

## Ballantyne Syndrome: Maternal Mirror of Fetal Hydrops, Diagnostic Challenges, And Contemporary Management

Viridiana Romo Cepeda<sup>1</sup>, Dania Yedith López Mora<sup>2</sup>, Melannie Paola Benavides Mier<sup>3</sup>

<sup>1,2,3</sup>Issste Hospital Regional De Monterrey

### ABSTRACT

Ballantyne syndrome, also known as mirror syndrome or triple edema, is a rare obstetric disorder characterized by the association of fetal hydrops, placentomegaly, and maternal edema. The syndrome reflects a maternal systemic response to severe fetal and placental hydrops and may closely resemble preeclampsia. Maternal manifestations include generalized edema, rapid weight gain, hypertension, proteinuria, dyspnea, pulmonary edema, and laboratory abnormalities. A key distinguishing feature from classic preeclampsia is maternal hemodilution rather than hemoconcentration. Ballantyne syndrome may occur with immune or non-immune fetal hydrops, including rhesus isoimmunization, parvovirus B19 infection, twin-to-twin transfusion syndrome, fetal structural cardiac disease, placental tumors, fetal tumors, chromosomal abnormalities, and vascular malformations. Perinatal prognosis is poor, with high rates of intrauterine fetal death and neonatal mortality. Management depends on identifying whether the underlying cause of fetal hydrops is reversible. When fetal therapy is possible, correction of the fetal condition may lead to maternal improvement and continuation of pregnancy. When hydrops is irreversible or maternal status deteriorates, delivery or pregnancy termination is often required. This review summarizes the definition, pathophysiology, etiologies, diagnostic approach, differentiation from preeclampsia, maternal and fetal outcomes, and management principles of Ballantyne syndrome.

**KEYWORDS:** Ballantyne syndrome; mirror syndrome; triple edema; fetal hydrops; placentomegaly; maternal edema; preeclampsia; pregnancy complications.

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### INTRODUCTION

Ballantyne syndrome is a rare but severe pregnancy complication in which maternal disease mirrors fetal and placental hydrops. It is also known as mirror syndrome because the maternal clinical picture reflects the edematous state of the fetus and placenta. The classic triad consists of fetal hydrops, placentomegaly, and maternal generalized edema [1–3].

The syndrome is clinically important because it can mimic severe preeclampsia. Patients may present with edema, hypertension, proteinuria, dyspnea, pulmonary edema, and rapid weight gain. However, unlike typical preeclampsia, Ballantyne syndrome is usually associated with maternal hemodilution, reflecting plasma volume expansion rather than hemoconcentration [4].

The disorder is not a single-cause disease. Rather, it is a final maternal syndrome triggered by severe fetal-placental

pathology. Any condition capable of producing significant fetal hydrops may potentially lead to Ballantyne syndrome. The central diagnostic and therapeutic task is therefore to identify the cause of fetal hydrops and determine whether it is reversible.

### METHODS

This narrative review was developed from the uploaded literature synthesis on Ballantyne syndrome. The synthesis included systematic reviews, case reports, clinical reviews, and recent literature addressing fetal hydrops-associated maternal disease, diagnostic overlap with preeclampsia, maternal outcomes, fetal outcomes, and management strategies.

References are numbered according to their first appearance in the manuscript.

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## Definition and Terminology

Ballantyne syndrome is defined by the coexistence of:

1. **Fetal hydrops**
2. **Placentomegaly**
3. **Maternal edema**

Because all three compartments become edematous, the condition has also been called **triple edema syndrome**. The term **mirror syndrome** emphasizes that maternal findings mirror the fetal and placental condition [1,2].

Fetal hydrops is generally defined as abnormal fluid accumulation in at least two fetal compartments, such as ascites, pleural effusion, pericardial effusion, skin edema, or placental edema. In Ballantyne syndrome, placental edema is central because it appears to play an important role in the maternal systemic response.

