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Mirror syndrome: a literature review

Zespół lustrzany – przegląd literatury

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Abstract

Mirror syndrome, or Ballantyne syndrome (BS), was described for the first time in 1892 by John W. Ballantyne. It is classically defined as triple oedema: association of foetal, placental and maternal oedema. In this syndrome, a pregnant woman with hydrops fetalis reflects (as in a mirror) the signs present in the foetus and develops symptoms similar to those displayed by the foetus. Mirror syndrome is not a widely described disease, its prevalence is unknown and there are relatively few literature reports of the condition. It coexists with various pathologies of pregnancy and structural anomalies of the foetus and placenta; it may occur after viral infections during pregnancy. The pathogenesis of Ballantyne syndrome remains unexplained and there is a wide variety of clinical signs. There exist theories for the pathomechanism of the syndrome, but none of them is fully satisfactory. Mirror syndrome is potentially life-threatening and is associated with increased foetal mortality and maternal morbidity. The treatment of choice for mirror syndrome in the mother is the treatment of oedema in the foetus, as the aetiology of the condition suggests. In mirror syndrome, the prognosis for the foetus is unfavourable and many cases end with intrauterine death. The treatment of hydrops fetalis, regardless of its cause, often leads to the resolution of symptoms in the mother, and, at the same time, better prognosis for the foetus.

Keywords: mirror (Ballantyne) syndrome, pregnancy complications, hydrops fetalis

Streszczenie

Zespół lustrzany lub zespół Ballantyne'a (*mirror syndrome*, *Ballantyne syndrome*, BS) został po raz pierwszy opisany przez Johna W. Ballantyne'a w 1892 roku. Klasycznie jest definiowany jako asocjacja potrójnego obrzęku, na który składają się obrzęk płodu, obrzęk łożyska i obrzęki u kobiety w ciąży. W zespole tym kobieta ciężarna, u której doszło do wystąpienia obrzęku uogólnionego u płodu, odzwierciedla (jak w lustrze) objawy występujące u płodu i manifestuje symptomy zbliżone do tych występujących u płodu. Zespół lustrzany nie jest powszechnie opisywaną jednostką chorobową; częstość jego występowania jest nieznana, w literaturze przedstawiono stosunkowo nieliczne jego przypadki. Współistnieje z różnorodnymi patologiami ciąży, anomaliami strukturalnymi płodu i łożyska, może wystąpić po przebyciu zakażeń wirusowych w ciąży. Patogeneza zespołu Ballantyne'a do tej pory pozostaje niewyjaśniona, a jego objawy kliniczne są mocno zróżnicowane. Istnieją teorie wyjaśniające patomechanizm opisywanych zmian, jednak żadna z nich nie jest w pełni satysfakcjonująca. Wystąpienie zespołu lustrzanego jest potencjalnie zagrażającą życiu patologią, związaną ze zwiększoną umieralnością płodów i zwiększoną zachorowalnością matek. Leczenie z wyboru zespołu lustrzanego u matki stanowi – zgodnie z jego etiologią – leczenie obrzęku płodu. Prognoza dla płodu w przypadku wystąpienia opisywanego zjawiska jest niekorzystna, a wiele przypadków kończy się zgonem wewnątrzmacicznym. Leczenie obrzęku płodu, niezależnie od przyczyny jego powstania, często prowadzi do ustąpienia objawów u matki, poprawiając równocześnie rokowanie dla płodu.

Słowa kluczowe: zespół lustrzany (Ballantyne'a), powikłania ciąży, obrzęk płodu

INTRODUCTION

Mirror syndrome was initially defined as triple oedema: association of foetal, placental and maternal oedema. At a later time, other manifestations apart from oedema were indicated as characteristic for mirror syndrome in the pregnant woman, such as progressive weight gain, elevated arterial pressure and albuminuria, among others. In this syndrome, a pregnant woman with hydrops fetalis reflects pathological signs present in the foetus and develops symptoms similar to those present in the developing foetus[1].

HISTORY

Mirror syndrome (Ballantyne syndrome, BS) was described for the first time in 1892 by John W. Ballantyne in his book *The Diseases and Deformities of the Foetus*. Ballantyne used the term "general dropsy of the foetus" to present cases of hydrops fetalis with fluid in the abdominal cavity and the chest and with concomitant oedema of the placenta which were associated with Rh immunisation. Ballantyne also noted that maternal oedema and uterine enlargement were more common in mothers with hydrops fetalis than in normal pregnancies. In patients with hydrops fetalis albuminuria was also more common[2]. The term "mirror syndrome" was introduced in 1956 by O'Driscoll, who reported cases of co-occurrence of hydrops fetalis and massive maternal oedema, particularly in the lower part of the body. O'Driscoll noted that oedema could develop very rapidly in pregnant women, as soon as in a few days' time, while it resolved within a few hours after delivery[3].

NOMENCLATURE

Various names are used for the condition in the literature: apart from "mirror syndrome" and "Ballantyne syndrome" the following names are most often mentioned: pseudotoxemia, triple oedema, preeclampsia-like disease and maternal hydrops syndrome[4].

EPIDEMIOLOGY

Mirror syndrome is not a common condition[5]. Its prevalence is unknown and presumably some cases are not diagnosed[6]. There are relatively few reports of Ballantyne syndrome in medical literature. The majority of cases occur before week 30 of gestation[1]. The lowest reported gestational age for mirror syndrome is 16 weeks; in this case there was a dichorionic twin pregnancy[7].

