Preoperative preparation of vascular patients undergoing nonvascular surgery

Nataša Kovačević-Kostić, Radmila Karan, Mile Vraneš, Dejan Marković, Miloš Velinović, Zivan Maksimović
1 Cardiac Surgery Clinic, Clinical Center of Serbia, Belgrade, Serbia
2 Medical School, Belgrade University, Belgrade, Serbia
3 Vascular and Endovascular Surgery Clinic, Clinical Center of Serbia, Belgrade, Serbia

Patients with vascular diseases mainly caused by atherosclerosis, that are undergoing nonvascular surgery, often have co-existing conditions which affect their cardiovascular system. Cardiovascular complications are among the most common perioperative complications including respiratory complications and infections. These include coronary disease, hypertension, heart insufficiency, pulmonary hypertension, and renovascular hypertension, among others.

Preoperative preparation must include the use of ß-blocker therapy, antihypertensive, antithrombotic and antilipogenic therapy. Electrocardiogram (ECG) and trans-thoracic echocardiography are the minimum preoperative diagnostic evaluations that should be performed, because complications may arise even in patients without prior cardiovascular symptomatology.

Venous diseases are the most common contemporary diseases affecting people of all age groups and races. Invasive-diagnostic-therapeutic procedures may cause lesions of venous endothelium, hence perioperative prevention of deep vein thrombosis (DVT) with the use of heparin or low-molecular-weight heparin (LMWH) should be undertaken.

Key words: vascular diseases, nonvascular surgery, preoperative preparation, perioperative complications, venous system diseases.

INTRODUCTION

Pre-operative evaluation of patients and adequate preparation for any surgical procedure decreases the risk for perioperative complications. Comorbidities, on the other hand, increase the risk of perioperative morbidity and mortality.

Patients with some form of vascular disease who are undergoing any non-vascular surgical procedure have a high risk for developing peri-operative and post-operative complications. Most common nonsurgical complications that arise in surgical patients are infections and complications related to respiratory and cardiovascular systems.

Those cardiovascular complications can be divided into following groups:
1. Coronary disease. Patients with coronary disease have a high risk for developing myocardial infarction regardless of the type of surgery (vascular or non-vascular).
2. Hypertension alone represents a high risk for anesthesia and for surgery. Renovascular hypertension is considered to be even a greater risk, if it can not be treated before surgery.
3. Systemic vascular diseases. Stenosis or occlusion of visceral arteries present in these diseases leads to end-organ dysfunction and consequently to possible peri-operative and post-operative complications.
4. Peripheral vascular diseases, as comorbidities, carry high surgical and anesthesiological risk that requires specific evaluation, preparation and treatment.

Vascular diseases affect arterial, venous and lymphatic systems. They are the leading cause of morbidity worldwide. They can be acute and/or chronic in onset.

ATHEROSCLEROSIS

Pre-operative preparation and management of anesthesia in patients depends upon the type of vascular disease which is, in most cases, caused by atherosclerotic processes in arterial blood vessels.

There is a saying: "One’s age depends on the stage of atherosclerosis of one’s vascular system".

Atherosclerosis (atherosclerosis obliterans) is a systemic vascular disease, and even though it has been systematically studied both on a basic and a clinical level, its etiology has not been fully understood. There are many hypotheses...
proposed trying to explain the pathogenesis of atherosclerosis: mechanical, immunological, endocrinological, or mononuclear, and the lipid hypothesis².

Atherosclerosis starts in childhood. Important roles in the development of atherosclerosis are played by inflammation, lipid infiltration of subendothelium and smooth muscle cell proliferation. Endothelial dysfunction develops as an early consequence of atherosclerosis³.

Atherosclerotic changes in blood vessels lead to turbulent blood flow, preceding the dysfunction of endothelium and inhibiting the production of nitric oxide, a molecule with vasodilatory and anti-inflammatory effects. Turbulent blood flow also stimulates endothelial cells to produce adhesion molecules which engage and bind inflammatory cells.

Risk factors that lead to atherosclerosis include hypertension, smoking, hypercholesterolaemia, diabetes mellitus, genetic predisposition, obesity, stress, and physical inactivity¹,¹².

