Annals of African Medicine
Vol. 1 No. 2; 2002: 60 - 71

MANAGEMENT OF CARDIAC EMERGENCIES

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INTRODUCTION

Emergencies are clinical conditions caused by life threatening disorders requiring quick and urgent intervention. Cardiac emergencies are part of a long list of emergencies that medical practitioners (general and specialist) are faced with daily. Cardiac emergencies will vary from one continent to the other or from one country to the other. This review discusses some of the cardiac emergencies in our environment and highlights others that are less common in our practice.

DISSECTING AORTIC ANEURYSM

All mechanisms that weaken the aortic wall, the aortic lamina media in particular, lead to higher wall stress, which can increase aortic dilatation and aneurysm formation, resulting in aortic dissection or rupture.[1] The major inherited disorders include Marfan's syndrome, Ehlers Danlos syndrome and other inherited familial forms of aortic thoracic aneurysm and dissection. [2] Atherosclerosis is the main cause, which leads to compromise of the integrity of the intima. The above leads to intimal rupture, most often at the edge of the plague. Aortic dissection is classified (staged) as Type A and Type B [3]. Type A dissection involves the ascending aorta whereas Type B does not involve the ascending aorta. De Bakery's classification subdivides the disorder into type I (entire aorta), type II (ascending aorta) and type III (descending aorta) [4]. There is a new classification proposed based on further studies that demonstrated intimal haemorrhage, intramural haematoma and aortic ulcer which may be signs of evolving dissection or dissection subtypes.[1] Majority of true cases of dissection starts from the ascending aorta. Symptoms may include sudden pain severe/central, which radiates to the back or sometimes to the arm and neck, can be difficult to diagnose because of spinal vessels involvement, pain with cerebrovascular accident (CVA), pain with cardiac failure, pain with evidence of myocardial infarction etc .[1]

Diagnosis

Back pain without ECG or enzyme changes of myocardial infarction and chest X-ray will show wide mediastinum. [3] The diagnosis is aimed at localizing the tear, extent of dissection, calcification, indications of emergency and aortic arch vessel and side branches involvement. Computerized axial tomography (CT Scan) and echocardiography will show double wall of the aorta. Echocardiography involves Trans thoracic (TTE) and Trans esophageal (TEE). Aortography alone or with Magnetic resonance imaging will confirm the diagnosis. An echocardiography machine with colour flow will also identify the area of dissection. Intravascular ultrasound has also been employed the diagnosis of aortic dissection. Tears, false lumen, leaks and calcification are identified using these imaging techniques though the sensitivity and

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specificity differs from one application to the other. The symptom that exceeds four days is regarded as chronic dissection.

Table 1, Three classifications highlighted above

Stanford Classification[2]	
Type A	Dissection of ascending/descending aorta
Type B	Dissection of descending aorta
De Bakey Classification[3]	
Type I	Dissection of entire aorta
Type II	Dissection of ascending aorta
Type III	Dissection of descending aorta
New Classification[1]	
Class I	Classical aortic dissection with an intimal flap between true and false lumen
Class 2	Medial disruption with formation of intramural haematoma/haemorrhage
Class 3	Discrete/subtle dissection without haematoma, eccentric bulge at tear site
Class 4	Plague rupture leading to aortic ulceration, penetrating aortic atherosclerotic ulcer with surrounding haematoma usually subadventitial
Class 5	Iatrogenic and traumatic dissection

Class 1-5 represent a subdivision to the Stanford or De Bakey classification. (Eur Heart J 2001; 22:1374-1450)

Emergency room steps

An immediate ECG is mandatory, this will help differentiate between myocardial infarction and dissection, diagnostic imaging highlighted above, chest x-ray might not be sufficient to make a diagnosis, other investigations include C-reactive protein, mild to moderate leucocytosis, sight rise in bilirubin and lactic dehydrogenase. It should noted that sometimes ECG might be abnormal when the dissection involves the coronary vessels. The most current specific indicator is raised smooth muscle myosin heavy chain.[1, 4]

Therapeutic decision is base on diagnostic imaging to avoid wrong diagnosis, this entails admitting the patient in intensive care unit, replacement of fluid to haemodynamic deterioration, drugs administered in a separate venous line, unstable patients will require intubation and invasive blood pressure monitoring. The blood pressure should be controlled between 100-120 mmHg. TTE or preferably a TEE will identify pericardial tamponade so that pericardiocentesis is done immediately and it does give clearer view of the ascending aorta without interference.

