Problem Based Learning Discussion 6

Epiaortic Scanning: How to do it and why it’s important

Learning Objectives
At the conclusion of this discussion, the participant will be able to:

1. Define the risk of perioperative neurological injury
2. Review the basis of grading atheromatous lesions
3. Describe the rationale and technique of performing an epiaortic exam

Case:
A 74 year old male presented to his local doctor with chest pain on mild exertion. He had a history of hypertension, diabetes and moderate chronic obstructive pulmonary disease from 54 years of tobacco abuse. He had a previous myocardial infarction three years earlier that was treated with percutaneous revascularization. Eventual cardiac catheterization revealed diffuse multivessel coronary disease, requiring surgical revascularization. He was scheduled for a CABG surgery. In the surgical work-up, the patient was found to have bilateral carotid plaques with hemodynamically insignificant stenosis. There was no history of stroke or TIA in the past. He also had mild LV dysfunction with an ejection fraction of 35% and mild mitral and aortic regurgitation.

After an uneventful induction, a TEE probe is passed and a comprehensive exam is performed. Among the findings include a sclerosed aortic valve without stenosis, but with mild aortic regurgitation, mild central mitral regurgitation with a normal sized left atrium, sinotubular calcification in the ascending aorta, and a 4.5 mm irregular plaque in the descending aorta close to the junction with the aortic arch.

Issues:

1. What is the patient’s risk of a postoperative neurological complication?
2. Dose he require a carotid intervention? If yes, when should this be performed?
3. How would you grade the detected atheroma?
4. Would you recommend an epiaortic ultrasound scan? If yes, how would surgical management change?

Discussion:

1. What is the patient’s risk of a postoperative neurological complication?
Atherosclerotic disease of the aorta is strongly associated with neurological complications after cardiac surgery. Both, stroke and neurocognitive dysfunction (NCD) can be devastating for the patient and their family, and have major implications for quality of life and hospital resource utilization.

Several large studies have categorized adverse cerebral outcomes after cardiac surgery into type I (death to stroke, non-fatal stroke, TIA, stupor or coma) or type II (decline in intellectual function, memory deficit and seizures) outcomes. Roach et al reported a 3.1% and 3.0 % incidence of both outcomes respectively after CABG surgery. In a study examining patients undergoing intracardiac procedures combined with CABG, Wolman found adverse cerebral outcomes in 16% of patients, being nearly equally divided between type I (8.4%) and type II outcomes (7.3%). Their group also showed that resource utilization was significantly increased with this complication. Considering the large number of cardiac surgical procedures performed worldwide, the implications of these adverse events on health-care stay and costs are significant. The specific incidence of NCD alone after cardiac surgery has also been studied extensively and varies widely depending upon the definition used, population studied, tests used to evaluate NCD, and the timing of the evaluation after surgery. In a longitudinal study by the Neurological Outcomes Research Group (NORG) at our institution, the incidence of NCD after coronary artery bypass graft surgery (CABG) was found to be 53% at discharge, 36% at 6 weeks after operation, and 24% at 6 months. Neurocognitive decline significantly reduced quality of life after surgery, again highlighting the importance of preventing this complication.

The relationship between aortic atheroma and stroke following cardiac surgery has been confirmed in several studies. The location of atheromatous plaques in separate regions within the ascending aorta has also been evaluated in their association with stroke. From a mechanistic viewpoint, the relationship is simple – atheroma may be detached from surgical manipulation for cannulation, cross clamping and aorto-coronary anastomoses, and get redirected to the cerebral circulation where they cause ischemic complications. However, the relationship between aortic atherosclerosis and NCD is more complex.

Some known risk factors for NCD after cardiac surgery include advanced age, lower level of education, apolipoprotein E4 genotype, rate of rewarming after hypothermic cardiopulmonary bypass (CPB), and postoperative hyperthermia. A major mechanism implicated in NCD after cardiac surgery is multiple brain microemboli, and a correlation exists between atherosclerotic aortic disease and the number of cerebral emboli detected by transcranial Doppler (TCD). Some studies have a reported a correlation between degree of aortic atheroma burden and incidence of NCD, while others have not confirmed such an association.

A relatively recent development is the finding that certain genetic variations may increase susceptibility for cardiac surgery associated adverse cerebral outcomes. Mathew and colleagues have shown that certain platelet glycoprotein polymorphisms and P-selectin and C reactive protein variants predispose individuals to NCD after CABG surgery.

