitoring and early intervention can prevent adverse events.

SUMMARY

PREOPERATIVNA PRIPREMA BOLESNIKA SA VALVULARNIM SRČANIM OBOLJENJIMA KAO KOMORBIDITETOM

Kod bolesnika sa oboljenjima srčanih zalistaka koji se pripremaju za operativno lečenje u bilo kojoj grani hirurgije preoperativna evaluacija i priprema mogu biti od ključnog značaja za uspešan ishod hirurškog lečenja. Preoperativna priprema i intraoperativni tretman bolesnika sa oboljenjima srčanih zalistaka se razlikuju u odnosu na vrstu valvularnog oboljenja: aortna stenoza, aortna insuficijencija, mitralna stenoza i insuficijencija, prolaps mitralne valvule. U radu su dati kriterijumi za procenu težine vaskularne bolesti, budući da je rizik od operativnog lečenja u srazmeru sa težinom valvularnog oboljenja. Imajući u vidu da je operativni rizik takodje u proporciji sa vrstom i obimom nekardijalne operacije, prikazane su i preporuke za intraoperativni monitoring radi procene hemodinamskog stanja bolesnika, kao i preporuke za perioperativno lečenje hipotenzije, tahikardijske i drugih hemodinamskih poremećaja.

U radu ce biti posebno razmotrena i profilaksis bakterijskog endokarditisa do koga može doći posle hirurškog lečenja bolesnika sa valvularnim manama. S obzirom da su bolesnici sa valvularnim bolestima, a posebno oni sa ugradjenom veštačkom valvulom i sa poremećajima srčanog ritma obično na oralnoj antikoagulantnoj terapiji, date su i preporuke za postupanje sa bolesnicima sa oralnoj antikoagulantnoj terapiji u sklopu preoperativne pripreme.

REFERENCES