Different factors stimulate smooth muscle cell replication and formation of extracellular matrix. The end result of these changes is the formation of complex subendothelial atherosclerotic fibrous plaque with fibrous cap. Atherosclerotic plaques may be stable or unstable³. Stable plaques grow slowly and they may even regress. It takes decades for them to form clinically significant stenosis or occlusion.

Unstable plaques are susceptible to spontaneous rupture, intraplaque hemorrhage, erosion, and fissure. Accordingly, they may cause acute thrombosis long before they cause clinically and angiographically apparent stenosis.

In the case of plaque rupture, the fact that its contents are now exposed to circulating blood triggers thrombosis.¹³

The resultant thrombus may:
- Organize and incorporate into the plaque, causing its rapid growth. The plaque fills with blood and swells, occluding the artery.
- Occlude the blood vessel, causing an acute ischemic event.
- Embolize in the blood vessel.

Plaque stability depends on its composition and configuration relative to blood flow. Intraplaque hemorrhage may have an important role in destabilizing the plaque.¹³

Unstable coronary plaques are rich in macrophages. They have a thick body rich in lipids and a thin fibrous cap. They narrow the coronary artery lumen by less than 50% and may rupture spontaneously. Plaques that are considered low-risk have a thicker cap and less lipid content. They produce stenosis greater than 50%, and may be responsible for predictable exertional stable angina.

Clinical symptoms and signs of coronary artery plaque rupture depend on the anatomy of the plaque, the balance of procoagulant and anticoagulant factors in blood, and myocardial vulnerability to arrhythmias.¹⁴ Coronary endothelial cells modulate myocardial blood flow by producing vasoconstrictors, vasodilators, and substances that produce anticoagulatory, fibrinolytic and antithrombotic effects.¹⁴

Unstable carotid artery plaques are of similar composition as coronary plaques, but are unlikely to rupture; rather, they tend to cause severe stenosis, occlusion or formation of platelet-rich thrombus which will likely embolise.¹⁴

While taking a patient’s history, we can presume potential atherosclerosis, and possible anesthesia-related and surgery-related complications. Diagnostic procedures for angina pectoris, myocardial infarction and evaluation of cardiac function should include electrocardiography, echocardiography, and stress-echocardiography. A coronary angiogram should be performed in patients with unstable angina. Before any non-vascular surgical procedure is undertaken, endovascular or surgical myocardial revascularization should be performed, if possible. Beta blockers should be taken even on the day of surgery.

**DISEASES OF THE AORTA**

Atherosclerotic processes affect the aorta as well, primarily its intimal layer, and spread to the other two layers over time, or to preexisting aortic aneurysms. The disease can affect the thoracic or abdominal aorta, especially the infrarenal part (Mb Leriche), or the aorta can be affected diffusely including its visceral or peripheral branches¹. An aneurysm is a permanent dilation of the aortic diameter to more than 50% of the normal diameter. It is caused by many etiological factors: age, gender, genetic predisposition, and hemodynamic disturbances (such as hypertension). Aneurysms can be either true aneurysms, where the wall of the aneurysm contains all three layers of the vessel, or false, dissecting or pseudoaneurysms where the wall is comprised only of an outer layer of adventitia. Etiologically, aneurysms can be divided into congenital, degenerative, of infectious etiology, inflammatory, dissecting, post-stenotic, pseudoaneurysms, and aneurysms caused by connective tissue disorders or post-vascular graft aneurysms¹. Histopathological findings in the aneurysmatic wall can include atherosclerotic changes or cystic medial necrosis.

Aortic aneurysms are potentially lethal, and their pathogenesis is complex and multifactorial as is their rupture. Some of the factors that can lead to the rupture of the aneurysm are its size and type, persistent high blood pressure or its sudden increase⁴,⁶,⁷. Ruptured aneurysms are associated with high mortality rates. It is assumed that 2/3 of patients die before reaching a cardiovascular facility, and about half of surgically managed patients survive. This fact makes the treatment of such condition very stressful for a surgeon as well as for an anesthesiologist.⁸

In patients with an aneurysm who are undergoing nonvascular (usually urgent) surgery, the most important thing is perioperative blood pressure control. Variations in blood pressure cannot be allowed at any moment. Blood pressure increase is stressful to an already pathologically afflicted aneurysmatic arterial wall, possibly causing its rupture⁹.