## Pathophysiology

The pathophysiology of Ballantyne syndrome is incompletely understood. The most accepted concept is that severe fetal and placental hydrops leads to placental dysfunction, release of antiangiogenic or inflammatory mediators, maternal endothelial activation, and abnormal maternal fluid balance.

Proposed mechanisms include:

- Severe placental edema
- Trophoblastic dysfunction
- Maternal endothelial activation
- Increased vascular permeability
- Plasma volume expansion
- Hemodilution
- Renal involvement with proteinuria
- Possible imbalance of angiogenic and antiangiogenic factors

The maternal phenotype may overlap with preeclampsia because both conditions involve abnormal placental signaling and maternal endothelial dysfunction. However, Ballantyne syndrome is more directly linked to fetal hydrops and placental enlargement.

## Etiology

Ballantyne syndrome may occur secondary to immune or non-immune fetal hydrops. Reported causes include a wide range of fetal, placental, infectious, hematologic, and vascular conditions.

### Immune Causes

Historically, rhesus isoimmunization was a classic cause of fetal hydrops and Ballantyne syndrome. Severe fetal anemia leads to high-output cardiac failure, hydrops, placental edema, and maternal mirror manifestations [5].

### Infectious Causes

Parvovirus B19 is an important infectious cause because it can produce severe fetal anemia, myocarditis, hydrops, and placental edema. Successful management may be possible when fetal anemia is diagnosed and treated appropriately [6]. Other infections may also cause fetal hydrops and should be considered depending on maternal history, serology, and fetal findings.

## Twin-to-Twin Transfusion Syndrome

Twin-to-twin transfusion syndrome may cause hydrops in one or both fetuses, especially in monochorionic pregnancies. Severe placental and fetal edema may trigger maternal mirror syndrome. Management depends on gestational age, severity, and feasibility of fetoscopic laser therapy or delivery.

## Structural Cardiac Disease and Arrhythmia

Fetal cardiac malformations, severe cardiomyopathy, or sustained tachyarrhythmias can cause hydrops through cardiac failure. If the cause is treatable, such as selected arrhythmias, fetal therapy may reverse hydrops and improve maternal status.

## Placental and Fetal Tumors

Large placental chorioangiomas and fetal tumors may produce arteriovenous shunting, high-output cardiac failure, and hydrops. Treatment depends on lesion type, gestational age, fetal compromise, and procedural feasibility.

## Chromosomal and Genetic Disorders

Chromosomal abnormalities, including Down syndrome, have been reported in association with Ballantyne syndrome when fetal hydrops is present [7].

## Vascular Malformations

Rare fetal vascular malformations, such as vein of Galen aneurysm, may cause high-output cardiac failure and fetal hydrops, leading to maternal mirror syndrome [8].

## Clinical Presentation

Maternal symptoms often develop progressively and may be mistaken for preeclampsia or heart failure. Common features include:

- Generalized edema
- Rapid maternal weight gain
- Facial and peripheral edema
- Hypertension
- Proteinuria
- Dyspnea
- Pulmonary edema
- Oliguria
- Headache or malaise
- Anemia or hemodilution
- Hypoalbuminemia
- Elevated uric acid or liver enzymes in some cases

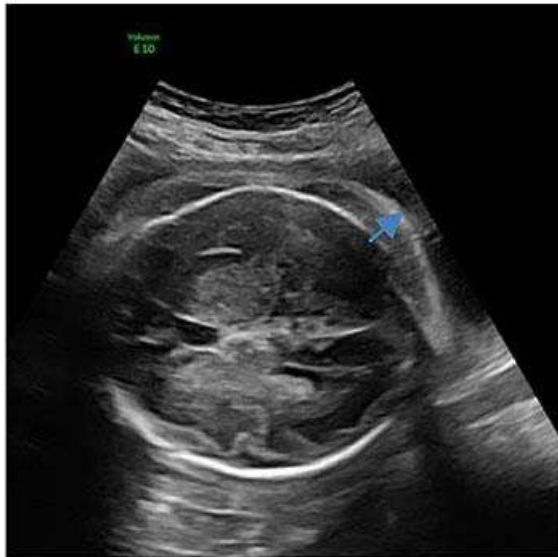
The uploaded synthesis reports edema in approximately 80–100% of cases, hypertension in 57–78%, and proteinuria in 20–56%, based on summarized literature data.

