Refresher Course

# The Investigation and Management of Intrauterine Fetal Hydrops

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Hydrops fetalis is characterized by extensive accumulation of fluid in fetal tissues or body cavities. Its finding is non-specific with extensive different disorders. The mechanism is due to a gradual deterioration of fetal water balance which manifests in heterogenicity including wide variety degree of fetal ascites, anasarca, pleural effusion, pericardial effusion, placental oedema and polyhydramnios.[1,2] Non-immune hydrops was firstly reported by Potter[3] with less than 20% incidence of all cases. Late manifestation of several serious disorders may become non-immune fetal hydrops[3] with poor prognosis, but not fatal.[2] Most cases of hydrops persist until after delivery but some can resolve spontaneously. The improvement of fetal outcome is due to the specific antenatal diagnosis and treatment of primary cause in utero. Determination of the primary cause, including fetal karyotype, will also be important to make a decision for whether the aggressive management or a rational therapeutic strategy will proceed, i.e., caesarean section should be avoided if the fetus posses severe chromosomal abnormality or cardiac defect. The overall mortality rate of fetal hydrops is around 50-98%.[4]

Immune hydrops may be due to maternal alloimmunization, which is caused by Rh, Kell or other factors, but many conditions of fetus, placenta and mother may lead to the same syndrome. In the last two decades, the proportion of non-immune hydrops type has dramatically increased which is probably from increasing routine antenatal anomaly scanning and the effective prophylaxis of Rh (D) alloimmunization.[2,5] An incidence of non-immune hydrops is 1:2,500-3,700 births, but in the referral center it might be as high as 1:165 pregnancies;[2] whereas the last report of the non-immune hydrops ratio was 6.6:1.[6]

The most important concern is to understand the causes or fetal and maternal diseases for the improvement of treatment and prognosis and also the pregnancy outcome. From many reports , the aetiologies of fetal hydrops could be obtained around 50%-84%[6-8] and cardiovascular disease is the most common cause of hydrops fetalis in Caucasians, whereas alpha-thalassemia is the major cause in Asians.[6] Approximately one-third of non-immune fetal hydrops has chromosomal abnormalities but the correlation of hydrops and cystic hygromas; regardless the chromosomal or other fetal abnormalities, is the most common subgroup.[6] In non-immune hydrops group, antenatal scanning can detect around 2/3 of the fetal structural abnormalities as the aetiology; while maternal serum screening can detect 1.3% of the extrinsic aetiology and 22% remains idiopathic.[6] Nevertheless, the idiopathic rate may be overestimated because more than 50% of this group has no karyotypic results

The improvement in the accurate diagnosis of fetal hydrops will increase the number of good neonatal outcome, reduce the maternal complications

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of pregnancy such as polyhydramnios, pregnancy-induced hypertension, severe anaemia, antepartum and post partum haemorrhage, preterm labour, birth injuries, gestational diabetes and retained placenta; therefore, the maternal mortality and morbidity will be reduced.[8,9]

## Ultrasonographic Characteristics of Fetal Hydrops

Ultrasound scanning becomes very useful tool in diagnosis of prenatal fetal hydrops. Routine ultrasound scanning can detect 2/3 of non-immune fetal hydrops. Polyhydramnios is the most common abnormality for which the patient is refered for scanning around 1/3 of all cases.[7] The other reasons are large for gestational age, pregnancy-induced hypertension, antepartum haemorrhage, fetal tachycardia and decreased fetal movement.[4]

Ultrasonographic definition of fetal hydrops, regardless of the aetiology, is skin thickness greater than 5 mm[10] which is associated with different polyhydramnios or serous effusions and believed to be the late sign of genuine immune fetal hydrops. Both immune and non-immune hydrops have the same ultrasonographic features which are depended on the severity of the accumulation of the fluid. If the accumulation of the fluid is limited to only one cavity; which is known as "prehydropic sign",[11] the prognosis is better than those with hydrops.[2] Prehydropic sign will lead us to carefully follow-up the fetuses by serial ultrasound scanning because it may be an early sign of later developed fetal hydrops. The common ultrasonographic features of fluid accumulation are ascites, skin oedema, pleural effusion and pericardial effusion.[4]

