INTRODUCTION

Cardiothoracic surgery is associated with a significant risk of serious complications. So, cardiac surgical patients require intensive care management postoperatively. Many of these complications are likely caused in some part by the exaggerated systemic proinflammatory response to cardiopulmonary bypass (CPB). Systemic inflammation can occur with or without infection. Tissue injury, endotoxemia, and contact of the blood with foreign substances are likely major factors that help initiate this systemic inflammatory response. Organ dysfunction is attributed to a multitude of factors involving a cascade of inflammatory responses culminating in the inappropriate recruitment of leukocytes from the circulation ***(Warren et al., 2009).***

The recent developments in invasive cardiology, the improvement of surgical techniques, and the improved pharmacological treatments have contributed to the application of surgical treatment among elder patients with advanced cardiovascular disease and significant comorbidity. Preoperative atrial fibrillation significantly worsens the heart functional status, increasing the incidence of postoperative complications, such as delirium, stroke, and low cardiac output syndrome, which lead to negative healthcare patient outcomes, including higher mortality and prolonged ICU and in-hospital stay ***(Eltheni et al., 2012).***

The incidence of postoperative respiratory failure (PRF) is 9.1%. The highest incidence of RF is observed following combined valve/coronary artery bypass graft (14.8%) and aortic procedures (13.5%). Lung function and oxygenation are impaired in 20 to 90% of CPB cardiac surgery patients. The development of atelectasis in the postoperative period of cardiac surgery is approximately six times higher than that observed after abdominal surgery ***(Maillet et al., 2008)***.

Post-operative delirium is a common condition following cardiac surgery, can lead to adverse events and relatively longer hospital stays. Incidence rates of post-operative delirium in cardiac surgery patients range from 3% to 52% and it's prevalence range from 37.7% to 44.3%.The incidence of chronic pain after cardiac surgery varies from 21% to 56%. 33% to 66% experience chronic pain lasting more than 3 months, and 25% to 33% experience more than 1 year of chronic pain. Ineffective pain management may lead to serious pulmonary complications because of insufficient clearance of secretions, mucous plugging, and atelectasis ***(Alston and Pechon, 2005).***

Postoperative hemorrhage in cardiac surgery is a serious complication that independently is associated with significant postoperative adverse events. The incidence is estimated to be between 3% and 10% of all patients undergoing cardiac surgery with excessive postoperative hemorrhage, whereas 3% to 7% require a second operation to control hemorrhage ***(Rogers et al., 2009).***

AIM OF THE WORK

So, the aim of this essay is to review the postoperative complications that may occur in patients undergoing cardiothoracic surgery, and to review how to diagnose, prevent, and manage these complications.

Chapter One

CARDIOTHORACIC SURGERIES ADMITTED TO ICU

Intensive care has become a standard component of postoperative treatment for most cardio-thoracic patients. The complex physiological interactions resulting from underlying patient's pathological conditions and co-morbidities, as well as anesthetic and operative interventions. The post-operative care extends beyond the immediate post-extubation period to ensure adequate monitoring for potential complications. It's important to determine the intensive care unit (ICU) utilization by cardio-thoracic patients and to highlight the common indications for admission ***(Osinaike et al., 2012).***

I- Cardiac surgeries admitted to ICU:

A- Congenital cardiac surgeries:

Congenital heart disease is a problem with the heart's structure and function that is present at birth. Congenital heart disease causes more deaths in the first year of life than any other birth defects. Congenital heart disease is divided into two types: cyanotic and non-cyanotic. Heart defects can be part of genetic and chromosome syndromes. Drugs such as retinoic acid for acne, chemicals, alcohol, and infections (such as rubella) during pregnancy can contribute to some congenital

heart problems. Poorly controlled blood sugar in women who have diabetes during pregnancy has also been linked to a high rate of congenital heart defects ***(Webb et al., 2011***).