**Hypertension** can be primary - idiopathic, or secondary - renal (in parenchymal renal disease or of renovascular origin), endocrinological (feochromocytoma, Cushing’s Syndrome, hyperaldosteronism, pregnancy-related tox-
caemia), mechanical (coarctation of the aorta) and neurogenic (in elevated intracranial pressure)\(^2\). Despite the origin of hypertension, its continuous pre-operative control as well as peri-operative and post-operative control, is mandatory.

Special consideration is necessary in a case of renovascular hypertension. This type of hypertension is usually caused by atherosclerotic processes affecting the walls of a renal artery. Over time this leads to stenotic changes or even occlusion of the renal artery. Atherosclerotic plaque may rupture, causing embolisation of the renal artery, also embolisation may be caused by an aortic atherosclerotic plaque rupture, or if a part of the aortic thrombus is dislodged. Etiology of renal artery disease also includes arteritis, fibromuscular dysplasia or other kidney or renal artery anomalies, and the changes that follow renal transplantation\(^2\). All these conditions lead to renal dysfunction, renal atrophy and hypertension. Renovascular hypertension may clinically present as malignant hypertension. Symptoms may be: chest pain—(angina pain), pulmonary edema, confusion, hematuria, sudden deterioration of renal function, hypertensive retinopathy, left ventricular hypertrophy, and cerebral infarct\(^9,10,11\).

If a patient with renovascular hypertension is undergoing planned, elective surgery, that procedure should be postponed until after a surgical procedure on the appropriate renal artery for the treatment of hypertension and consequent decrease in morbidity and mortality risk.

Management and control of blood pressure and renovascular hypertension is achieved by preoperative preparation of patients, and the use of antihypertensive medications which include ACE inhibitors, and \(\alpha\) and \(\beta\) blockers\(^12\). During surgery and anesthesia, hypertension is controlled by maintaining an adequate depth of anesthesia, maintaining stable intravascular volume (crystalloids, colloids), and by the use of intravenous antihypertensive medications. The use of \(\beta\)-blockers in the pre-operative period and on the day of surgery decreases complications in patients by 10\%\(^1\).

During surgical procedures, where changes or disorders of peripheral vascular resistance might be anticipated (liver transplantation), and/or where there are changes in circulatory volume (on-pump cardiac surgery)\(^3\) or a decrease in venous return to the heart (vena cava clamping)\(^5,13\) are expected, the use of vasoconstrictors is sometimes necessary. Having all these in mind, an anesthesiologist must be familiar with all phases of the surgery, as well as with physiological and pathophysiological changes in circulatory hemodynamics, in order to respond in timely fashion and prevent intraoperative complications, such as aortic rupture, that are concomitant with a high mortality rate\(^6,12\).

In patients with an aneurysm undergoing elective surgery where hemodynamic instability can be expected, continuous hemodynamic monitoring of intra-arterial blood pressure, II and V lead ECG, pulse oximetry, central venous pressure, duressis, and body temperature is necessary. The evaluation for the placement of a Swan-Ganz catheter in a pulmonary artery, in order to assess adequate therapy and to maintain stable hemodynamics, is sometimes needed\(^3,5\).

In patients with abdominal aorta aneurysm (AAA) exists the possibility of embolisation with a part of mural thrombus. This causes acute ischemia of the limb irrigated by the affected artery. The thrombus may embolise to one of the visceral arteries, but this does not cause clinically relevant target-organ ischemia, owing to the existence of a rich vascular collateral network. However, an exception is the mesentery artery\(^12\).

Rarely, large AAA may cause disseminated intravascular coagulation (DIC). The exact pathophysiological mechanism is unknown. It is assumed that large areas of the pathologically changed endothelial surface cause rapid thrombosis and consequent consumption of coagulation factors, which triggers the development of DIC\(^4\).

Aneurysms can also compress local structures (such as veins, nerves) leading to vein stasis and neurological problems (such as paresthesias and pain). Most aneurysms are asymptomatic, accidentally discovered during some other diagnostic procedure (chest X-ray, echocardiography, magnetic resonance imaging) or pre-operative evaluation of the patients\(^14\).