Ballantyne\_syndrome.pdf

Fetal findings may include:

- Skin edema
- Ascites
- Pleural effusion
- Pericardial effusion
- Cardiomegaly
- Abnormal Doppler findings
- Placentomegaly
- Polyhydramnios
- Evidence of anemia or cardiac failure

A. Scalp edema



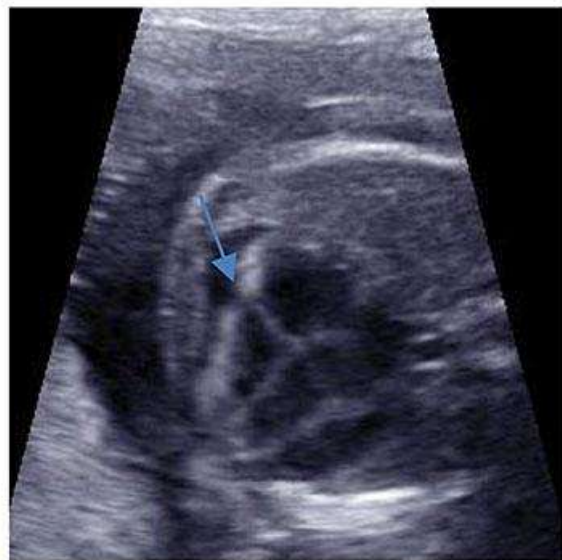
B. Pleural effusions



C. Ascites



D. Pericardial effusion



### Diagnostic Approach

Diagnosis requires suspicion in a pregnant patient with maternal edema and evidence of fetal hydrops or placentomegaly.

### Maternal Evaluation

Maternal assessment should include:

- Blood pressure measurement
- Weight change assessment
- Urine protein testing
- Complete blood count
- Liver enzymes
- Renal function
- Serum albumin
- Uric acid

- Coagulation profile if clinically indicated
- Oxygen saturation and chest imaging if dyspnea is present

Hemodilution is an important clue. In typical preeclampsia, hemoconcentration is common because of intravascular volume contraction. In Ballantyne syndrome, anemia or low hematocrit may reflect plasma volume expansion [4].

### Fetal and Placental Evaluation

Ultrasound is central and should evaluate:

- Presence and severity of fetal hydrops
- Placental thickness
- Amniotic fluid volume
- Fetal anatomy
- Cardiac function

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- Fetal rhythm
- Middle cerebral artery peak systolic velocity
- Umbilical artery and venous Doppler findings
- Evidence of fetal tumors or placental masses
- Monochorionic twin complications

The presence of fetal hydrops plus a thickened placenta in a mother with edema should strongly suggest Ballantyne syndrome.

### Etiologic Workup

The cause of hydrops should be investigated because management depends on reversibility. Evaluation may include:

- Maternal blood type and antibody screen
- Parvovirus B19 testing
- TORCH or targeted infection studies
- Fetal echocardiography
- Karyotype or chromosomal microarray when indicated
- Evaluation for twin-to-twin transfusion syndrome
- Assessment for fetal anemia
- Search for placental or fetal tumors
- Consideration of fetal structural anomalies

### Differential Diagnosis

The most important differential diagnosis is preeclampsia.