An usual early sign of fetal hydrops is ascites.[12] It is characterized by an echolucent edge of vary sizes in the fetal abdomen which is sometimes similar to the normal echolucency rim of the fetal abdomen.[4] When the ascites is increased , the bowel loops will be compressed and surrounded by the fluid[13]. The whole liver shadow will also be seen easily. Pulmonary hypoplasia is found to be a common cause of neonatal death in fetal hydrops due to lung compression from large amount of ascitic fluid.[7,9,14]

Pleural effusion is difficult to be detected as well as pericardial effusion due to small amount of fluid inside the cavities. From De Vore's report[15], pericardial effusion is the earliest sign of immune fetal hydrops.

The most common sites for detection of skin oedema are the scalp and the thoracic area which are usually generalise oedema.[4] Abnormal placental thickening, greater than 4-6 cms[10,16], is defined as a sign of fetal hydrops. 75% of non-immune fetal hydrops are associated with polyhydramnios which is one of the major causes of prematurity in neonate. Contrary, oligohydramnios may be found in some cases of late stage fetal hydrops.[4]

The onset of fetal hydrops is vary due to variety of aetiologies. Fetal hydrops from chromosomal abnormalities trends to develop early in gestation. The early diagnosis of hydropic onset is very important for early prompt management to improve perinatal outcome and reduce perinatal mortality.

## Pathogenesis of Fetal Hydrops

The fetal body is largely composed of fluid in every compartment. Tissue diffusion pressures and the heart take responsibility for transposition of these fluids and blood circulation. The transposition of fluid is a complex function of many systems including amniotic fluid circulation, placental circulation and maternal circulation. Moreover, the fetus seems to be in a stage of water excess because of low colloidal osmotic pressure compared with the adult, a larger extracellular fluid than the intracellular fluid and a hypotonic urine.[2] Eventhough the water balance mechanisms seems to be abnormal in the fetal hydrops, three main circumstances affecting the pathogenesis have been involved.

1. The disruption of lymphatic drainage creates marked oedema due to fluid accumulation. The common causes are fetal congenital cardiac abnormalities and/or rhythm alteration. Moreover, a haemodynamic compromise, distension of the venous circulation, hepatomegaly, oedema and poor tissue perfusion could be occured.

2. The exceeding venous pressure over the lymphatic pressure leads to blockage of lymphatic drainage.

3. The impairment of liver synthesis of albumin or the plasma protein loss cause fetal hypoproteinaemia and then leads to decrease the intravascular osmotic pressure.[2]

## Investigation of Fetal Hydrops

Counselling the parents about the investigations, the management and the prognosis, including referral to a specialized center should be performed after ultrasonographic confirmation of the diagnosis of hydrops fetalis. Fetal daily movement recording, fetal heart rate monitoring and biophysical profiles can be very helpful for fetal surveillance; but sometimes it is too late if abnormal findings are detected in advanced disease. Therefore, the early accurate diagnosis and management of fetal hydrops are the best way instead of awaiting for the signs of fetal deterioration.[2] Any intervention must be carefully considered because it may both correct and worsen the other conditions.

Getting more informations from the patient should be performed in the first step of investigation. A genetic diseases or familial congenital abnormalities, consanguineous marriage, ethnic background and maternal infections or exposure are the important datas to indicate the precise investigations. Maternal blood should be taken for complete blood count; including differential counts and blood cell indices, Kleihauer-Betke stain for fetal blood cell in maternal circulation, ABO type and Rh antigen status, indirect Coombs test, TORCH titres, serologic testing for syphilis, human Parvovirus B19, a G-6PD deficiency screening, and haemoglobin electrophoresis. Level III ultrasonographic scanning of structural and functional abnormalities of fetus, placenta, amniotic fluid and color Doppler scanning including fetal echocardiography are beneficial for identification of the causes.[2] High velocity Doppler waveforms of umbilical vein indicates anaemic fetuses[17] and some with congenital abnormalities.[18]

