Mechanical Interventions In Cardiogenic Shock

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In established cardiogenic shock mortality is close to 100% when all these parameters are present.

High wedge pressure.
Hypoperfusion.
Low urine output.

People do not survive unless you do further intervention.

I would like to divide my lecture into two parts.

Early intervention; when the cardiogenic shock is not established, (patient is hypotensive).

When the patient has established cardiogenic shock, they have low cardiac output and hypoperfusion.

Patients who have myocardial infarction and develop hypotension their blood pressure is low may be (60,70 mmHg) and have extensive infarct on electrocardiogram. These are the patients who may go into fully developed cardiogenic shock. Once the patient has developed cardiogenic shock, no matter what medication you use, it may be dopamine, dobutamine, Swan Ganz catheter, intraaortic balloon pumps, mortality is very high.

We can divide the patients into two groups:

1. Patients who have M.I. and present to emergency room within 2-4 hours.
2. Patients who have other reason for cardiogenic shock including acute M.I., cardiomyopathy or valvular heart disease, and also the patients who develop specific lesions of myocardial infarction like VSD, papillary muscle rupture or ventricular aneurysm formation.

If a patient presents to the emergency room in hypotension.(blood pressure being low in the range of 70-80), and has extensive infarct on electrocardiogram, that means the patient might develop full picture of established cardiogenic shock, and will have more and more damage. Main goal is to prevent that damage so that the patient can live with preserved myocardium and come out of the shock.

There are two types of therapies in acute intervention:-

1. Thrombolytic therapy.
2. Mechanical recanalization of the vessel which is completely occluded and is responsible for cardiogenic shock.

Thrombolytic Therapy

The medications which are commonly used are streptokinase, urokinase and tissue plasminogen activator (TPA).

Streptokinase

When you want to use thrombolytic therapy, patients have to reach the emergency room in time. Young patients do not have time to develop collateral flow. Their myocardial infarction is completed within 3 hours in the absence of collateral circulation. Myocardial damage starts in 1/2 hour and if you do any intervention after 3-4 hours it is not of much benefit. Advantage with the older people is that they usually have collateral circulation and you can extend that period to a few hours, may be even a day after pain starts.

Streptokinase can be given by infusion and you usually give steroids before streptokinase because some people do have allergic reaction to streptokinase and develop anaphylactic shock.

Inj. Solucortef 200 mg I/V is given. Then we use streptokinase.

Different protocols are being used. Some people use 7,50,000 units of streptokinase and this should be infused over 30-40 minutes. If infused quickly it can cause severe hypotension. At the same time you give heparin bolus because you have to maintain anticoagulation along with thrombolysis by streptokinase and then start them on heparin drip. Always monitor their P.T.T. very carefully. You should keep P.T.T. above 80-90 sec. P.T.T.1 1/2 to 2 times the control is not enough.

Area in the vessel is very thrombogenic and there is always a tendency even at that high P.T.T. for the clot

to re-form. Therefore, after thrombolytic therapy, you must use heparin to keep the artery clear.

At the same time there is tendency for the spasm in that area where there is injury to the vessel wall, you must use something to prevent the spasm and the best thing to prevent the spasm is calcium channel blockers. I mostly use Diltiazem which does not cause tachycardia and tachycardia as you are well aware, is harmful in myocardial infarction. You try to preserve as much myocardium as possible, and you have to prevent tachycardia.

Also you need to use nitrates to prevent spasm, and antiplatelet agents like asprin as dipyridamole Some patient cannot tolerate asprin and they have allergic reaction to asprin, in them you can use ibuprofen.

There are side effects of streptokinase.

- Patient may develop an anaphylactic reaction especially if patient had recent streptococcal. infection.
- Many people develop hypotension. This is a common phenomenon with streptokinase infusion especially given rapidly. For that reason infusion of streptokinase should be slow. If the patient develops hypotension, mostly they do respond to I/V fluid therapy.
- Bleeding complications are fairly common. Mostly they develop 24-48 hours after the patient is on heparin. Initially, bleeding is less common after streptokinase infusion although it affects all the coagulation system. Mostly it is G.I. bleed or a retroperitoneal bleed. Some people do develop intracranial haemorrhage and one should be aware of that in patients with a history of stroke. For patients with history of recent surgery one should not use streptokinase therapy.
- Arrhythmias are very common, called reperfusion arrhythmia. Sometimes you have ventricular tachycardia and ventricular fibrillation with streptokinase infusion or other thrombolytic therapy or mechanical recanalization for that matter. What you need to do is to use anti-arrhythmic treatment. Mostly what I do in my practice is to give a Lidocaine bolus and keep the patient on lidocaine infusion.

If lidocaine is unable to control the arrhythmias, as is the case many times, you can use other drugs.

You can give procainamide 100mg I/V slowly every 5 minutes, (total 500-1000 mg) and then put the patient on procainamide drip-3 mg/min. and then check procainamide blood level. Or you can use bretyllium in combination with lidocaine or alone.