**Aortic dissection**

The main feature of aortic dissection is an intimal tear in an already pathologically changed aortic wall, through which blood surges into the medial layer. Certain diseases (Marfan’s syndrome, Ehlers-Danlos syndrome, bicuspid aortic disease, familial aortic dissection) predispose the aorta to dissection. Acquired aortic dissection is usually caused by atherosclerosis, trauma or cardiac surgical manipulation\(^1,14\). Dissection may originate anywhere in the aorta or its branches, and can include other arteries. It can originate in a part of the aorta alone, or diffusely in the aorta as a whole (mega aorta syndrome). If the sinotubular junction is afflicted with dilation of ascendent part of the aorta, this will lead to aortic valve insufficiency\(^1\).

Dissection may cause compression of the adjacent organs or erode into them. It can also cause thromboembolic processes, rupture or leak. An ascendent aortic aneurysm may rupture when its diameter is greater than 5.9 cm, while the diameter of other parts of the aorta is not a crucial factor for the rupture. Symptoms may vary: hoarseness owing to compression of the left recurrent laryngeal nerve; dysphagia owing to compression of the esophagus; dyspnea owing to compression of the lungs; edema, facial and upper body plethora owing to compression of the superior vena cava, or congestive heart failure and aortic valve insufficiency symptoms caused by dilation of the aortic valve annulus\(^1\).

Acute onset of the disease is usually provoked by an increase in blood pressure. The main symptom is excruciating pain localized in the precordium, neck and in the interscapular area. It is typically abrupt in onset and described as tearing or ripping. The patient is in shock - pale, diaphoretic, and cold because of vasoconstriction and pain. Systemic blood pressure is usually elevated, while major arterial pulses and arterial pressure may differ between
arms. Occlusion of a carotid artery leads to somnolence and other neurological complications. If the renal artery becomes occluded, an abrupt decrease or even a stop in urine output, and an increase in serum BUN and creatinine levels will occur. Peripheral arterial occlusion leads to target limb ischemia. Spinal cord ischemia causes paraparesis and paraplegia. Myocardial infarction is the consequence of a coronary artery occlusion, while bowel ischemia develops as the result of upper mesentery artery occlusion. If an aortic aneurysm dissection leaks into pericardium, cardiac tamponade develops as a consequence, with a high mortality rate.

Given all the considerations mentioned above, a good and thorough pre-operative evaluation and preparation, mainly of blood pressure control and hemodynamic stability, is vital for the prevention of possible consequences that follow non-vascular surgeries (especially for some life-threatening condition) of patients with an aortic aneurysm that could not have been previously treated endovascularly or surgically.

During the post-operative course, the most important factor is pain management using analgesics and local anesthetics. When using epidural catheters for continuous anesthesia, vascular volume redistribution. Hypertension management and control is achieved using continuous antihypertensive medication infusion and continuation of beta blockers.

PERIPHERAL ARTERY DISEASES

Atherosclerotic changes of the arteries of the lower extremities have a high morbidity, affecting about 2% of the worldwide population, and causes symptoms of intermittent claudication. Peripheral artery disease may have an acute onset, owing to embolism or chronic onset owing to atherosclerotic processes that in time will cause occlusion of distal aorta, femoral artery, or subclavian "steal" syndrome or coronary-subclavian "steal" syndrome. Atherosclerosis increases with age, so more than 70% of the population older than 75 years has atherosclerotic changes in their peripheral arteries.

Simultaneously, with peripheral arteries, changes affect coronary and cerebral arteries as well. Patients with occlusive peripheral disease and hypertension are at three times greater risk of developing renal artery stenosis. They are prone to cardiovascular disease, myocardial infarction, cerebrovascular disease, acutization of chronic renal insufficiency, especially if they are undergoing some surgical procedure. It is very important to take a good patient history and to perform a thorough physical examination, with focus on possible intermittent claudication, weak peripheral artery pulses, skin pallor, cyanosis, etc.