The following are signs of severe dissection; severe pain, pulse loss, bleeding stopping, fluid extravasations into pericardium, pleural space and mediastinum, aortic regurgitation murmur, increase LV contraction and signs of myocardial ischaemia. Surgery is the ultimate and it is in various forms depending on the complications found. It might require aortic valve

replacement, CABG and implantation of ascending aorta prosthesis. [3,4]

The risk factors associated with dissection of the aorta include hypertension, diabetes mellitus, smoking and any other cause of atherosclerosis.[2] Hypertension should be controlled immediately with necessary drugs. Pregnancy is also associated increased risk of dissection. Steps should be taken towards prevention of aortic dissection by control or eliminating the factors.

ACUTE HEART FAILURE

This occurs mainly in the setting of hypertension, myocardial infarction, pulmonary thrombo-embolism, sudden failure of prosthetic valve etc. Acute heart failure may be in the setting of acute left or right failure and sometimes the whole heart involved. [5]

When there is excessive loss of cardiac muscle in myocardial infarction involving the left or right ventricle, the interventricular septum might rapture leading to ventricular septal defect or there could be acute valvular regurgitation. The valvular regurgitation is sequalae to papillary muscle or chordae rupture; producing sudden mitral or aortic regurgitation. Acute endocarditis might lead also lead to sudden regurgitation; acute left or right ventricular failure, which usually may take the form of diastolic dysfunction. There is usually no peripheral fluid retention but inn the case of left ventricular failure there will be severe pulmonary edema causing (described as cardiac asthma) the patient to present with severe shortness of breath. This presentation especially in the setting of hypertension might be mistaken for bronchial asthma. A third or fourth heart sound might be heard in the apex or left lower sternal edge.

Acute pulmonary edema is a common cardiac emergency, which might be the manifestation of acute heart failure. The patient is managed in cardiac position, high concentration of oxygen is administered by facemask, intravenous diuretics (loop diuretics), venodilators (nitrates), aminophylline and venesection when necessary. Cardiogenic shock results in less tissue perfusion. It is as a result of hypotension and, low cardiac output. Because of poor tissue perfusion there will be oliguria, and cold extremities. To confirm a cardiogenic shock, a pulmonary wedge pressure will be normal and mortality is usually very high. Management involves administering 100% oxygen, infusion of fluids if pulmonary wedge pressure is below 18 mmHg; short acting venodilators (glyceryl nitrate), cardiac inotropes, mechanical assisted device (intra aortic balloon pump).

Management of acute cardiac failure is usually directed to the primary cause, which sometimes might involve surgical intervention. The most common cause in our practice is hypertension, which requires immediate control [6,7]; it is treated as one of the hypertensive emergencies. Acute cardiac failure in setting of hypertension will manifest as diastolic dysfunction.[8, 9] The role of nitrates in the treatment of pulmonary edema is very important apart from diuretics, morphine and oxygen. Pericardial effusion with tamponade, ventricular septal defect from myocardial infarction and sudden mitral or aortic regurgitation from valve failure will require surgical intervention.

CARDIAC TAMPONADE

It is as a result of accumulation of fluid in the pericardial sac that leads to compromise of cardiac function.[10] The

fluid could be hemorrhagic, suppurative or straw colour. Cardiac tamponade can be detected clinically or using imaging techniques like echocardiography, CT scan and magnetic resonance imaging. The clinical signs includes, tachycardia, small volume pulse, pulsus, paradoxosus, raised jugular venous pressure with a "Y "descent. Cardiac apex might not be palpable and heart sounds are usually distant. Echocardiography can detect tamponade even before the manifestation of clinical signs.[11] The echocardiographic signs include, diastolic collapse of right or left ventricular wall and sometimes right atrial wall collapse, apart from detecting the pericardial effusion[12]. Other echocardiographic features include, "swinging heart", left atrial collapse in late diastole and early systole, and mitral E velocity reduction greater than 25% during inspiration.[12] The distance of separation between parietal pericardium and the wall of the ventricle or atrium gives an idea of the severity of the effusion. Management is by pericardiocentesis, which is done under fluoroscopy in the cardiac catheterization laboratory to allow the operator to monitor where the catheter is placed and haemodynamic statues of the patient is also monitored including the ECG and blood pressure. In most third world country set up, pericardiocentesis is done in the open wards hardly with any ECG monitoring needless to talk about haemodynamic monitoring. Sometimes pericardial window might be required especially when there is re-accumulation of fluid. Pericardial effusion and tamponade is not uncommon in our practice, tuberculosis was the main culprit but with the HIV/AIDS emergence the trend seems to be changing. A third of our recent series yet to be published were related to HIV/AIDS[13]