Cyanotic heart diseases include Tetralogy of Fallot, Eisenmenger's syndrome, Transposition of the great vessels, Truncus arteriosus, Ebstein's anomaly, Hypoplastic left heart, Pulmonary atresia, Total anomalous pulmonary venous return and Tricuspid atresia. Non-cyanotic heart diseases include Coarctation of the aorta, Patent ductus arteriosus, Aortic stenosis, Atrial septal defect, Atrioventricular canal (endocardial cushion defect), Pulmonic stenosis and Ventricular septal defect ***(Webb et al., 2011).***

1- Cyanotic heart diseases:

a- Tetralogy of Fallot (TOF) (fig 1):

Tetralogy of fallot is one of the most common congenital heart disorders (CHDs). Tetralogy of Fallot (TOF) represents approximately 10% of cases of CHD. This is classified as a cyanotic heart disorder, because it results in an inadequate flow of blood to the lungs for (right-to-left shunt). Patients with tetralogy of Fallot initially present with cyanosis shortly after birth. Asymptomatic infants need no special medical treatment. Surgery is the definitive treatment for the cyanotic patient with TOF.The primary role of medical therapy is in preparation for surgery ***(Robinson et al., 2011).***

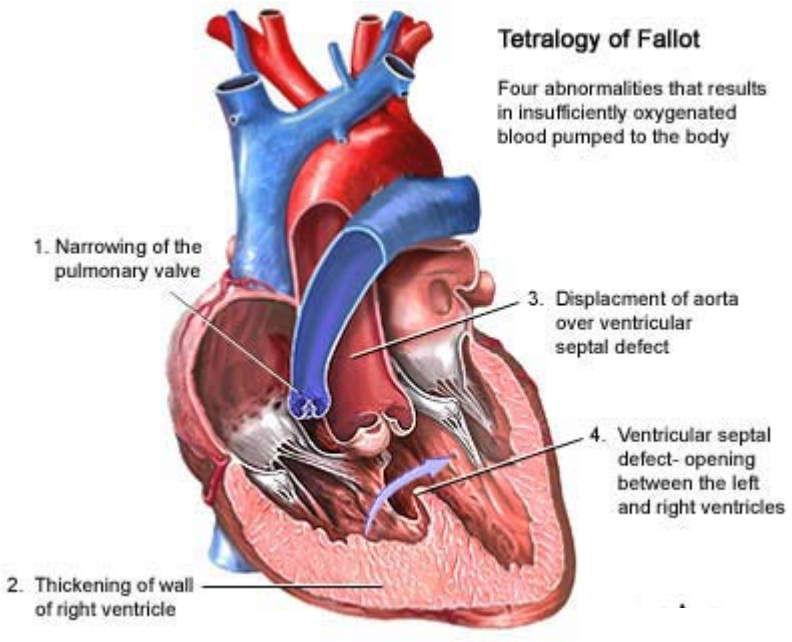

Fig. (1): Tetralogy of fallot ***(Parkar et al., 2010).***

Most infants have adequate saturations and undergo elective repair. Progressive hypoxemia and the occurrence of cyanotic spells are indications for early surgery. The timing of complete surgical repair is dependent on symptoms and any associated lesions (eg, multiple ventricular septal defects [VSD], pulmonary atresia). Currently, the trend is to perform a complete surgical procedure (often electively) before the age of 1 year and preferably by the age of 2 years. Most surgeons today recommend the primary corrective procedure, and current results are excellent. Infants with cyanosis are stabilized by administering prostaglandins (to maintain the ductus in an open state). The use of prostaglandins has significantly decreased the need to perform urgent surgery. Instead of performing systemic-to-pulmonary artery shunts on critically ill cyanotic-

hypoxic infants, surgeons now have the luxury of having extra time to assess the patient's anatomy and to perform the primary procedure on an elective basis ***(Park et al., 2010).***

b- Transposition of the great arteries (TGA)(fig 2):

The hallmark of transposition of the great arteries is ventriculoarterial discordance, in which the aorta arises from the morphologic right ventricle and the pulmonary artery arises from the morphologic left ventricle. Transposition of the great arteries is isolated in 90% of patients and is rarely associated with syndromes or extracardiac malformations. Long-term complications are secondary to prolonged cyanosis and include polycythemia and hyperviscosity syndrome. These patients may develop headache, decreased exercise tolerance, and stroke ***(Rao, 2009).***

Infants with transposition of the great arteries (TGA) are usually born at term, with cyanosis apparent within hours of birth. The clinical course and manifestations depend on the extent of intercirculatory mixing and the presence of associated anatomic lesions. Initial treatment consists of maintaining ductal patency with continuous intravenous (IV) prostaglandin E1 infusion to promote pulmonary blood flow, increase left atrial pressure, and promote left-to-right intercirculatory mixing at the atrial level. This is important in patients with severe left ventricular outflow tract stenosis or atresia ***(Wypij et al., 2003).***