This patient has a few known risk factors for postoperative stroke: age, hypertension, diabetes and carotid plaques. Per the STS risk index, his risk of postoperative stroke and death is 2.1% and 2.0% respectively. Should the patient have required concurrent aortic valve replacement for significant stenosis, the risk of stroke and death would have increased to 3.1% and 3.6% respectively.

2. Dose he require a carotid intervention? If yes, when should this be performed?
Approximately 22% of patients undergoing CABG surgery have more than 50% stenosis of at least one carotid artery. The stroke risk after isolated CABG is about 2% while after carotid endarterectomy (CEA), the stroke risk is about 5% (independent of symptom status). Combining CEA with CABG surgery may have some advantages in symptomatic patients in reducing post CABG stroke risk, rather than subjecting a patient to the same level of risk twice for independent surgeries. Early attempts at concurrent CABG and CEA resulted in high rates of postoperative stroke of up to 8%.

In a review of the New York State Cardiac Database, Ricotta et al found that the combined rate of stroke or death was 2.2 times higher in the concurrent CEA-CABG group (8.1%) than in the isolated CABG group (3.7%), but after risk adjustment, stroke and death rates were similar in both groups.\textsuperscript{14} Their findings suggest that intrinsic vascular risk is a more powerful determinant of adverse outcome than the surgical procedure itself. However, the combined procedure is not recommended for asymptomatic patients. Although the combined procedure may not increase the stroke risk in symptomatic patients, several centers in North America have now turned to carotid stenting prior to CABG surgery as an acceptable alternative to CEA-CABG surgery.

The patient presented has carotid disease but is asymptomatic and his stroke risk is not elevated from this comorbid condition. He is therefore not a suitable candidate for CEA surgery, either isolated or in combination with CABG surgery.

3. How would you grade the detected atheroma?

The appearance of an atheroma primarily determines its prognostic value. An ideal grading system must take several factors into consideration. First, a smooth intimal surface poses less risk compared to irregular intimal thickening. Second, protruding atheromas carry a higher risk compared to simple intimal thickening. Third, atheromas with mobile elements have the highest risk and must receive special attention. Complex plaques with calcification and/or ulcerations also carry a higher risk of detachment and embolization. They may represent a more severe form of generalized atherosclerosis and are strongly associated with adverse outcome.

There is considerable variability between qualitative and quantitative interpretation of atheroma. There is no uniform definition of irregularity of surface, echocardiographic appearance of ulcerated plaques, calcified lesions or grayscale intensity. Therefore, these features cannot be universally applied to a common grading protocol. For instance, grayscale intensity of an atheromatous lesion can be altered by the machine operator, while the distinction between a calcified plaque versus a densely fibrotic one may not be simple. On the other hand, size and mobility are objective criteria that are less subject to variability in interpretation.

Kronzon and associates introduced a widely used grading protocol in 1992.\textsuperscript{5} They classified atheromas based on intraoperative TEE in any location in the aorta. They also correlated the grade or severity of atheroma with postoperative stroke. They found a highly significant association between mobile plaques and stroke and concluded that aortic manipulation during surgery played an important role in detachment of these mobile atheromas.

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<th>Grade</th>
<th>Severity</th>
<th>Description</th>
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<tr>
<td>Grade 1</td>
<td>Normal</td>
<td>Normal, no intimal thickening</td>
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<tr>
<td>Grade 2</td>
<td>Mild</td>
<td>Intimal thickening ≤3 mm</td>
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Atheroma grading system proposed by Katz et al\textsuperscript{5}
4. Would you recommend an epiaortic ultrasound scan? If yes, how would surgical management change?

Epiaortic ultrasound (EAU) is an ideal method to diagnose and evaluate patients for ascending aortic atherosclerosis during heart surgery. With this technique, a high frequency ultrasound transducer is placed in a sterile sheath and passed onto the surgical field and placed directly on the ascending aorta by the surgeon after sternotomy. To create a suitable air-free interface through which the ultrasound may pass, sterile water is placed within the sheath and into the pericardial well. The fluid between the transducer surface and the sheath provides the ‘stand-off’ needed to adequately image the aorta. The depth of the display is adjusted to center the aorta in the image, usually 6 to 8 cm. The focus of the instrument is placed at the middle of the aorta. The gain is adjusted so the echoes are just barely visible in the aortic lumen. Since the anterior and posterior walls of the aorta are perpendicular to the ultrasound, they will appear to be brighter than the sides, which are more parallel and less reflective. The intima is seen as a thin line less than 2 mm thick around the inner edge of the aorta.