CARDIAC ARREST

Cardiac or cardiopulmonary arrest has a big danger of causing permanent cerebral dysfunction.[14] This is a common situation where medical students and all doctors must know how to manage it. This condition usually causes panic, it is therefore essential that basic procedures are known and followed carefully. Different countries have resuscitation councils that formulate procedures, which enable different health personal, work together e.g. UK Resuscitation Council.

Basic life support

First step is to ensure whether the patient or victim is unconscious and whether there is pulse. This is done by shaking or shouting at the patient and also palpating the radial or carotid pulse (carotid is preferable). If there is no pulse, call for help, begin basic life support: A (air ways), B (breathing), and C (Circulation).[15]

The airway should be cleared by removing mucus, blood clots and objects. Open the airways by flexing the neck and extending the head ("sniffing the moving air position"). To establish whether the patient is breathing, look for rise and fall of the chest wall and abdomen. If there is no respiration begins expired air resuscitation. This is done with the head of the victim titled backwards and the chin pulled forward. The resuscitator takes a deep breath and seals his lips around the victim's mouth and nose. At least four quick puffs are given but mechanical methods of Holger and Nelson, Shaefter and Sylvester are thought to be completely useless because they result in the movement of less air than is required to fill the dead space. A firm thrust to the epigastric region can dislodge foreign body that is difficult to remove.

Circulation is achieved by external chest compression. The heel of one hand is placed over the lower part of the sternum and the heel of the second over the first with fingers interlocked. The hands are kept straight and the sternum is rhythmically depressed by 1½ - 2 inches. Chest compression does not message the heart but the thorax acts as a pump and the heart provides a system of one-way valves to ensure forward circulation. Respiration and compression is continued as follows:

a) Single rescuer -
   Compressions at the rate of 8bpm with two respirations after 15 consecutive compressions.
b) Two rescuers -
   Continuous compressions at a rate of 60bpm and one respiration given at every 5 compressions.

Cardiac Support

In developed countries by the time simple or basic life support has been established more help would have arrived and advance cardiac support can begin. These consist of ECG monitoring, endo-tracheal intubation and setting up of fluids. Immediate therapy includes defibrillation, oxygen and cardio-active drugs. The nature of cardiac arrest and the skill of the personnel will determine what type of drugs is to be administered. The ECG will detect malignant arrhythmias e.g. ventricular fibrillation, which must be defibrillated immediately.

Cardiac CFR for up to 2minutes after each day, do not interrupt for more than 10seconds, except to defibrillate. If an intravenous line cannot be established, consider giving double dose adrenaline, lignocaine or atropine via an endotracheal tube. (Based on recommendation of UK Resuscitation Council 1989).

Mechanisms of sudden cardiac arrest

a) Ventricular fibrillation
b) Asystole
c) Electromechanical dissociation.