Cardiac catheterization is indicated for a balloon atrial septostomy in severely hypoxemic patients with an inadequate atrial level communication and insufficient mixing. The balloon atrial septostomy is used to increase the atrial level shunt and to improve mixing. For the ill neonate, metabolic acidosis should be corrected with fluid replacement and bicarbonate administration. Mechanical ventilation is necessary if pulmonary edema develops in concert with severe hypoxemia. The patient requires surgical repair or palliation early in life. Surgical approach depends on the age of the patient at presentation, the presence of associated congenital cardiac lesions. Most full-term neonates with uncomplicated transposition of the great arteries can undergo an arterial switch procedure in one operation. Transposition of the great arteries with ventricular septal defect: The preferred operation is an arterial switch procedure with ventricular septal defect closure. If the ventricular septal defect is large and nonrestrictive and coronary artery anatomy makes an arterial switch operation inadvisable, a Rastelli-type intracardiac repair may be feasible. With the Rastelli-type procedure, waiting until the infant is older and larger may be preferred because of the need for a right ventricle–pulmonary artery conduit in the Rastelli operation ***(Khairy et al., 2013).***

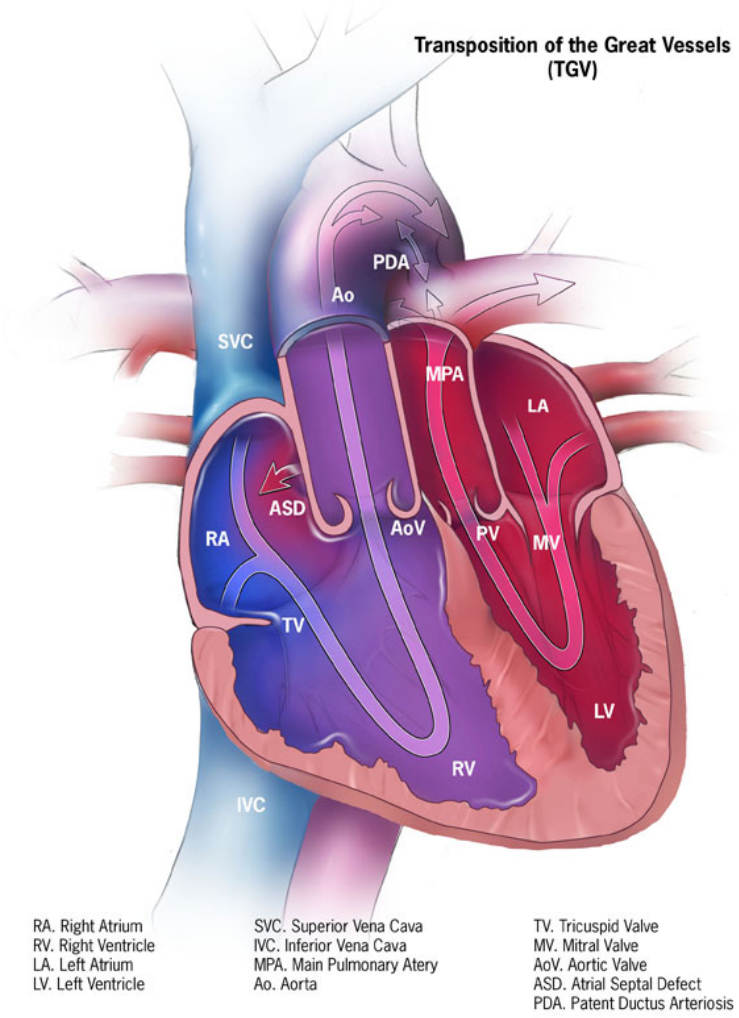

Fig. (2): Transposition of great arteries ***(Parkar et al., 2010).***

c- Truncus arteriosus (TA):

Is characterized by a single arterial trunk arising from the normally formed ventricles by means of a single semilunar valve. In addition, the pulmonary arteries originate from the common arterial trunk distal to the coronary arteries and proximal to the first brachiocephalic branch of the aortic arch.