Fetal karyotype should be performed by chorionic villous sampling, amniocentesis or fetal blood sampling due to high incidence of chromosomal abnormalities in non-immune hydrops. Moreover, these techniques can detect single gene disorder at the same time. A quick karyotypic result and blood disorder testing are the advantage of fetal blood sampling; which could be detected for anaemia, thrombocytopenias and haemoglobinopathies.[19] Serologic studies for the infectious agents should be promptly investigated.[20]

Biochemical and cytologic blood analyses will reassure the function of specific organs;[19] whereas fetal blood gas and metabolic status analyses will reassure the placental function and fetal well being.[21] The quality control of blood sampling is very important in order to avoid contamination of maternal blood cell; which can lead to a mistaken conclusion of congenital infection due to presence of IgM in the sample. Amniotic contamination can cause a misleading of a fetal anaemia and the coagulation activation which results in mistaken thrombocytopenia.[22] Intrauterine manometry can be used for the study of pathophysiology of the underlying circumstance and management.[11,23]

## Management, Findings and Prognosis of Fetal Hydrops

### *Immune Fetal Hydrops : Maternal Alloimmunization*

Haemolytic disease of the fetus and newborn was firstly described in 1609,[24] when the midwife observed the birth of twins, one had fetal hydrops while the other developed severe jaundice and both died. Afterward, Diamond[25] reported the relationship among fetal hydrops, icterus gravis and anaemia of the newborn and were found to be the same disorder (a haemolytic mechanism) but with different presentations and erythroblastosis (circulating erythroblasts) was detected in all conditions. After the maternal-fetal antibody passage theory was confirmed, Darrow[26] hypothesised that the cause of haemolysis was maternal anti-fetal haemoglobin (antibody) and the specific antigen (fetal haemoglobin) were misleading. Lansteiner and Weiner[27] recognised the real antibody from the agglutination test of rhesus monkey red cell antisera in the rabbits and guinea pigs combined with Caucasian blood samples. Since then it was characterized that transfusion reactions occured in Rh-negative patients given Rh-positive blood.[28] The common cause of haemolytic disease of the fetus and newborn was the appearance of Rh antibodies in the rhesus-negative mother.

#### *Pathogenesis and Findings of Immune Fetal Hydrops*

Blood production starts from the 3rd week of gestation, and Rh antigen on the membrane of the red blood cell is found by the 6th week. Erythropoie-

sis in the liver and spleen starts from the 8th to the 10th week; and then switches to the bone marrow by the 6th month, and is specified in the bone marrow at birth. Both IgM and IgG are produced in the maternal circulation against fetal red blood cells. IgM does not cross the placenta, whereas IgG can pass into the fetal circulation to attach to the surface of the fetal red blood cell. The main pathogenesis of haemolytic disease of the fetus and newborn is caused by the antibody-coated fetal red blood cells are destroyed by the fetal reticuloendothelial system. The fetal extramedullary erythropiesis in liver, spleen, kidney, adrenal and intestinal mucosa are then increased in functions to compensate the erythrocyte destruction. Progressive anaemia, erythroblastosis, hepatosplenomegaly, hypoxaemia, acidosis, liver failure and heart failure are presenting signs of decompensation for the haemolysis.[2]

In haemolytic disease, the fetus usually has anaemia. In the 2nd trimester, if fetal haematocrit is less than 15% or haemoglobin is less than 4 g/100 ml, hydrops is always presented.[2] Its manifestations are pericardial, pleural and peritoneal fluid in the early onset and hepatosplenomegaly, cardiomegaly, polyhydramnios and hyperplacentosis in the late onset. The placental enlargement, caused by oedema, can lead to the increased villous surface area and the marked oedematous villi; together with the hyperdynamic stage of fetal circulation, may be the cause of facilitated exchange crossing the barrier of placenta, and the hyperglycaemic stage with reducing of gas and substance exchange may occur later.[21]

In both haemolytic disease and alpha-thalassaemia, pancreatic hyperplasia; related to hyperinsulinaemia, is frequently found and inversely associated with lower haematocrit. However, whether the pancreatic hyperplasia and hyperinsulinaemia are the result of the hyperglycaemia state or haemolysis process is not definitely indicated.