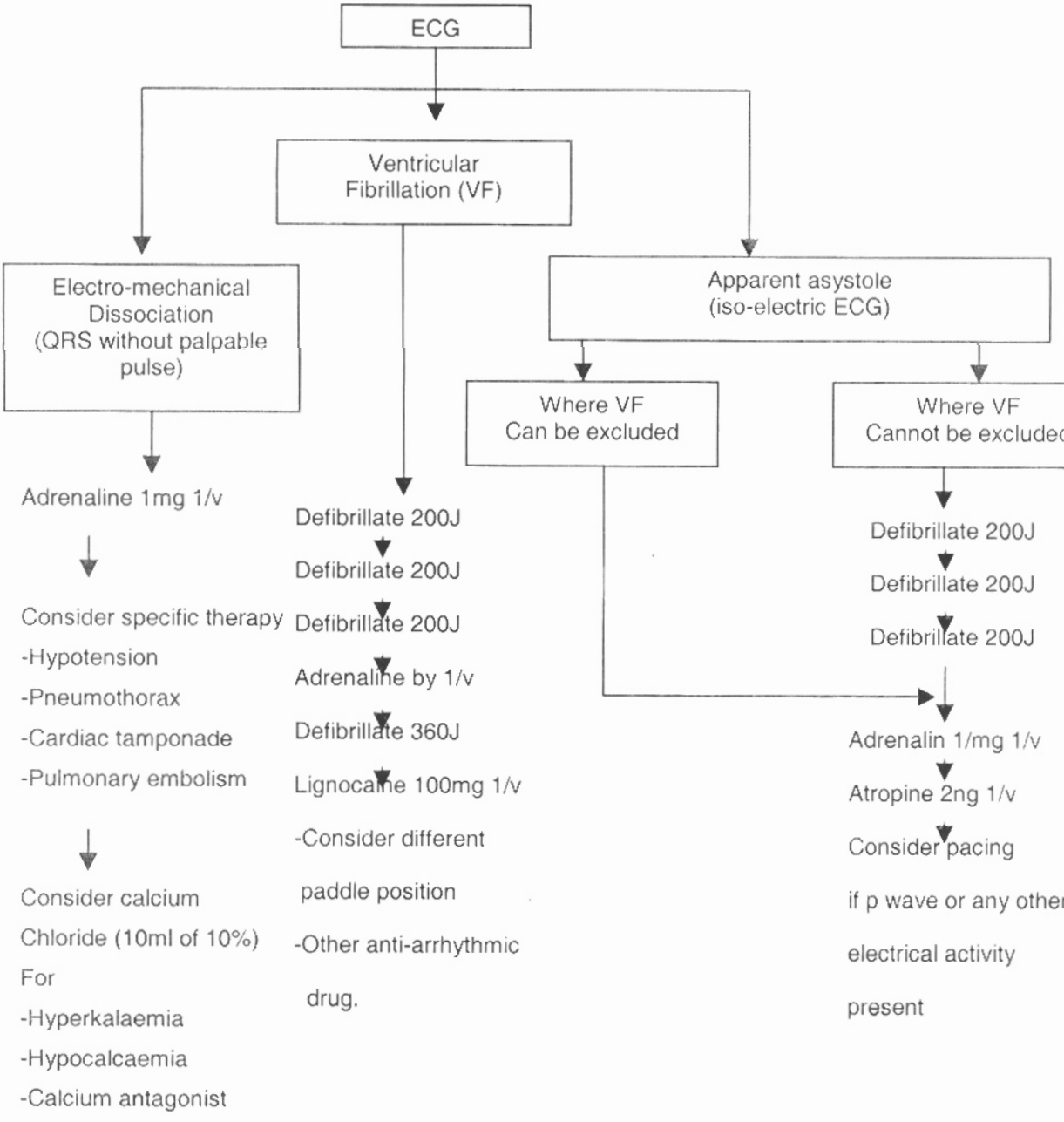
In the western experience ¾ of cardiac arrest is due ventricular fibrillation, statistics in Nigeria are difficult to come by, but ventricular fibrillation might also rank very high. The absence of well-established intensive care units or medical emergencies will make any available statistics suspect. Cardiac arrest following pneumonia gives a cardiac or heart rhythm described as "agonal" (characterized by inexorable slowing and widening of QRS complexes associated with falling blood pressure and cardiac output).[15] It is a difficult arrhythmia to reverse and usually no attempt should be made because it is a result rather the cause of death.

Management of sudden cardiac arrest

- Ventricular Fibrillation readily treated with defibrillation anti-arrhythmic drugs and cardiac stimulants.
- Asystole in more difficult to manage. The heart might respond to atropine or adrenaline. If there is any sign of electrocardiographic activity, emergency pacing should be used.
- Electromechanical dissociation. It is usually due severe mechanical problem like pericardial effusion and tamponade or massive pulmonary embolism. It should be given urgent attention. Other causes include toxic levels of cardio depressants drugs such as beta-blockers; antidote like adrenaline should be administered.