If the coronary disease is discovered in a hospital just before the planned surgical procedure, the morbidity is 10% higher. Thus, proven carotid or coronary stenosis if clinically important, should be treated before a planned elective surgical procedure. If that is impossible, owing to the emergency nature of the current surgical condition, then β - blockers and low molecular weight heparins should be used perioperatively.

Coronary-subclavian steal syndrome is present when a patient with a coronary left internal mammary artery bypass develops distal stenosis of a subclavian artery. The symptoms include angina pectoris, central nervous ischemia and a lower blood pressure in the ipsilateral hand. Peripheral vascular diseases also include systemic vasculitides: Takayasu’s arteritis, thromboangiitis obliterans (Morbus Bürger), Wegener’s granulomatosis, temporal arteritis and poliarteritis nodosa, Raynaud’s phenomenon and Kawasaki syndrome.

Takayasu’s arteritis is granulomatous inflammatory aorto-arteritis of an unknown origin that damages the aorta and its major branches, and also the pulmonary artery. In 80% of cases, it affects women of the Far East. It is an autoimmune disease with genetic predisposition. As a complication of Takayasu’s arteritis, neurological deficits may develop (from vertigo to cerebral ischemia and infarction), ischemic and valvular heart disease, uncontrolled hypertension, V/Q mismatch, retinopathy, renal artery stenosis with renovascular hypertension, rheumatoid arthritis, etc.

During preoperative evaluation of these patients, it must be kept in mind that this is a multiorgan disease. From the anesthesiologist’s point of view, the most important symptomatology of Takayasu’s arteritis is hypertension. It is treated with calcium channel blockers and ACE inhibitors, and if necessary, nitroprusside or nitroglycerin infusions. Intra-arterial cannulation must be performed in order to measure blood pressure, because non-invasive blood pressure management is impossible. These patients are frequently on corticosteroid and immunosuppressive treatment, which enhances perioperative risk. Corticosteroid treatment should be continued perioperatively and glucocorticoid supplementation adjusted in order to avoid adrenal insufficiency.

Thromboangiitis obliterans - Buerger’s disease is an inflammatory disease of small- and medium-sized arteries and veins of the lower and upper extremities. It is common among male smokers. Presentation includes gangrene of lower extremity digits and unremitted pain. Because of the effects of chronic smoking, these patients may have chronic obstructive pulmonary disease or emphysema, which represent additional problems for anesthesia. Owing to susceptibility to infections, prophylactic use of antibiotics is mandatory.

Wegener’s granulomatosis is characterized by necrotizing granulomatous inflammatory change in a blood vessel wall in multisystem organs. Cerebral aneurysm, peripheral neuropathy, sinusitis, laryngeal stenosis, epiglottis destruction, pneumonia, V/Q mismatch, hemoptysis, bronchus destruction may develop, as well as myocardial ischemia, heart valve destruction with consequent insufficiency, hematuria, and azotemia. Multisystem affection and immunosuppressive therapy present a real anesthesiologic challenge. These patients have anemia, leukopenia, infection, and possible laryngoscopy-related injuries followed by bleeding from necrotic laryngeal tissue. Intra-ar-
terial cannulation may be difficult. The use of succinylcholine and regional anesthesia must be done with caution.

**Poliarteritis nodosa** is a vasculitis of medium- and small-sized arteries triggered by Hepatitis B antigens, and medicamentous reaction. It is more frequent in women. Inflammatory changes in arteries lead to myocardial ischemia, glomerulonephritis, and peripheral neuropathy. Corticosteroids and immunosuppressers are the mainstays of treatment. Preoperative preparation includes management of systemic hypertension and prevention of renal failure.

**Kawasaki syndrome** occurs in children and is manifested with mucocutaneous nodes. Symptoms include fever, conjunctivitis, inflammation of the mucosa, erythema of palms and soles, neck lymphadennopathy, etc. The most serious complications develop owing to the changes in coronary arteries that lead to ischemic heart disease, angina pectoris, and cerebral hemorrhage owing to medium-sized artery aneurysms.

**Thoracic Outlet Syndrome (TOS)** is an important co-morbidity because of the symptoms that develop as a result of compression of neurovascular structures above the first rib and behind the clavicle (subclavian artery and vein, brachial plexus trunks). It can develop as a result of a redundant cervical rib, an enlarged processus transversus in, brachial plexus trunks). It can develop as a result of compression of neurovascular structures above the artery aneurysms.