### Ballantyne Syndrome Versus Preeclampsia

Both conditions may show:

- Edema
- Hypertension
- Proteinuria
- Headache
- Elevated uric acid
- Pulmonary edema
- Abnormal liver or renal tests

Features favoring Ballantyne syndrome include:

- Fetal hydrops
- Placentomegaly
- Polyhydramnios
- Maternal hemodilution
- Rapid maternal edema paralleling fetal condition
- Improvement after correction of hydrops or delivery

Features favoring preeclampsia include:

- Hemoconcentration
- Absence of fetal hydrops
- Classic placental insufficiency with fetal growth restriction
- Severe hypertension with end-organ dysfunction

However, the two conditions can coexist. Some cases may begin as Ballantyne syndrome and progress to a preeclampsia-like state, or both syndromes may be present simultaneously [9].

Other differential diagnoses include:

- Peripartum cardiomyopathy
- Renal disease
- Nephrotic syndrome
- Severe anemia

- Hypoalbuminemia
- Liver disease
- Heart failure
- Pulmonary embolism
- Severe thyroid disease

### Maternal Complications

Maternal morbidity can be significant. Reported complications include:

- Pulmonary edema
- Severe hypertension
- Eclampsia
- Renal dysfunction
- Respiratory compromise
- Need for urgent delivery
- Intensive care admission
- Hemorrhagic complications
- Persistent postpartum edema

A systematic review reported severe maternal complications, including pulmonary edema, in a substantial proportion of patients [1].

Maternal symptoms usually improve after delivery, pregnancy termination, or successful correction of fetal hydrops. The uploaded synthesis notes that maternal symptoms disappeared approximately 4.8–13.5 days after delivery in reviewed cases.

Ballantyne\_syndrome.pdf

### Fetal and Perinatal Outcomes

Perinatal prognosis is poor because Ballantyne syndrome reflects severe fetal disease. Outcomes depend on:

- Cause of hydrops
- Gestational age
- Severity and duration of hydrops
- Presence of fetal anemia
- Cardiac function
- Availability of fetal therapy
- Timing of diagnosis
- Maternal stability
- Feasibility of neonatal intensive care

A systematic review of 56 cases reported intrauterine fetal death in approximately 56% of cases [1]. Later reviews have reported perinatal loss rates of approximately 56–67% [7,10]. Reversible causes, such as fetal anemia from parvovirus B19 or alloimmunization, may have better outcomes when treated promptly.

### Management Principles

Management must address both maternal safety and fetal prognosis.

The main steps are:

1. Confirm fetal hydrops and placentomegaly.
2. Assess maternal severity.
3. Identify the cause of hydrops.
4. Determine whether fetal therapy is possible.
5. Stabilize the mother.

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6. Treat the underlying fetal or placental disorder when feasible.
7. Deliver or terminate pregnancy if maternal condition worsens or fetal disease is irreversible.

Ballantyne syndrome should be managed by a multidisciplinary team including maternal-fetal medicine, obstetrics, neonatology, anesthesia, cardiology or intensive care when needed, and relevant fetal therapy specialists.

### Etiology-Directed Fetal Therapy

When the cause of hydrops can be corrected, maternal symptoms may improve and pregnancy may continue.

Potential fetal interventions include:

- Intrauterine transfusion for fetal anemia
- Treatment of fetal arrhythmia
- Fetoscopic laser therapy for twin-to-twin transfusion syndrome
- Drainage or treatment of selected fetal fluid collections
- Treatment of selected placental tumors
- Supportive management with close monitoring when spontaneous improvement is possible

Cases associated with parvovirus B19 may respond to intrauterine transfusion if severe fetal anemia is present [6].

The key principle is that maternal disease may resolve when fetal-placental hydrops resolves.

### Delivery or Pregnancy Termination

When fetal hydrops is irreversible, fetal demise has occurred, gestational age is advanced, or maternal status deteriorates, delivery or pregnancy termination is often required.