You may use tissue plasminogen activator (TPA) It is much more expensive than streptokinase. Effectiveness especially in these patients who are heading towards cardiogenic shock, probably, is not any different as compared to streptokinase. In initial two hours of myocardial infarction, whether you use streptokinase or TPA, patency rates are the same. Later on after two hours the patency rate is better with TPA.

TPA is given as a bolus 10 mg and then you start drip. Total 100 mg are given over a period of three hours. Same time, you start them on heparin after giving a heparin bolus. Calcium blockers, antiplatelet agents, and nitrates are themselves a part of the treatment.

Complications are almost the same as streptokinase. Hypotension is not very common.

Bleeding is quite common but less common than streptokinase.

Arrhythmias are very common and are to be treated the same way.

Mechanical Recanalization

Initially, we did not have any drug available for treating cardiogenic shock and thrombolytic therapy is not effective all the time. In best studies the patency rate after streptokinase or TPA is 70-80% or lower. In some studies patency rate of streptokinase is 50-60% and TPA in the range of 60-80%.

To take the patient for coronary arteriography you have to have very efficient people, who are trained and can diagnose and treat the patient properly.

You have to have very efficient cath. lab personnel who can set up things very quickly, and obviously efficient staff in terms of nursing staff and doctors who have experience and can deal with the complications immediately.

After doing coronary arteriography once you find out which vessel is occluded you can proceed with proper procedure.

The advantage of recanalization by mechanical means over thrombolytic therapy is that with thrombolytic therapy it takes long time to recanalize these vessels, and time as you know is of importance in acute myocardial infarction and also residual stenosis after thrombolytic therapy is high. Most of the times 80-90% of residual stenosis, although you have established circulation but still you are left with very severe stenosis in that area and it can become more stenosed. With mechanical means you have recanalized the vessel more

quickly and more efficiently and have supplied circulation to that area immediately.

Other medication along with mechanical recanalization you need to use is calcium blockers, because of spasm, even with routine angioplasty there is spasm in that area and we use nifedipine sublingual to relieve the spasm. You need to put them on calcium blockers. I do not use routinely nifedipine after recanalization. I use diltiazem most of the times for the reason that it is tolerated better. Usually I use 60 mg diltiazem four time a day or you can go up to higher doses which will effectively prevent spasm. If there is spasm and patient still has pain you can always use (Nifedipine) in addition to diltiazem. You may use sublingual nifedipine as well for immediate effect.

In addition to calcium blocker most of the times we use nitrates I/V. Why I/V nitrates? because easier to maintain the therapeutic levels with I/V nitrates as compared with the tablets. I/V nitrates are effective immediately whereas cutaneous preparations take about 30 min, to be effective. I/V NTG later can be changed to cutaneous patches. I would not recommend 24 hours patches because effectiveness of the patch is lost after some hours. Most of the times we use percutaneous patches which you can change every 6 hours. It is also important to give the patients nitroglycerin free intervals so that patients do not get tachyphylaxis with nitroglycerin, and nitroglycerin should not become ineffective.

Antiplatelet agents should be instituted at the same time. Heparin should be continued for at least 2-3 days after myocardial infarction.

Now let us discuss the established cardiogenic shock.

If on initial presentation to emergency room patient is unstable (Hypotensive), we can institute intraaortic baloon counter pulsation (IABP) and plan further care.

When patient requires IABP and other assist devices definitive treatment will be needed. If these patients have to survive they will need surgical intervention.

Different modalities used as supportive measures include:-

1. Intraaortic balloon pump.

2. Left ventricular assist devices.

3. Artificial heart.
A. **Bridging procedures:**

Patient while waiting for donor heart, is kept alive on artificial heart. These are the patients who are dying and need immediate assistance.

B. **Permanent Artificial Heart:**
This has been used few times with success but with very high complication rate.

When surgical correctable lesions like V.S.D. (ventricular septal defect), acute mitral regurgitation are not present only treatment is cardiac transplantation. Patients with specific lesion;

If patient has V.S.D. or acute mitral regurgitation and patient develops cardiogenic shock, he may initially be started with dobutamine. But these people will require IABP. Most of these patients will stabilize with IABP. Once these patients are stable they should be studied immediately and further surgical care should be planned. These patient will not remain stable for long time. For this particular reason time should not be wasted.

Most of the surgeons will like to wait 6-8 weeks for surgery, but one cannot wait that long as gradually patient will start deteriorating and multisystem failure will occur.

Although tissue is very friable in the early part and sutures do not hold in the necrotic tissue, but it is still the best time for surgery. Surgical mortality is 50%. But if we wait for tissue to heal up mortality is very high.

Aneurysm if present can be resected and rarely patients may benefit form coronary artery bypass.

I had a patient a few months ago who had cardiogenic shock. We took this patient to catheterization Lab., put him on aortic balloon pump and studied him. He had occluded right coronary artery, circumflex was occluded, and LAD had about 90% stenosis. As we injected LAD it filled right coronary artery. In other words, area supplied by the collaterals was ischemic. We did bypass surgery and patient survived and went back to work. It is rare that patients improve as such when they have disease that leads to cardiogenic shock.