***Management of Immune Fetal Hydrops***

Incidence of neonatal hydrops is now decreasing because of the correction of the fetal anaemia in utero. Poor prognosis always occurs in fetal hydrops without the prediction of the onset. However; the accurate predictors of the fetal anaemia are good obstetric history and maternal antibody level. Invasive investigations should be peformed if maternal anti-D level is more than or equal to 1:16.

Since 1961, spectrophotometry in optical density at 450 nm (delta OD-450) to detect bilirubin in the amniotic fluid has highly predictive value of 96.2% in severely anaemic fetus at or greater than 25 weeks of gestation,[2] and false negative rate of 26% in the fetus lower than 25 weeks of gestation.[29] If maternal antibody is rising before 25 weeks of gestation or amniotic fluid presents a delta OD 450 in the upper zone II or III of Liley's chart after 25 weeks of gestation, they are the indications for performing fetal blood sampling from the placental insertion of the umbilical cord[30] or the intrahepatic portion of the umbilical vein.[31] Determination of precise diagnosis and prognosis will be obtained from fetal blood group, haemoglobin level, blood gases and metabolites analysis.

Expectant management by serial ultrasound scannings, fetal movement observation, fetal heart rate testing and biophysical profiles could be performed if no hydrops signs is detected, fetal haematocrit is greater than 30%, maternal antibody titres is stable and serial amniotic fluid delta OD-450 is normal. In the other hand, severe anaemic fetus; haematocrit is lower than 30%, is indicated for intrauterine fetal transfusion to correct fetal anaemia in utero. The transfusion can be repeated until the fetus can survive after delivery without hydrops.[2] To determine the complications of over transfusion, observation of the fetal heart rate from ultrasonography, fetal blood gases and metabolites analysis, intravascular or intraperitoneal pressures alterations are useful in surveillance.

Reducing the haemolysis by using high dose immunoglobulin G (IgG) is still questionable.[2] Its mechanism is to attach the IgG to IgG receptors of the placenta and fetal reticuloendothelial systems. Haemolytic disease of the fetus and newborn has overall survival rate of 96% and 100% if intrauterine blood transfusion(s) was/were performed in the presence and abscence of fetal hydrops respectively.[32] Basically, 100-300 μg of Rh Ig could induce isoimmunization in non-immunized mother dramatically after labour which is not depended on fetal blood group, however, it is useless if the partner also has Rh-negative. Standard prophylactic dose of Rh IgG for every 25 ml of fetal blood or fraction should be administered to the Rh-negative mother who produced extensive transplacental bleeding of the

Rh-positive fetal red blood cells, and 50 μg of Rh Ig should be given after termination of abortion or ectopic pregnancy.[24]

## *Non-Immune Fetal Hydrops*

There are many aetiologies of non-immune fetal hydrops which are not easy to classify; for instance, chromosomal abnormalities are associated with a lot of cardiac defects, whereas fetal anaemia and some syndromes may be the results of viral infection. In order to decide for prompt investigation, management and prognosis, the possible causes may be beneficial to categorise.

### *Intrinsic Causes of Non-immune Fetal Hydrops (Fetal Origin)*

#### Chromosome Abnormalities and Other Genetic Disorders

Down's syndrome, trisomies 13, 18, Turner syndrome or Triploidy, as well as genetic disorders, including dwarfism, arthrogryposis, osteogenesis imperfecta, chrondrodystrophy, asphyxiating thoracic dystrophy, Noonan syndrome, familial cirrhosis and congenital myotonic dystrophy[2] may be the aetiologies of non-immune fetal hydrops whereas some metabolic disorders such as Gaucher disease, Tay-Saches disease and GM1-gangliosidosis can also be the aetiologies of fetal hydrops. An accurate diagnosis can be identified by history taking, postpartum examination, X-rays, blood for chromosome and DNA analysis and pathological study. The definite disorder of organ dysfunction in each cause is the pathogenesis of hydrops. The management may be from expectant to termination of pregnancy; which depends on the gestational age, cause, and parents's decision. However; the overall prognosis is poor, regardless of hydrops.