SUDDEN CARDIAC DEATH

Natural death due to cardiac cause heralded by abrupt loss of consciousness within an hour of onset of acute symptom, pre-existing heart


ECG
Ventricular Fibrillation (VF)
Electro-mechanical Dissociation (QRS without palpable pulse)
Apparent asystole (iso-electric ECG)
Where VF Can be excluded
Where VF Cannot be excluded
Adrenaline 1mg 1/v
Consider specific therapy
-Hypotension
-Pneumothorax
-Cardiac tamponade
-Pulmonary embolism
Consider calcium Chloride (10ml of 10%) For
-Hyperkalaemia
-Hypocalcaemia
-Calcium antagonist
Defibrillate 200J
Defibrillate 200J
Defibrillate 200J
Adrenaline by 1/v
Defibrillate 360J
Lignocaine 100mg 1/v
-Consider different paddle position
-Other anti-arrhythmic drug.
Defibrillate 200J
Defibrillate 200J
Defibrillate 200J
Adrenalin 1/mg 1/v
Atropine 2ng 1/v
Consider pacing
if p wave or any other
electrical activity
present

disease may have been known to be present, but the time and mode of death are unexpected [16] and about half of this death is almost instantaneous.[16] The clinical condition is usually used as surrogate implying a specific mechanism is involved. In the Western countries ventricular fibrillation (VF) constitutes 75-80% in adults and the general incidence of sudden cardiac death is put at 0.36-126/1000 [17]. The causes include; cardiac arrhythmias (ventricular fibrillation or ventricular tachycardia), sudden pump failure (myocardial infarction), acute circulatory obstruction (pulmonary embolism) cardiovascular rupture (dissecting aortic aneurysm or myocardial rupture) and Vasomotor collapse (pulmonary hypertension). Most deaths are due ventricular fibrillation and a few due severe bradycardias. The risk factors include atherosclerotic coronary artery disease (CAD), increasing age, male gender, family history of CAD, increased LDL cholesterol, hypertension, smoking and alcohol [18]. It is probably true the most common risk in Nigeria is hypertension with atherosclerosis contributing less than half. Specific risk factors for the event are (sudden cardiac death) increased heart rate and heavy alcohol consumption. The genetic basis for sudden cardiac death include; inherited genetic abnormality affecting key proteins of the heart e.g. Long QT syndrome, Brugada syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia and dilated cardiomyopathy.[16]

Table 2: Recommendations for risk stratification for sudden cardiac death: myocardial infarction and hart failure

	Recommendations	Level of evidence
Demographic variables	Class I	A
LV ejection fraction	Class I	A
HR variability or baroreflex sensitivity	Class I	A
Left ventricular volume	Class I	A
Ventricular premature beats	Class IIa	A
Non sustained VT	Class IIa	A
Resting Heart rate	Class IIa	A
Late potential	Class IIb	A
QT interval	Class IIb	B
Electrophysiological study	Class IIb	A
T wave alternans	Class IIb	B
Heart turbulence	Class IIb	B
Patency of infarct related artery	Class IIb	B
QT dispersion	Class III	B

The risk stratification is for SCD only (adapted from Eur Heart J 2001; 22:1374-1450)

The act of defibrillation is very important, the placement of paddles and the amount of joules (energy) to be delivered is important. The sketch below will provide a working guide.

Sometimes a temporary pacing is

event the patient has a pulmonary embolism that requires direct thrombolysis. Intra-aortic balloon pumping is also a very important technique that is used to temporarily assist the failing left ventricle.

MYOCARDIAL INFARCTION

Ischaemic heart disease though less common in Nigeria, current reports show increasing prevalence..[19 · 21] It has the following risk factors; hyperlipaedimias, smoking, hypertension, diabetes mellitus, insulin resistance etc. The following are marks of cardiovascular risk; total plasma homocysteine, fibrinogen, lipoprotein (Lp) a, fibrinolytic function as ascertained by tPA or PA1-1 antigens, CRP, ICAM and IL6. [22] Myocardial infarction is the most common cause of death in the UK and other developed countries.[23] It is one of the acute syndromes associated with atherosclerosis. It almost always occurs in patients with coronary atheroma. It is usually due to fissuring or rupture of the atheromatous plague leading to thrombose formation, spasm of the artery, the thrombosis leads to stasis. The myocardium turns pale 6hrs after the event and at 24 hours it becomes necrotic.

