A 70 year old male with a history of chronic hypertension and hyperlipidemia is undergoing a minimally invasive aortic valve replacement for severe calcific aortic stenosis with preserved LV function. The surgeon has elected to cannulate the femoral artery and femoral and jugular veins for connection to the extracorporeal circuit (ECC). The patient is being monitored with a left radial artery line, a PAC, cerebral oximeter and TEE. Shortly after going on CPB his systemic arterial pressure falls to 20 mm Hg and resists usual therapy.

**Study Questions:**

1. What is your differential diagnosis and how would you evaluate the various possibilities?
2. What other evidence would you look for which could suggest an acute (iatrogenic) aortic dissection?
3. What is the best way to make the diagnosis?
4. How would you handle this complication?
5. How might the diagnostic evidence and management be different if he had undergone conventional arterial cannulation of the distal ascending aorta?
6. What are the mechanisms/sites of origin/consequence of acute (iatrogenic) aortic dissection associated with cardiac surgery. When might it occur?
7. What is its incidence and mortality?
8. What are the risk factors for its occurrence?
9. Can it occur in infants and children and in off-pump cases?
10. What can/should be done to minimize the risk and assure early diagnosis and prompt management?
Discussion:

In a review of 7 series of nearly 60,000 cases of cardiac surgery the incidence of acute (iatrogenic) aortic dissection ranged from 0.08-0.35% (average 0.21%) and was associated with a mortality of 15-50% (average 25%) (Murphy 1983, Gott 1992, Still 1992, Ruchat 1998, Aoyagi 2000, Fleck 2006, Hurt 2008). The aortic cannula was the origin in about 40% of cases, with an incidence of 0.06-0.1%. The incidence of arterial cannula associated dissection is about 0.06-0.09 for ascending aorta cannulation, 0.7% with axillary cannulation and about 0.2-1.3 with femoral cannulation. [Also see results of a recent French survey by Charriere et al (2007)].

The site of origin most commonly is the arterial cannulation site. This can be due to direct trauma (at time of insertion or subsequent movement or manipulation) or indirect trauma from the high velocity jet which may lift up the atheromatous endothelium. Other sites, in approximate order of decreasing frequency, include the aortic cross clamp, the antegrade cardioplegia cannula, the partial occluding aortic clamp, the site of anastomosis of coronary grafts, and an aortotomy.

When associated with the arterial cannula dissection typically presents shortly after initiating CPB but can occur at anytime including late postoperatively. That associated with other sites dissection more commonly occurs with release of clamps or later.

With dissection, intra-luminal blood gets into the walls of the aorta and dissects (usually in the direction of flow). This results in loss of perfusate, obstruction of flow out various side branches of the aorta, and sometime frank free rupture of the aorta with massive hemorrhage.

Clues to its occurrence include hypotension, loss of venous return and perfusate, increase arterial line pressure or impedance, evidence of impaired organ perfusion (oliguria, dilated pupils, changes in BIS, EEG, Cerebral oximeter, TCD, ECG and systemic acidosis), blue discoloration and or distension of the ascending aorta or around the cannulation site, and bleeding from needle holes, arterial incisions, and cannulation sites. The latter may be less obvious when the source of the dissection is the femoral artery cannulation. The best way to establish the diagnosis is by TEE or epiaortic scanning.

Suggested risk factors include location of cannulation (femoral > axillary > aorta), older age, chronic hypertension, diseased or dilated aorta, atherosclerotic peripheral vessels, cystic medial necrosis, high arterial pressure at time of cannulation, decannulation and application and removal of aortic clamps, and type of purse-string suture and cannulation technique. In Hurt’s series (2008) the incidence was about the same with CAGB (0.3%) as with valve cases (0.2%) and age was not a risk factor. Acute dissection has been reported in pediatric cases and off-pump cases.
Proposals to minimize its occurrence include lowering arterial pressure during cannulation, decannulation and application and removal of aortic clamps, care with the insertion of cannulae, checking for pulsatility and pressure in the arterial line after cannulation, and checking impedance/resistance/line pressure with a test infusion before going on full CPB. Use of a high pressure audible alarm with pump shut off of the perfusion inflow line may limit the extent of the injury. DeBois and colleagues (2003) have described an ingenious test to rule out a misplaced arterial cannula. During retrograde arterial priming (RAP), fluid is drained from the arterial cannula into a collection bag. Absence of fluid return or a flow below 500 mL/minute is indicative of either arterial line occlusion or cannula misplacement.

For early diagnosis, paying attention to, and evaluating all possible causes of hypotension that occurs during initiation of bypass (and not committing to CPB by giving cardioplegia or opening the heart before one has ruled out dissection), frequent visual inspection of the cannulation site and TEE monitoring of the aorta are recommended. It is also worth noting that placement of the antegrade cardioplegia cannulae may cause dissection and one should be wary of high pressures in this line during administration of cardioplegia. When femoral cannulation is used TEE monitoring of the descending thoracic aorta during initiation of CPB and frequently during CPB is advisable.

Management of this catastrophe is influenced by the type of cannulation, the stage of the cardiac surgery, and the extent of the dissection (evaluation aided by TEE). When the dissection occurs at the initiation of CPB via a femoral arterial cannula, immediate discontinuation of CPB and re-establishment of cardiac output by the patient’s own heart (may require volume and drug resuscitation), if feasible, may resolve the situation. (The dissected flap may close and the false channel disappear). One could then consider re-instituting CPB via an alternate arterial cannulation (ascending aorta, but into the true lumen, subclavian, or innominate artery) and continue with the planned cardiac procedure with or without repair of the ascending aorta. When the ascending aorta is the site of the initial cannulation, CPB should be stopped immediately and flow reinitiated via the femoral, subclavian or innominate (after assuring that the dissection has not extended into these vessels) or into the true lumen in the aortic arch through the flap (perhaps with the aid of ultrasound and a guidewire). Initiating deep hypothermia in anticipation of need for circulatory arrest to repair the ascending dissection is often advocated. Adjunct cerebral protective techniques including selective cerebral perfusion may also be necessary if the dissection is extensive and involves the arch vessels. Depending upon the extent of the dissection, this may be repaired by closed exclusion suturing, insertion of a patch, or graft replacement of the ascending aorta ± some of the arch, but usually graft replacement is recommended and has given the best results.
Bibliography:

Books


Case Series


Other pertinent articles

