Transcatheter aortic valve implantation(TAVI)

TAVI is becoming a popular option for patients with severe aortic stenosis who are poor surgical candidates. The procedure is less invasive than open aortic valve replacement; however, perioperative complications from this procedure can be serious and life-threatening. Vascular access is usually obtained via the common femoral artery; the prosthetic valve must travel from this entry point to the heart, and then the valve must be deployed in a manner that disables native valve function.

Complications resulting in vascular injury include **dissection/perforation/occlusion along the ileofemoral axis (incidence 11.7%)** , suture delivery system failure, aortic annular dissection, and aortic annular rupture.

Complications from valve positioning and deployment include **atrioventricular block requiring pacemaker implantation (incidence 15.7%)** , prosthesis dislocation/embolization, retrograde embolization, acute coronary obstruction, paravalvular regurgitation, pericardial tamponade, TIA/stroke, acute renal failure requiring renal replacement therapy, and prosthesis-related endocarditis.

Acute ST changes observed on EKG and or LV dysfunction observed on TEE immediately after deployment of the valve suggest acute coronary occlusion. This complication can be managed by percutaneous coronary intervention and stent placement. “Reported cases of coronary obstruction after TAVI occurred more frequently in women, in patients receiving a balloon-expandable valve, and the LCA was the most commonly involved artery. Percutaneous coronary intervention was a feasible and successful treatment in most cases.”

Sources

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2. Acute mitral insufficiency

Caused by papillary rupture, acute MR can present itself with the following symptoms and signs.

Symptoms/Signs: acute CHF, hypotension, increased pulmonary congestion. Increased Left atrium pressures because no time for left atrial compensatory mechanisms to occur and a V wave seen on the LAP, PAP, or PCWP tracing. On echocardiography, the LV and LA size will be normal (since it just occurred), yet the MR jet V wave will be increased

The treatment of choice for acute mitral insufficiency is MV Replacement if insufficiency is severe. However, most of these patients need some sort of medical stabilization/treatment. In the acute period, IV vasodilators are the first line of treatment. Specifically, sodium nitroprusside is the most commonly used medication. Nitroprusside will help to vasodilate which will reduce afterload and thus increasing cardiac output, improving mitral incompetence, and diminishing pulmonary congestion. The use of nitroprusside may, in turn, lead to hypotension that is best treated with a positive inotrope (i.e. dobutamine). In the patient with acute severe MR and cardiogenic shock from ischemic rupture of a papillary muscle, pharmacologic support of the left ventricle, often accompanied by mechanical support with IABP counterpulsation, may be necessary.

Long term treatment for mitral insufficiency depends on the degree to which the left ventricle is impaired. If the LV function is preserved, then vasodilator therapy is not indicated. If the pt has impaired LV function and is not a surgical candidate, then vasodilator therapy is indicated (i.e. hydralazine).

In mitral insufficiency (or regurgitation), blood flows from the LV to the LA during systole Physiology differs between acute and chronic MR:

- Chronic MR = Chronic Volume Overload
- Atrial and ventricular chamber enlargement
- Ventricular wall hypertrophy
- Increased ventricular compliance
- Decreased EDP and decreased O2 consumption
- Decreased contractility
- Normal or near normal EF despite significant ventricular dysfunction
-
- Acute MR = Acute Volume Overload
- No compensatory ventricular enlargement
- Increased filling pressures
- Increased pulmonary pressures
- Decreased cardiac output
- Pulmonary edema

In both cases, regurgitant volume is related to:

- Size of the regurgitant orifice (depends on ventricular size)
- Time available for regurgitant flow
- Pressure gradient across the valve

Measures that reduce regurgitant flow:

- Preload reduction: reduces ventricular size and size of regurgitant orifice
- Increase heart rate: also reduces ventricular size and size of regurgitant orifice, decreases time for regurgitant flow
- Arteriolar Dilators: reduce ventricular pressure gradient

Therefore, medical management of chronic MR includes:

- Preload: Normal to low normal
- Afterload: Reduce
- Rate: Normal to high normal
- Rhythm: Maintain sinus rhythm
- Contractility: Normal

Management of acute MR includes:

- Afterload reduction with arteriolar dilators
- Use of an inotrope with a vasodilator if the patient is hypotensive
- Use of an intra-aortic balloon pump to stabilize the patient prior to surgery

Sources

> Keys to the Cart: September 25, 2017; A 5-minute video review of ABA Keywords
>
> See also Mitral Insufficiency: Pharmacologic Treatment

3. Cardiac tamponade

Clinical findings consistent with cardiac tamponade include tachycardia, dyspnea, chest pain, diaphoresis, decreased arterial pulsation, muffled heart sounds, elevated jugular venous pressure and pulsus paradoxus. Classically Beck's triad is used to characterize three signs of cardiac tamponade: hypotension, jugular venous distention and muffled heart sounds.

Diagnosis of cardiac tamponade relies mainly on index of suspicion especially for patients who are at risk for acute cardiac tamponade physiology (those who have recently had cardiac surgical procedures, electrophysiology procedures). Patients also may have large chronically accumulating pericardial effusions. These patients usually present with significant shortness of breath and chest discomfort prior to developing tamponade physiology since the pericardium stretches and grows appreciably with chronic fluid accumulation.

Imaging modalities helpful in the diagnosis of cardiac tamponade include chest X-ray, electrocardiogram, cardiac catheterization and echocardiography. On chest X-ray the patient will have an enlarged, widened mediastinum with the heart appearing enlarged after effusion of > 200mL has accumulated. The heart may appear as a flask or water bottle. The ECG will demonstrate low voltage throughout the leads in addition to electrical alternans which is an alternating normal-large voltage with reduced voltage due to anterior-posterior swinging of the heart in its fluid-filled pericardial sac. On right heart catheterization there will be an attenuated Y-descent on the right atrial waveform with equilibration of central venous pressure, pulmonary artery pressure and pulmonary capillary wedge pressure. Cardiac catheterization will show elevation and equalization of pressures in all the cardiac chambers in advanced cardiac tamponade along with varying peak aortic pressures by > 10-12 mmHg (due to pulsus paradoxus) as well as decreased cardiac output.

Echocardiography is perhaps the most helpful method of diagnosing pericardial effusions and cardiac tamponade. Echocardiography is relatively easy to perform (as compared to cardiac catheterization) and is minimally invasive. The size of the effusion can be measured by freezing the image and measuring the echo-free pericardial space (which normally is not able to be discerned) with the caliper function. Size of effusion is classified into small (< 9 mm), moderate (10-19 mm) and large (> 20 mm). Several characteristic features of pericardial effusions and cardiac tamponade are evident on echocardiograms, either transthoracic or transesophageal. Chamber collapse is easily visible on echocardiography and the effect of variation with spontaneous respiration may also be visualized. Right atrial collapse is very common (and not specific for cardiac tamponade) but it is a more sensitive sign when it persists for at least 30% of the cardiac cycle. During diastole the right ventricular free wall inverses and the right atrial wall inverses at end diastole. Left atrial collapse is less common in cardiac tamponade, only occurring in 25% of patients, but it is a specific sign for cardiac tamponade. Left ventricular collapse is rare due to muscular left ventricular wall. Left shift of the interventricular septum is a specific sign for cardiac tamponade, visible on echocardiography, and accounts for the clinical sign of pulsus paradoxus.

Sources

Keys to the Cart: March 19, 2018; a 5-minute video review of ABA Keywords

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