**Venous Disease**

Venous disease is the leading cause of morbidity worldwide. Venous blood flow depends on a heart and venous pump, and gravitation. It is more frequent in women. The main manifestations of venous disease are venous stasis and insufficiency. The disease can be classified either as acute (deep vein thrombosis (DVT), and thrombophlebitis of the superficial veins), or chronic (primarily varicose disease, postthrombotic syndrome, dermal phlebosclerosis and venous ulcer). It is multifactorial disease with proven genetic component. The most important etiological factor is the Virchow Triad that includes hemodynamic changes such as stasis or turbulence, endothelial injury/dysfunction, and hypercoagulability.

Venous stasis is caused by immobilization, obesity, general anesthesia, pregnancy, congestive heart disease, acute myocardial infarction, already existing venous varicosity, and age above 40 years.

Endothelial injury is caused by trauma, invasive diagnostic-therapeutic procedures (surgery, catheterisation, implantable pacemaker, infections, hemotherapy), hypotension and a positive history for thromboembolism.

Hypercoagulability may be a consequence of pregnancy, malignancy, hormonal replacement therapy, polycythemia vera, nephrotic syndrome, antiphospholipid syndrome, and other conditions (congenital or acquired thrombophilies, etc.)

A thrombotic process in the vessel starts at the valve cusps, from where it grows and can occlude the vein leading to stasis.

The most serious complication of DVT is pulmonary embolism (PE). Pulmonary embolism can develop in patients with symptomatic and asymptomatic venous thrombosis. Its clinical course is difficult to manage and often lethal. Pulmonary hypertension is one of the consequences that arise from PE.

Every surgical procedure is a risk factor for DVT. The incidence of DVT is 50-75% after pelvis injuries, hip replacement surgeries, or knee surgery. It can develop after spinal cord injuries, or after stroke with lower extremity paralysis.

Prevention of DVT and PE is an important measure for any surgical procedure, or any other cause of immobilization. It is divided into primary preventive measures that are undertaken before the development of DVT. Those measures include medical and physical therapy. Secondary prevention is used in patients, where primary prevention is contraindicated or unavailable. It consists of methods that help in early discovery of DVT and prevention of PE.

General prophylactic measures include adequate fluid balance, correction of hypovolemia and hemococoncentration, adequate positioning of immobilised patients, and early rehabilitation either active or passive.

DVT prophylaxis consists of reducing venous stasis and coagulability of the blood. Venous stasis reduction is done by early rehabilitation, lower limb elevation, Trendelenburg’s position, elastic bandaid, etc. Elastic prophylactic bandaid with stockings should be applied to all high-risk patients undergoing any surgery. Reducing coagulability of blood is done with medications: LMWH, heparin, ultralow molecular weight heparin, and oral anticoagulants.

**Low Molecular Weight Heparins (LMWHs)** are fragments of glycosaminoglycans that decrease the production of coagulation factor Xa in plasma activated by plasmin. Their antithrombotic effect is similar to the effect of heparin and is achieved through antithrombin III factor and heparin cofactor II. LMWH increases levels of tissue-type plasminogen activator (t-PA), enhances erythrocyte flexibility, decreases blood viscosity, and enhances blood flow through small-sized blood vessels. The inhibitory effect of Low Molecular Weight Heparin on factor Xa lasts for 12 hours, so its use once or twice a day subcutaneously is possible. Bleeding or coagulation disorders associated with the use of LMWH are very rare. Preoperative prophylaxis is achieved with the use of LMWH 1-12 hours.
prior to surgery, and postoperative prophylaxis with LMWH at 2-12 hours after surgery.22.

Heparin is a mucopolysaccharide with an anticoagulant and antithrombotic effect. It increases the inhibitory activity of antithrombin III 1000 times. The use of heparin can cause bleeding or Heparin-Induced Thrombocytopenia (HIT). Contraindications include subarachnoid hemorrhage, epilepsy, neurosurgical procedure, and hemorrhagic diathesis.