#### Non-immune Fetal Anaemia

Homozygous alpha-thalassaemia, an autosomal recessive disorder, is one of the most common cause of fetal hydrops in Asia with a recurrent rate of 25% in the subsequent pregnancy.[33] Affected fetus with Hb Barts is invariably hydropic at birth. Bart's haemoglobin (γ 4) posses high affinity for oxygen with no response for pH changes; but 10-20% of these hydropic fetuses poss Hb Barts and have normal oxygen dissociation curves with trend to survive until term.[2] Ultrasonographic features of affected fetus provide ascites, hepatosplenomegaly, pleural and pericardial effusion, oedematous placenta, subcutaneous oedema, cardiomegaly and oligohydramnios without any structural abnormalities.[33] Prenatal diagnosis can be performed by DNA analysis using either chorionic villous sampling in the first trimester or amniocentesis; or fetal blood sampling along with haemoglobin electrophoresis in the second trimester. An accurate diagnosis should be performed for counselling, risk assessment and planning for prenatal diagnosis in subsequent pregnancies; eventhough there is no definite therapy.[4]

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is one of other rare causes of non-immune hydrops. It is inheritated as an X-linked recessive disorder which is activated by sulfa drugs, aspirin and feva beans.[4] Counselling the patient to avoid consuming some drugs and food during pregnancy is very important.

#### Fetal Structural Abnormalities related with Non-immune Fetal Hydrops

- **Cystic Hygroma**

Cystic hygroma, a benign developmental lymphangioma, arises from lymphatic drainage blockage which leads to the local or general distension of this system. Its ultrasonographic feature is septated cysts within the soft tissue; usually involving the neck, and are related with 50-80% of chromosomal abnormalities, mostly is monosomy X. Regardless chromosomal abnormalities, the overall prognosis is poor.[34] Intrauterine drainage of cystic hygromas in normally chromosomal fetus may be helpful.[2]

- **Cardiovascular abnormalities**

40% of non-immune fetal hydrops posses cardiac abnormalities.[34] According to high relationship between chromosomal abnormalities and cardiac defect, karyotype of the fetus should be performed. The pathogenesis of fetal hydrops could be described from severe cardiac abnormalities; i.e., large atrioventricular defects, premature closure of foramen ovale and ductus arteriosus, and myocarditis.[2] Further investigations are beneficial if cardiac dysfunction are detected.. The presence of pericardial effusion which is larger than 2 mms is indicated of fetal heart failure. The management is depended on the severity of cardiac abnormalities; however, the overall survival rate is only 20%. Cardiac abnormalities have low associations with chromosomal or other lethal congenital abnormalities.[35] In the abscence

of the other lethal congenital abnormalities, intrauterine medical treatment with some medications such as digoxin, verapamil, propanolol, etc. may be helpful in the fetuses with cardiac arrhythmia.[12,34]

Serial ultrasound and umbilical artery velocity waveform assessments, together with fetal surveillance and good maternal conditional monitoring should be performed. An alternative intrauterine fetal therapy by direct fetal administration of drugs; under ultrasound guidance, is debated due to the poor placental transfer of drugs.[2]

- **Lung Abnormalities**

Pleural effusion can be found to be primary or secondary to fetal hydrops. It is sometimes associated with chromosomal abnormalities, congenital defects and tumor. Ultrasonographic features present anechoic fluid with the compressed lung; and polyhydramnios is also commonly found. The prognosis for spontaneous resolution of isolated hydrothorax is good; but those with generalised fetal hydrops or other congenital abnormalities are poor.[11,36] The most common cause of neonatal death is lung compression from the effusion. Pluero-amniotic shunts seem to improve lung development and give rise to the good results.[11,12,36]

