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Nonimmune Hydrops Fetalis

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Introduction

Hydrops fetalis is an excessive accumulation of fluid within the fetal extracellular compartments and body cavities generally characterized by:
- placental enlargement
- ascites
- pericardial effusions
- pleural effusions
- perirenal effusions

Pathophysiology

The main pathophysiologic factor implicated in the development of NIHF is abnormal fluid movement between the plasma and tissues, leading to edema, ascites, and pleural and perirenal effusions. Pernatal mortality with this severe diagnosis is high, between 50–90% (Kayiran, 2015, p. 266). Four main theories have been suggested to explain the distribution of fluids that occur with hydrops fetalis:

1. an increase in hydrostatic capillary pressure (resulting from heart failure) or from obstruction of venous return
2. a reduction in plasma oncotic pressure (from decreased albumin production or increased albumin loss)
3. obstruction of lymphatic flow
4. damage to perihilar capillary light aggregates

(Nylander, 2014, p. 282.)

Cardiac Anomalies

Fetal echocardiography should be performed when the diagnosis is made of hydrops fetalis since fetal cardiac anomalies are among the highest cause of NIHF (Hariton, 2015, p. 235). The incidence of cardiac defects and rhythm abnormalities make up 13-25% of NIHF (Kayiran, 2013, p. 140). The cardiac anomaly is present due to the development of fetal heart failure; this can be a rhythm issue or a structural defect. Fetal supraventricular tachycardia is the most common tachycardia causing NIHF, accounting for 35-40% of all cases presenting with rhythm disturbances (Kayiran, 2013, p. 159). Fetal supraventricular tachycardia results in decreased ventricular filling time during diastole which further reduces cardiac output resulting in poor perfusion, inadequate tissue oxygenation, elevated central venous pressure, and elevated venous congestion (Randenberg, 2010, p. 207). The treatment of choice for fetal arrhythmias is the administration of medications to the mother such as digoxin or beta-blockers. Structural heart defects can affect both the right and left side of the heart as an isolated congenital heart defect usually born with hydrops fetalis. Right-sided heart defects such as Hypoplastic right heart syndrome, Tetralogy of Fallot, and Ebstein’s anomaly all result in right-sided outflow tract obstruction thus impeding flow from the heart. Cardiac malformations and left-sided heart defects such as Hypoplastic left heart syndrome, and aortic valve abnormalities can result in left-sided outflow tract obstruction resulting in severely decreased oxygen-rich blood flow to the body. All of these structural heart defects associated with NIHF are indicative of open-heart surgical intervention, most of them within several days of life. Poor prognosis of survival of NIHF is associated with structural cardiac defects and fetal arrhythmias diagnosed before 24 weeks gestation (Turpil, 2015, p. 357).

Clinical Management

Clinical management is indicative for the fetus and the infant in the setting of NIHF. Once a prenatal diagnosis has been made, focus is aimed at the coordination and collaboration with obstetrics/gynecology, social work, cardiology, genetics, and neonatology specialists. The family gathered is used to help with education and possible causes so the family can make an informed choice regarding treatment options. Supportive care measures and education are vital goals in the prenatal period. The parents must be kept informed of what to expect during labor and delivery and especially in the neonatal management in the delivery room; particularly subsequent tests and procedures.

Nursing Implications

Nursing implications are directed at monitoring neonatal respiratory measures. Preventing cold stress with the use of radiant warmers and a warmed room will help in decreasing added stress to an already compromised infant. Respiratory support with the help of noninvasive intubation, high peak inspiratory pressures and HFOV, umbilical arterial and venous catheter placement for management of hemodynamics and blood gas interpretation, helping in the procedure of bilateral thoracentesis for fluid removal in the pleural space, initiation of volume resuscitation with albumin or other colloids, and cardiovascular support with inotropes to increase cardiac output are all vital by nursing and medical staff to aid in the support and survival of NIHF. Neonatal management requires a skilled and coordinated resuscitative team by a well-equipped birthing hospital and neonatal intensive care units.

Conclusion

In conclusion, despite many advances made in the treatment, management, and diagnosis, NIHF still carries a high mortality rate. Further research is still needed to help with the management and treatment of NIHF to decrease intrauterine and perinatal mortality.

References