The presentation is usually with chest pain, similar to angina, but usually occurring at rest but lasting for hours. The pain might be severe that it might lead to fear of death ("angor animi"). There might be restlessness, nausea, and vomiting and sudden collapse. Diabetics, hypertensives and elderly patients might present with silent or painless myocardial infarction. It might go unnoticed or present with hypotension and arrhythmias. Physical findings might include hypotension, assumed precordial pulsation because of bulging infracted myocardium, forth heart sound, sinus tachycardia, raised venous pressure and pulmonary edema (crepitations). Muscle neurosis might lead to fever; lasting up to 7 days and a pericardial friction rub may develop.

Myocardial infarction could involve right or left ventricles; it could be involve different regions of the heart e.g. inferior, inferior lateral, anterior or anterior lateral, septal etc. The worst prognostically subtype is anterior and anterior lateral. Investigations will include.[17] The ST segment elevation and imaging findings are also used to determine the prognosis of the event..

1. Cardiac enzymes

a) CK-MB, which peaks within 24hrs and back to minimal in 48hrs. It can also be produced by skeletal muscle and brain. The myocardial bound (MB) is an iso-enzyme that is specific to the heart. Cardio version can also increase CK-MB.
b) Aspartate aminotransferase (AST), it peaks 24-48hurs and fall by 72hrs. It is an unspecific because it is also released by damaged blood cells, kidney, liver and lungs.
c) Lactic dehydrogenese. It peaks at 3-4 days and can last 10-14 days. It is also released by damaged skeletal muscle, red blood cells and liver. It has five iso-enzymes, the cardiac damage causes increase in LDH-1.

2. Electrocardiography

It is valuable in most cases; the acute event is associated with ST elevation convex upwards, few days (3days) later the Q wave appears (> 1mm broad and 2mm or more than 25% of the R amplitude). Q wave may also be caused by abnormalities like bundle branch block. It is usually present in full thickness myocardial infarction. The ST segment elevation results from ischaemia and injury. The Q wave does not develop in sub-endocardial infarction. The ECG tells which area of

the heart is involved.[24]

3. Echocardiography

It is an investigation when performed will show area of infarction and confirm ventricular septal defect and mitral regurgitation from papillary muscle or chordae rupture. Echocardiography can also help in the evaluation left and ventricular function.[11]

Other investigations include chest radiograph and blood biochemistry (Urea and Electrolytes, Blood sugar, lipid profile, uric acid and markers of cardiovascular risk where available.

Management

Patients are admitted into coronary care or intensive care or medical emergency unit immediately. ECG should be monitored continuously because patient develop might develop malignant arrhythmias and ECG also is used to monitor the revascularization therapy. Patient should have above investigations done including the ancillary one. An intravenous canula should be inserted for emergency intravenous medication. The routine medications include oxygen, nitroglycerin, aspirin, analgesics (like morphine or diamorphine), anxiolytics, anti-emetics and atropine when required. The anti-thrombotic therapy include; glycoprotein IIb/IIIb inhibitors, low molecular heparin and thrombolysis. Fibrinolytic therapy or revascularization is very important [17]. It is dependent on the time taken before the patient presented. They include IV drugs like streptokinase, urokinase and recombinant plasminogen activating factor (rTPA). TPA is usually administered to patients with anterior myocardial infarction, which is worse prognosis. It possible to salvage a less effective medical revascularization by doing rescue angioplasty (PTCA), and sometimes angioplasty is applied as the first step (primary angioplasty). The usual maintenance therapy is administered at the same time and patients are adequately educated on the condition.

Surgical intervention in the form of coronary artery by pass graft (CABG), which is employed when there is recurrent pain even after PTCA and thrombolytic therapy, had been employed. The management of mechanical defects like mitral regurgitation, ventricular septal defect, left ventricular free wall rupture and left ventricular aneurysm would require surgical treatment. Malignant arrhythmias are handled using lidocaine, bretylium. Procainamide and amiodarone.