- **Gastrointestinal Tract Abnormalities**

Isolated ascites may cause from jejunal atresia, midgut volvulus, intestinal malrotation and meconium peritonitis; which result in mesenteric vascular, lymphatic constriction later. The overall prognosis is fair with survival rate of 50%. Conservative management should be carried on until delivery.[2]

- **Kidney and Urinary Tract Abnormalities**

Few of renal causes are related with fetal hydrops. A ruptured bladder secondary to a urinary tract obstruction may characterized as hydrops. A moderate hydronephrosis is always related with fetal hydrops. The pathogenesis of fetal hydrops and polyhydramnios is still be the issue for discussion.[2]

- **Other Fetal Tumors**

Teratomas; common sites at the presacral and precocygeal area, is classified as true tumors composed of all three embryonic germ layers. Its incidence is 1/40,000 births and commonly occur in female.[2] 18% are associated with other congenital anomalies while karyotype is normal. The common ultrasonographic features are polyhydramnios and placentomegaly which are found in 30-35% of cases.[2] The pathogenesis of fetal hydrops is the high cardiac output failure, which is secondary to anaemia from neoplastic vascular steal and ateriovenous shunting throughout the tumor.[2] The prognosis depends on tumor size, tumor growth rate and gestational age. The overall prognosis is poor due to frequent fetal death in hydrops cases. The conservative management should be carried on until delivery and the tumor should be removed after birth as soon as possible, regardless of the size and type of tumors.[37] Intrauterine fetal therapy by ultrasound guidance. Laser or electro-coagulation of the neoplastic vascular supply or the arterio-venus shunting also play a role in the treatment.

Congenital neuroblastoma can produce bone marrow invasion and anaemia, compression of the vena cava and then develops hydrops as another cause.

- **Placenta and Umbilical Cord Abnormalities**

Acute feto-maternal haemorrhage may cause death to the fetus but hydrops may be resulted from the chronic case. Careful examination of the placenta should be performed. Non-immune hydrops may be resulted from chorioangioma, choriocarcinomas, angiomyxoma of the umbilical cord, aneurysm of the umbilical artery and cord venous thrombosis. The pathogenesis of fetal hydrops is caused by tumor-producing fetomaternal haemorrhage which leads to the increasing pressure in the fetal vascular compartment of the placenta before developing of hydrops or fetal death.[2] If it is associated with other fetal abnormalities, the prognosis is poor.

- **Twin to Twin Transfusion Syndrome (TTTS)**

Fetal structural abnormalities and vascular anastomosis between placenta can lead to hydrops in the twins pregnancy. 85% of monochorionic placentas posses vascular anastomosis; although only 5-17.5% posses TTTS.[38,39] Ultrasonographic feature presents oligohydramnios in the sac of the donor twin which is correlated with polyhydramnios

in the sac of the recipient twin. Growth discordance is also found by ultrasonography. Prenatal diagnosis, managemnet and planning of delivery may improve the outcome of pregnancies; however, the mortality rate is still high (40-70%).[39]

Respiratory difficulty with discomfort of the mother, premature ruptured of membranes, preterm labour and delivery of compromised premature infants are secondary to severe disease. Bed rest with tocolytic agents are not proved to improve the outcome. The active management with serial amniocentesis for the treatment of polyhydramnios, digoxin and selective fetocide to improve the outcome need more studies before becoming standard practice. The studies of in utero laser coagulation of anastomotic vessels through fetoscopy are further needed.

An acardiac twin is occured around 1/ 30,000 of all cases of twins with simply detection by ultrasound. Both fetal circulations are provided by the normal twin, whom cardiac hypertrophy and hydrops might develop. The management depends on gestational age at the time of diagnosis. Elective fetocide is also a choice of management to improve the outcome of pregnancy.

### *Extrinsic Causes of Non-immune Fetal Hydrops*

#### Maternal Infection

Some microorganisms can cause infection in both mother and fetus which results in fetal hydrops, IUGR or late intrauterine and neonatal death. Non-immune hydrops can be associated with maternal infections, such as toxoplasmosis, CMV, parvovirus, syphilis, rubella, leptospirosis, Chagas disease and congenital hepatitis. It is characterized by ascites, hepatomegaly, splenomegaly, cardiomegaly, pericardial effusions and generalised hydrops.[2] Detection of specific IgM in fetal blood samples can be used for prenatal diagnosis.