The common trunk typically straddles a defect in the outlet portion of the interventricular septum. It originates completely from the right or left ventricle. In patients with a patent and normal caliber aortic arch, the ductus arteriosus is either absent or diminutive ***(Lund et al., 2011).***

Most neonates with truncus arteriosus display some evidence of congestive heart failure; they are treated with digitalis and diuretic medicines. Intravenous prostaglandin is administered in patients with truncus arteriosus upon presentation because the differential diagnosis includes numerous anomalies with duct-dependent systemic or pulmonary blood flow. It is beneficial only in patients with associated interruption of the aortic arch or aortic coarctation. Full-term and premature newborns with truncus arteriosus at increased risk for necrotizing enterocolitis, either preoperatively or postoperatively, and evaluation undertaken in any newborn exhibiting signs or symptoms of necrotizing enterocolitis. Truncus arteriosus requires operative repair. Symptoms typically develop in the early neonatal period, indicating that complete repair is required at this point. Surgical management consists of complete primary repair, with closure of the ventricular septal defect, committing the common arterial trunk to the left ventricle, and reconstruction of the right ventricular outflow tract. In patients with both branch pulmonary arteries arising from the common trunk, the standard method of right ventricular outflow tract reconstruction entails

removing the central pulmonary arteries from the common trunk en bloc and placing a valved conduit from the right ventricle proximally to the central pulmonary arteries distally ***(Russell et al., 2012).***

d- Ebstein's anomaly:

Ebstein anomaly is a congenital malformation of the heart that is characterized by apical displacement of the septal and posterior tricuspid valve leaflets, leading to atrialization of the right ventricle with a variable degree of malformation and displacement of the anterior leaflet. Ebstein anomaly accounts for 0.5% of congenital heart diseases. Mean age of presentation is in the middle teen age years. 5% of these patients survive beyond age 50 years. The oldest recorded patient lived to age 85 years. During fetal life: Ebstein anomaly is diagnosed incidentally by echocardiography and in neonatal life and infancy: Ebstein anomaly presents with cyanosis and/or severe heart failure; symptoms present in infancy improve as pulmonary vascular resistance decreases. During adult life: Ebstein anomaly presents with fatigue, exertional dyspnea, cyanosis, tricuspid regurgitation and/or right heart failure, and palpitations; arrhythmias are common ***(Attenhofer et al., 2012).***

Treatment includes medical therapy, radiofrequency ablation for arrhythmia, and surgical therapy. Surgical care includes correction of the underlying tricuspid valve and right ventricular abnormalities, correction of any associated

intracardiac defects, palliative procedures in early days of life as a bridge to more definitive surgical treatment later, and surgical treatment of associated arrhythmias. In patients ≥50 years of age with Ebstein anomaly, surgery is associated with good long-term survival and improved functional status. **Indications for surgery:** New York Heart Association (NYHA) class I-II heart failure with worsening symptoms or with a cardiothoracic ratio of 0.65 or greater, NYHA class III-IV heart failure, history of paradoxical embolism, significant cyanosis with arterial O2 saturation of 80% or less and/or polycythemia with hemoglobin of 16 g/dL or more and arrhythmias refractory to medical and radiofrequency ablation. Palliative procedures include creation of atrial septal defect, closure of tricuspid valve with plication of the right atrium, and maintenance of pulmonary blood flow through aortopulmonary shunt. In these patients, although early mortality is greater with tricuspid valve surgery, the late results are favorable and left ventricular function seems to improve postoperatively. Surgical treatments of arrhythmias include: ablation of the accessory pathways, Maze procedure for atrial arrhythmias and cardiac transplantation is appropriate in selected patients ***(Egidy et al., 2013).***

e- Hypoplastic left heart syndrome (HLHS) (fig 3):

Is a rare congenital heart defect in which the left ventricle of the heart is severely underdeveloped. In babies with HLHS, the aorta and left ventricle are underdeveloped before

birth, and the aortic and mitral valves are either too small to allow sufficient blood flow or are atretic altogether. As blood returns from the lungs to the left atrium, it must pass through an atrial septal defect to the right side of the heart. Surgical operations to assist with hypoplastic left heart are complex. Infants undergo either the staged reconstructive surgery (Norwood or Sano procedure within a few days of birth, Glenn or Hemi-Fontan procedure at 3 to 6 months of age, and the Fontan procedure at 3 to 5 years of age) or cardiac transplantation ***(Galantowicz et al.,2008).***