Intraaortic balloon pump counter pulsation (IABP) is a very important measure in the treatment of cardiogenic shock. Because, as I said, the drugs are just temporary measure they do not help for a long time. When intraaortic balloon pump can be instituted, there is high rate of dependency on intra aortic balloon pump. Once you put the balloon pump in then it is very hard in the established cardiogenic shock to wean off the patient from the balloon pump. But post-bypass surgery the situation is different. These people do improve on

the balloon pump and it is easier to wean off the balloon pump. Balloon pump was introduced in sixties, it really never became popular until 70's when percutaneous technique was introduced. Before that you had to have surgeons to do cut down and approach the artery and put the balloon pump, and now you can do it percutaneously. It is very simple, you anaesthetise the area in the groin, stick the artery like you normally do for angiography, and put the sheath in and advance the balloon to a place jjust distal to the origin of the subclavian artery through the sheath.

When the aortic valve is closed, balloon is inflated and it displaces about 40cc of blood. Some of the blood goes up & some of the blood goes down but it prevents further run off of blood.

During systole, it deflates and heart pumps the blood into an empty space so decreasing work load of the heart, and during diastole it inflates and pushes the blood and perfuses the coronary arteries. Myocardium will get more blood supply and pumping function should improve with this maneuver.

There is increase in cardiac output, in different studies, anywhere from 10-20%. Most of cases have 300-500cc increase in cardiac output which is significant. If somebody has increase in cardiac output of suppose from 2 liter to 2.5 liters, it is a blessing. There is also reduction in systemic systolic pressures. There is rise in systemic diastolic pressure. Mean pressure basically remains unchanged. There is decrease in heart rate because once you are playing with counter pulsation you are taking away load off the heart and response of sympathetic amines, epinepherine and norepinephrine is decreased. So the heart rate is decreased. There is increase in urinary output and decrease in capillary wedge pressure.

There is high incidence of complications with intra aortic balloon pump. Gangrene is fairly common, due to occlusion of diseased vessel. And almost all of these people who need balloon pump have diffuse disease of the distal vessels of the legs.

Embolization is common and also sometimes strokes have been reported.

Rupture of the ventricular free wall is more common as compared to control when you are using an IABP. The reason is probably prolongation of life which otherwise would not have been prolonged, and they would have died with acute myocardial infarction. When you are using intra-aortic balloon pump, monitor their blood count every day. They develop thrombocytopenia so you need platelets count every single day. Patient should also be started on rheomacrodex to prevent thrombosis.

Now the more modern treatment of cardiogenic shock. These are to be used when virtually all the therapies and all the modalities have failed. You are unable to keep the patient stable on intra-aortic balloon pump and all inotropics available and now the patient is dying. These are basically bridging procedures waiting for heart transplantation or ideal artificial heart. There are many kind of assist devices available and they are not in very common use. Some of them are coming in use more and more with the improvement in technology.

The first one is the Pierce pump. You take blood out of the atrium it goes to the pump and then is sent back into the ascending aorta. It has been used a few times with variable success. You must recall that all these patients are basically dying. If these patients live that will be a success.

The other heart which is used is a Penn State heart which has different components to it. One is a polyurethane blood sac. In addition to these it has got a poly-sulfone case two cannulae. One goes to the left ventricle and the other one is connected to the aorta. This is basically driven by air. It has one connection to the aorta, other one to the left ventricular apex. One needs thoracotomy to do that and it is a big surgery. It is only to try to keep the patient alive till donor heart is available.

Permanent Left Ventricular Assist Device

This pump was basically designed to be implanted. So it is a relatively smaller device. The pump is connected to left ventricular apex and other portion is connected to the descending aorta below the diaphragm. Then pump is placed below the diaphragm. The main purpose of this device is to implant it as a pump. F.D.A. has not approved it. There are a few centers authorized to use it.

Then we have other bridging procedure called Phoenix heart which has been used thrice in a centre in Arizona with the approval of F.D.A. as a bridging procedure. It was used for 24 hours and they did transplant the heart although the patient could not survive. He had first heart transplantation which was rejected. They put the patient on the artificial heart as bridging procedure again and got another heart after 24 hours and even that was rejected.

Then we have completely artificial heart like Jarvik-7 heart which has been used three times, with high incidence of complications. Obviously this was not the ideal heart and there is much more to be done,

before ideal heart is available for transplantation.

Ultimate goal at present is to have cardiac transplantation of all such patients. Also baboon heart has been used once in an infant, last year. In fact he was new born with defects with which patient would not have lived for more than a few hours. They transplanted baboon's heart but the patient died.

If the patients have lesion which can be dealt with surgery, surgery is done e.g. V.S.D. repair or mitral valve repair etc. It should not be delayed at all. If their is no surgically correctable lesions then one should go for the device and wait for donar heart to be available.