Human Parvovirus B19 has been associated with maternal infection, fetal hydrops and death.[12] Parvovirus is a single-stranded DNA virus which replicates in proliferative cells. It is embryocidal, but not a teratogen; and can transmit transplacentally from mother to the fetus around 10% of cases. Transient aplastic crisis and anaemia are secondary to infected red cell precursors of both mother and fetus; and can be developed hydrops and death in 2.5% of cases.[40] The risk of hydrops is greater during the second trimester of pregnancy due to increasing in fetal red cell mass. The fetal outcome is poor if fetal hydrops develops together with increasing levels of maternal serum alpha-fetoprotein. Cordocentesis can give the accurate prenatal diagnosis.[2]

The intrauterine management by intravascular fetal blood transfusion under ultrasound guidance can correct fetal anaemia and improve the fetal outcomes.[34,41] If there are no signs of hydrops, the overall prognosis after intrauterine therapy is quite good.

#### Other Maternal Complications associated with Fetal Hydrops

Uncontrolled diabetes may lead to fetal macrosomia, oedematous, fetal malformations and hydrops. Maternal nephritis and anaemia may associated with non-immune fetal hydrops.

#### Idiopathic Fetal Hydrops

Although there are many advance techniques of prenatal diagnostic methods, the incidence of "idiopathic" of non-immune fetal hydrops is still high around 12-25% of cases.[2] All cases of idiopathic hydrops show the same pathological changes such as marked generalised tissue oedema; pleural and peritoneal effusions and hepatosplenomegaly; while none of the cases presents congenital abnormalities or signs of infection. The mortality rate is as high as 100% when fetal hydrops is associated with polyhydramnios, pre-eclampsia and prematurity.[2] The benefit of intrauterine medical treatment with digoxin, furosemide and albumin are needed to be proved. However, the definite causes of fetal hydrops should be obtained for accurate diagnosis and management.

## CONCLUSION

Fetal hydrops is a common term definding the extensive accumulation of fluid in fetal tissue or body cavities; which is described in terms of involved site such as ascites, pericardial effusion, pleural effusion or polyhydramnios. Maternal alloimmunization seems to be the most common cause; but after increasing utilization of Rh (D) immunoprophy-

laxis, the non-immune hydrops is found to be common instead. Accurate diagnosis and prenatal management are very important to improve the prognosis and outcome of pregnancy. The main concept in approaching the fetal hydrops is to promptly investigate, diagnosis and manage the definite cause of fetal hydrops as soon as possible in order to have a good prognosis and outcome; although that almost depends on the primary cause. Many issues of fetal hydrops including aetiologies, investigations, management, pathogenesis and ultrasonographic findings are still discussed and debated. The most important tool for investigation, diagnosis and treatment is ultrasound. The immune hydrops posses excellent prognosis and fetal outcome due to Rh-immunization prophylaxis policy and prompt correction of anaemic fetus.

Although the prognosis of the non-immune hydrops is not good but it is important to attempt finding the aetiologies and associated fetal abnormalities for accurate management and improving the prognosis and the pregnancy outcome. In Caucasians, cardiovascular abnormalities and cardiac arrhythmias are the common aetiologies of non-immune hydrops and the prognosis is not good if it is associated with other chromosomal or structural abnormalities. In Asians, homozygous alpha-thalassaemia is the common cause of the fetal hydrops with high maternal mortality and morbidity rate. Counselling the recurrent risks in subsequent pregnancies should be proceeded. Parvovirus B19 is the most common microorganism which causes maternal infection in the non-immune hydrops. Fetal blood testing for specific IgM should be obtained to confirm the diagnosis. The management by intrauterine blood transfusion provides the excellent outcome. However, the prognosis of non-immune fetal hydrops will be improved soon after the accurate diagnosis of aetiology.

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