The ascending aorta is defined as the area from the sino-tubular junction to the origin of the innominate artery. Five views are recommended by the ASE/SCA for EAU of the ascending aorta: (1) short axis (SAX) view of the proximal aorta; (2) SAX view of the mid ascending aorta; (3) SAX view of the distal ascending aorta; (4) LAX view of the ascending aorta; and (5) LAX view of the proximal aortic arch.\[15\]

In each of the SAX views, the aorta is divided into four quadrants: anterior, posterior, left and right (Fig 1). Right and left are most easily distinguished by identifying the superior vena cava, which is adjacent to the right side of the ascending aorta. Figure 2 depicts the LAX view of the ascending aorta showing the three regions – proximal, mid and distal.

The examination of the ascending aorta should be thorough and systematic. Significant lesions are often may be adjacent to normal. Start by placing the probe over ascending aorta and rotate it SAX image is seen. The angled inferiorly until the aortic view. Then the probe is slowly proximal, mid and distal thirds is seen. The probe is then

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<tr>
<th>Grade</th>
<th>Moderate</th>
<th>Severe</th>
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<tr>
<td>Grade 3</td>
<td>Sessile atheroma &gt;3mm</td>
<td>Protruding atheroma with mobile components</td>
</tr>
<tr>
<td>Grade 4</td>
<td>Sessile atheroma ≥5 mm</td>
<td>Protruding atheroma with mobile components</td>
</tr>
<tr>
<td>Grade 5</td>
<td>Severe</td>
<td>Protruding atheroma with mobile components</td>
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Fig 1: SAX view of the mid ascending aorta. A=anterior, P=posterior, RL=right lateral, LL=left lateral; PA=pulmonary artery, SVC=superior vena cava.

Fig 2: Long axis view of the ascending aorta.
rotated 90 degrees until a long axis image of the aorta is seen and then the proximal, mid and distal thirds are imaged once again.

The location of lesions in relation to the four axial quadrants is determined with the short axis views and their location proximally or distally with the long axis. The planned locations for manipulation of the aorta (cannulation and clamping sites) are then each carefully examined in short and long axis. The surgeon can determine the location of lesions by noting the position of the probe when the lesion is in the image. Many lesions will also be palpable and can be located in this manner. Calcified lesions of the anterior wall of the aorta may obscure the posterior wall because of acoustic shadowing. Moving the transducer to either side of such a lesion and then angling it to direct the imaging plane under the plaque may allow imaging of the posterior wall.

The eventual goal of EAU is to identify atheromatous disease and modify surgical technique to prevent atheroembolism and stroke. Reported modifications range from simply changing the location of cannulation and clamping sites to replacing the entire ascending aorta when it is severely and diffusely diseased. Another approach is to find with EAU a suitable location for cannulation, construct the distal anastomoses with cold fibrillatory arrest while cooling the patient, and then construct a proximal anastomosis without clamping the aorta using a brief period of circulatory arrest. The other vein grafts may be connected to the first as the patient is rewarmed. With the increasing use off-pump techniques to revascularize the myocardium it is possible to avoid all manipulation of the ascending aorta (‘no touch’ technique) by constructing bypass grafts with arterial conduits such as the internal thoracic (mammary) arteries without cardiopulmonary bypass. Vein grafts and free arterial grafts may be connected to the in situ arterial conduits. Some devices were also developed to reduce embolic risk. One such device allowed rapid vein graft anastomosis with the aorta without the need for cross-clamping. However, the higher risk of adverse outcome, including early graft closure precluded its widespread adoption.

While it seems unlikely that trials of sufficient size and power will be performed to determine the ideal neurprotective strategy, we are probably in a better position to avoid atheroembolism and its consequences knowing when and where such lesions are present with EAU.

In the presented case, an EAU is desirable, in light of the TEE-detected atheroma in the descending aorta. Surgical management may change, depending on findings on the EAU scan.

References