PULMONARY EMBOLISM

Statistics in Nigeria is not readily available. The heart is affected by the haemodynamic changes in the lungs. The clinical symptoms of unexplained chest discomfort, shortness of breath and light-headedness should raise a suspicion of pulmonary embolism (PE). It accounts for hundreds of thousand hospitalizations in the United States [26]. Cardiologist must provide expertise treatment of Haemodynamically unstable patients with PE as well as those who have right ventricular failure with stable blood pressure and heart rate. The risk factors include genetic e.g. mutation in factor V gene, resistance to activated C, prothrombin gene mutation, mutation in protein C gene, protein C deficiency, anti-thrombin III deficiency, hyperhomocysteinemia and antiphospholipid antibodies.[27] The factors include surgery and immobilization/trauma, increasing age, cigarette smoking, obesity, systemic arterial hypertension, oral contraceptives, pregnancy, postpartum period, cancer (occult adenocarcinoma), cancer chemotherapy

spinal cord injury, stroke and CVP catheter..[28]

Diagnosis is based on symptoms, signs and investigations, but index of suspicion is paramount. The symptoms include shortness of breath and tachycardia and chest discomfort. The presence of severe dyspnoea, cyanosis and syncope indicates life-threatening event. The presence of acute cor-pulmonale (right ventricular failure) is established by the features of distended neck veins, S3 gallop, RV heave and tachycardia. Echocardiographic evidence of RV dilatation and hypokinesis or ECG features of acute cor-pulmonale defined by new $S_1Q_3T_3$, new incomplete right bundle branch block (RBBB) or right ventricular ischaemia. Differentia diagnosis includes pneumonia, cardiac failure, myocardial infarction and aortic dissection.

The clinical syndrome of pulmonary embolism is divided into massive, moderate to large, small to moderate, pulmonary infarction, paradoxical embolism and non-thrombotic embolism.[29] Massive embolism syndrome puts the patient at cardiogenic shock stage; the thrombose usually affects at least half of the pulmonary arterial system. And is present bilaterally. Systemic hypotension and dyspnoea are the major signs and subject will gain from pressor agent support. The patient with moderate to large does have RV hypokinesis on echocardiography but normal arterial systemic pressure. The lung scan will indicate more than 30% non-perfusion area and patients are at various stages of RV haemodynamic instability.

Diagnosis

It includes imaging and non-imaging methods. The imaging methods include chest x-ray usually abnormal in about 50% but its role in diagnosis is not well defined, venous ultrasonography (for DVT), nuclear venography (for DVT), contrast phlebography (for DVT), lung scan (for PE), chest computerized tomography (fro PE), magnetic resonance imaging (MRI) for PE, MRI pulmonary angiography for PE, LV and RV function. Echocardiography is increasingly ids becoming important in the imaging diagnosis of PE and risk stratification, it helps define RV overload. Standard pulmonary angiography is the gold standard method for the diagnosis of PE.[30]

The non-imaging methods include plasma D-dimer Elisa (most promising blood test for pulmonary embolism screening), arterial blood gases, and electrocardiograph and impedance plethysmography for DVT diagnosis. [26]

Management

Rapid risk stratification is of paramount importance. PE patients have a wide spectrum of clinical syndrome presentation. It might involve only prevention of further thrombosis by administration anti-coagulants, or insertion of filter at inferior vena cava. [26] Clot dissolution or removal is done in patients who have haemodynamic instability, which id done by thrombolysis or embolectomy. Anticoagulation is done by administering low molecular heparin. Other support measures include oxygen, adequate pain relief with non-steroidal anti-inflammatory drugs. The presence of hypoxia and toxic features in subjects call for mechanical ventilation. Inotropes (dobutamine) should be administered to patients that are hypotensive and poor oxygen perfusion. Inhale nitric oxide is given to patients with pulmonary hypertension. The most common side effect of heparin encountered acutely is bleeding.

In acute cases emotional support is very important with an educational component. The anatomic extent of the event will determine treatment and the

presence of the underlying cardiopulmonary disease and detection of RV dysfunction.[31]

Massive pulmonary embolism leads to severe haemodynamic compromise.[31] Thombolysis is an urgent treatment, drugs like streptokinase, which can be delivered directly using cardiac catheterization. Prevention of causes that can change or modified should be under taken.

OTHER CARDIAC EMERGENCIES

Other cardiac emergencies, which may not be seen in our practice, include acute atrial fibrillation following coronary artery by-pass graft (CABG), tear of the coronary artery during angiography, angioplasty, severe mitral regurgitation following percutaneous transluminal mitral valvuloplasty. Acute atrial fibrillation is managed using cardio version, intravenous amiodarone or sotalol a beta-blocker.[32]

Cardiac emergencies are part of our every day practice. It calls for training of more cardiologists, having a health policy set up that has a good reaction time to emergency situations. The basic equipments must be provided for any impact to be felt in this field

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