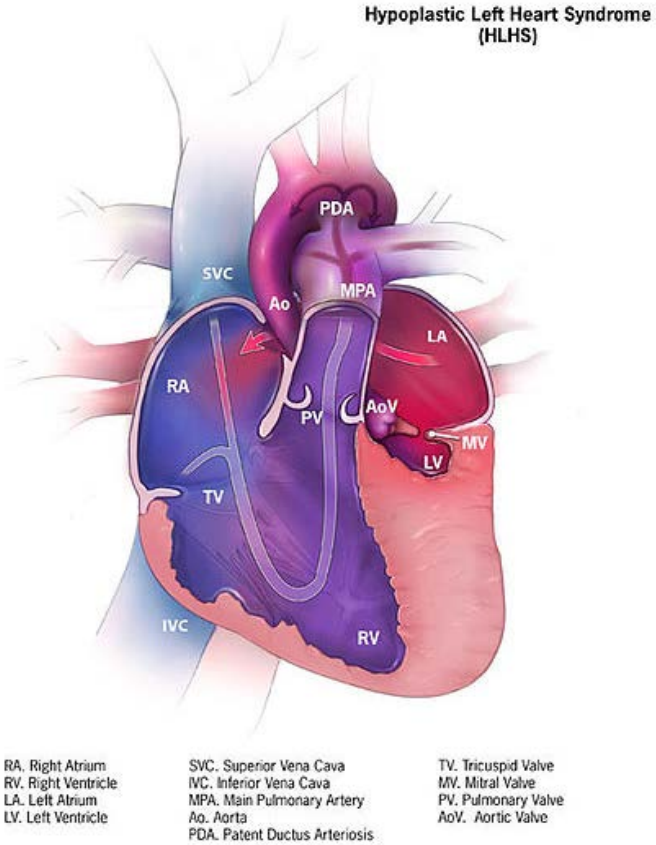

Fig. (3): HLHS ***(Parker et al., 2010).***

Other factors that increase an infant's risk include lower birth weight, additional congenital anomalies, a genetic syndrome or those with a highly restrictive atrial septum. For patients without these additional risk factors, 5 year survival approaches 80%. Further, 50% of those children who survived surgery in the early development of staged reconstruction have developmental delay or need special education; about 25% of these surgical survivors have severe disabilities. The three-stage procedure is a palliative procedure, as the child's circulation is made to work with only two of the heart's four chambers ***(Eghtesady et al., 2011).***

f- Total anomalous pulmonary venous connection (TAPVC):

It consists of an abnormality of blood flow in which all 4 pulmonary veins drain into systemic veins or the right atrium with or without pulmonary venous obstruction. Systemic and pulmonary venous blood mix in the right atrium. An atrial defect or foramen ovale is important in left ventricular output both in fetal and in newborn circulation. Surgical repair is used as treatment for total anomalous pulmonary venous connection whenever it best serves the individual patient. In a newborn with obstructive total anomalous pulmonary venous connection, stabilization often involves mechanical ventilation, correction of acidosis, inotropic support, and administration of prostaglandin E1 for patency of patent ductus arteriosus ***(Douglas et al., 2009).***

The goal of surgery is to redirect pulmonary vein flow entirely to the left atrium. In patients with a supracardiac or infracardiac connection, the common pulmonary vein is opened wide and connected side to side to the left atrium. The foramen ovale is closed, and the ascending or descending vein is ligated. In a cardiac connection (to right atrium or coronary sinus), the atrial septum is resected partially and a new septum is surgically created, directing pulmonary veins to the left atrium. A coronary sinus separately tunneled to the right atrium or left to drain with the pulmonary veins to the left atrium ***(Yap et al., 2009).***

2- Non-cyanotic:

a- Coarctation of the aorta (CoA)(fig 4):

It is a relatively common defect that accounts for 5-8% of all congenital heart defects. Coarctation of the aorta occurs as an isolated defect or in association with various other lesions, most commonly bicuspid aortic valve and ventricular septal defect (VSD). Associated problems contribute to death or morbidity include hypertension, intracranial hemorrhage, aortic rupture or early presentation of coarctation of the aorta. Significant hypertension or congestive heart failure (CHF) is an indication for intervention. Surgical relief of the aortic obstruction and catheter interventional techniques (balloon angioplasty and stents) are available alternatives. Various surgical techniques have been used to treat patients with coarctation of the aorta, namely, resection and end-to-end