Indications may include:

- Worsening pulmonary edema
- Severe or uncontrolled hypertension
- Eclampsia
- Progressive renal or hepatic dysfunction
- Maternal respiratory compromise
- Nonreassuring fetal status at viable gestation
- Irreversible fetal anomaly
- Failed fetal therapy
- Intrauterine fetal death

Maternal symptoms typically improve after delivery or uterine evacuation, supporting the placental origin of the syndrome [1,11].

### Postpartum Course

After delivery, maternal edema, hypertension, and laboratory abnormalities usually improve within days. However, close monitoring remains necessary because pulmonary edema, hypertension, and renal dysfunction may persist transiently.

Postpartum care should include:

- Blood pressure monitoring
- Fluid balance assessment
- Respiratory monitoring if dyspnea was present
- Renal and liver function testing
- Diuresis management if needed
- Thromboprophylaxis according to risk

- Psychological support after fetal loss or severe neonatal illness
- Counseling regarding recurrence risk based on etiology

If the cause was immune hydrops, future pregnancy planning requires specialized monitoring and prevention strategies.

### Proposed Diagnostic and Therapeutic Algorithm

1. **Suspect Ballantyne syndrome** in a pregnant patient with generalized edema and fetal hydrops.
2. **Perform detailed ultrasound** to confirm hydrops, placentomegaly, and amniotic fluid status.
3. **Evaluate maternal severity** with blood pressure, urine protein, CBC, renal function, liver enzymes, albumin, and respiratory status.
4. **Differentiate from preeclampsia**, noting that hemodilution supports mirror syndrome.
5. **Search for the cause of hydrops**, including anemia, infection, cardiac disease, fetal anomalies, placental tumors, and twin complications.
6. **Assess reversibility**.
7. **Treat the fetal cause** if feasible and maternal condition is stable.
8. **Monitor maternal and fetal response closely**.
9. **Proceed to delivery or pregnancy termination** if maternal status worsens or fetal hydrops is irreversible.
10. **Continue postpartum monitoring** until maternal edema, blood pressure, and laboratory findings resolve.

### DISCUSSION

Ballantyne syndrome is diagnostically challenging because its maternal presentation overlaps with preeclampsia. Both conditions may include edema, hypertension, proteinuria, and pulmonary edema. The key distinction is that Ballantyne syndrome occurs in the setting of fetal hydrops and placentomegaly, and maternal hemodilution is more characteristic than hemoconcentration.

The syndrome is best understood as a maternal manifestation of severe fetal-placental disease. This concept explains why treatment of the fetal condition can improve maternal disease in selected cases. It also explains why delivery or pregnancy termination usually leads to maternal recovery when the fetal condition cannot be corrected.

The evidence base remains limited because Ballantyne syndrome is rare and most publications are case reports, case series, and systematic reviews of heterogeneous cases. Despite this limitation, several principles are consistent: the triad defines the syndrome, preeclampsia is the main mimic, perinatal prognosis is poor, and maternal improvement depends on resolution of the fetal-placental hydrops.

Future research should focus on identifying biomarkers that distinguish mirror syndrome from preeclampsia, defining the role of angiogenic factors such as sFlt-1 and PlGF, and

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clarifying which fetal interventions most reliably reverse maternal disease.

## CONCLUSION

Ballantyne syndrome is a rare pregnancy complication defined by fetal hydrops, placentomegaly, and maternal edema. It frequently mimics preeclampsia but is distinguished by the presence of fetal-placental hydrops and often by maternal hemodilution. The syndrome may arise from any severe cause of fetal hydrops, including alloimmunization, parvovirus B19 infection, twin-to-twin transfusion syndrome, fetal cardiac disease, placental tumors, fetal tumors, chromosomal abnormalities, and vascular malformations. Perinatal prognosis is poor, and maternal morbidity can be significant. Management should be individualized and etiology-directed. If fetal hydrops is reversible, fetal therapy may lead to maternal improvement. If maternal condition worsens or hydrops is irreversible, delivery or pregnancy termination is usually required, with maternal recovery typically occurring after resolution of the fetal-placental pathology.

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