Orgaran is a low molecular weight heparinoid that consists of mixture of low molecular weight nonheparin glycosaminoglycans. It inhibits thrombosis formation without changing thrombocytic function. Orgaran is the first line of treatment in patients with HIT and acute renal failure.

Oral anticoagulants are usually used for long-term secondary prevention of thromboembolic complications. They are coumarin and indandione derivatives. They act by interfering vitamin K metabolism in the liver and by inhibiting vitamin K-dependent factors of coagulation such as prothrombin and Factors VII, IX, and X. This leads to formation of incomplete prothrombin molecules that cannot form a thromboembolus. The therapeutic effect of these medications is achieved after 24-36 hours, and in the meantime patients must be covered with heparin or LMWH. If a patient on anticoagulant therapy must undergo a surgical procedure, this therapy must be stopped 3-4 days prior to surgery, and heparin or LMWH therapy should be initiated. An overdose may lead to bleeding; in that case vitamin K and fresh frozen plasma should be administered intravenously.

Dextran is a polysaccharide that is used instead of plasma for volume resuscitation. It enhances hemodilution and blood perfusion, decreases thromboeyte adhesion, inhibits factor VIII and enhances the effect of thrombolytic agents.22,25,26. The thromboembolic prophylactic regime is 500 ml peroperatively, 500 ml postoperatively, and 500 ml for the next two days, and in high risk patients an additional two days.

Aspirin in a dose of 100 mg/24h is used to prevent DVT and coronary disease. It irreversibly acetylates cyclooxygenase by blocking conversion of arachidonic acid into thromboxane.22 Preoperatively, aspirin should be discontinued 2-3 days prior to surgery to reduce the possibility of perioperative bleeding.

CONCLUSION

Diseases of the aorta should be avoided as comorbidities for elective nonvascular surgeries, as they carry a high incidence of morbidity and mortality. Elective surgeries should be postponed until the aorta is surgically treated. If that is impossible, as in case of an urgent surgery, then preoperative and perioperative focus should be on maintaining normal blood pressure levels and hemodynamic stability.

As far as peripheral artery diseases are concerned, anesthesiologists must be familiar with the specificity of the disease, especially if a multi-organ systems are affected, and with the possibility of development of different complications. Besides the hypertension, and coronary and carotid disease that are almost always present in patients with peripheral artery disease, it is important to bear in mind the fact that those diseases frequently have an autoimmune component, and that glucocorticoids and other immunosuppressive medications are used for their treatment.

The most important fact concerning venous disease is the preoperative possibility of DVT and PE, so the focus must be on prophylactic measures against those complications.

SUMMARY

PREOPERATIVNA PRIPREMA BOLESNIKA SA VASKULARNIM BOLESTIMA U NEVASKULARNOJ HIRURGIJI

Bolesnici sa vaskularnim oboljenjima, najčešće uzrokovanim procesom ateroskleroze, koji se spremaju za nevaskularnu operaciju, često imaju i promene na kardiovaskularnom sistemu u sklopu svoje vaskularne bolesti. Komplikacije od strane kardiovaskularnog sistema su, pored infekcije i respiratornih komplikacija, najčešće u perioperativnom periodu. U njih spadaju koronarna bolest, hipertenzija, srčana slabost, plućna hipertenzija, renovaskularna hipertenzija itd.

U preoperativnoj pripremi potrebno je da se na terapiji β-blokatorima, antihipertenzivima, antiagregacionoj i antilipogenoj terapiji. Elektrokardiogram (EKG) i transtoralna ehokardiografija predstavljaju minimum preoperativne evaluacije, jer komplikacije mogu nastati i bez predhodnih tegoba od strane kardiovaskularnog sistema.

Bolesti vena spadaju u najčešća oboljenja savremenog doba svih uzrasta i rasa, a kako invazivno-diagnostičko-terapijske procedure podstiču leziju endotela vena neophodna je perioperativna profilakska tromboze dubokih vena (TDV-a) subkutanim heparinom ili niskomolekularnim heparinom (NMH).

Kljucne reči: vaskularne bolesti, nevaskularna hirurgija, preoperativna priprema, perioperativne komplikacije, bolesti venskog sistema

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