ABNORMAL COURSE OF PREGNANCY, SELECTED CLINICAL CASES

Mirror syndrome can co-occur with various pathologies of pregnancy. There are reports of cases associated with structural anomalies of the foetus and the placenta and with viral infections[8]; the selected potential causes of mirror syndrome are described below. It is estimated that fewer than 10% of cases of mirror syndrome are associated with congenital defects[9].

Vein of Galen aneurysm revealed on foetal ultrasound was reported in 1990 as a new cause of mirror syndrome[10]. Vein of Galen aneurysm is classified as a rare vascular anomaly with an unknown prevalence, which is estimated to be 1:25,000 births. Despite the possibility of intravascular therapy in neonates and infants, treatment outcomes are unsatisfactory due to a high risk of neurological disorders and the coexistence of cardiac failure: the potential mortality is as high as 90%[11].

Placental chorioangioma may cause a large number of pregnancy complications such as anaemia, polyhydramnios, cardiomegaly or intrauterine growth restriction; there are also reports in medical literature of mirror syndrome in the mother associated with placental chorioangioma. In terms of histology, placental chorioangioma belongs to a group of benign placental tumours of the hamartoma type. The complications mentioned above are most likely to occur when the tumour is more than 4–5 cm in diameter. The prevalence of placental chorioangioma is estimated to be 1:9,000–50,000 pregnancies. In one case the following sonographic signs were observed: foetal oedema with fluid in the chest cavity, ascites, polyhydramnios, moderate cardiomegaly, hyperkinetic circulation and anaemia. In the mother, the following signs were observed: oedema, oliguria, anaemia, elevated liver enzyme levels, hypoproteinaemia and hypokalaemia. The pregnancy was ended at week 29 through caesarean section due to the deteriorating condition of both the mother and the foetus. The newborn died a few hours after birth, while the mother's manifestations resolved 2 days after the end of pregnancy[12,13].

Sacrococcygeal teratoma (SCT) is one of the most common tumours occurring in neonates; the prevalence rate is 1:40,000 of live births. It is characterised by rapid growth and rich vasculature, which leads to the signs of circulatory failure. In a patient with mirror syndrome associated with foetal sacrococcygeal teratoma the following manifestations occurred: sacral pain, limb oedema, proteinuria, arterial hypertension and grade 1 hydronephrosis. Foetal ultrasound scan revealed skin oedema, ascites, pericardial effusion and fluid in the pleural cavities. The pregnancy was ended at week 28 with caesarean section. A stillborn neonate was delivered and the mother developed ovarian vein thrombosis postoperatively. Her hydronephrosis exacerbated to grade 3/4. The manifestations resolved a few days after appropriate treatment was administered[14].

Ebstein's anomaly (EA) is a rare congenital heart defect occurring at a rate of approximately 1:200,000 births. Patients with this pathology have a tricuspid valve malformation in that the leaflets are abnormally positioned, which

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is associated with apical displacement of the valvular ring and right ventricle enlargement. In a case of lethal hydrops fetalis caused by Ebstein's anomaly the pregnant woman developed mirror syndrome manifesting with oedema and anaemia[15,16].

Foetal cardiac dysfunction is another potential cause of mirror syndrome; cases of the syndrome have been reported in foetuses with tachycardia. In a case reported by Erdogdu et al., foetal ultrasound scan at week 27 of pregnancy revealed massive ascites, fluid in the right pleural cavity, polyhydramnios, placentomegaly and arrhythmia in the form of tachycardia of 300–350 beats/min with a normal foetal heart anatomy. The mother developed vulval and tibial oedema and a strong headache. After digoxin treatment was started, the signs resolved both in the mother and in the foetus. The pregnancy was ended at week 37 with caesarean section due to the release of amniotic fluid and breech presentation; no abnormalities were found in the newborn's heart[17,18].

Viral infections during pregnancy are a relatively common cause of Ballantyne syndrome.

Parvovirus B19 (B19V) belongs to the *Parvoviridae* family of viruses and its genome is composed of a single-stranded DNA. B19V causes numerous diseases such as erythema infectiosum, arthritis, gloves and socks syndrome or various haematological disorders; in addition, if the infection is transmitted to the foetus, it may develop non-immune hydrops fetalis. There are reports of B19V-related hydrops fetalis cases complicated by mirror syndrome in the mother manifesting with pulmonary oedema, effusion in the pleural cavities, arterial hypertension, elevated liver enzyme levels, proteinuria, anaemia and hyperuricaemia, among other signs[19–21].

Cytomegalovirus (CMV, HCMV), a DNA virus, is the cause of numerous conditions in humans; individuals with compromised immunity, transplant recipients, the elderly and foetuses are at the highest risk of CMV infection complications. Medical literature includes case reports of mirror syndrome associated with CMV[22–24].

Coxsackie viruses are RNA viruses from the *Picornaviridae* family. There are two groups of Cocksackie viruses: Coxsackie A and Coxsackie B. Coxsackie B may cause many diseases and their complications such as myocarditis, pericarditis, hepatitis, milder infections with fever, skin eruptions or upper respiratory infections. Ambrosini et al. reported Coxsackie B infection as the cause of mirror syndrome in a patient with generalised oedema (neck, abdomen, vulva), oliguria, hypotension, cyanosis, heart failure, anaemia and hypoproteinaemia. The foetus developed hydrops with ascites, polyhydramnios, pericardial effusion and skin and placental oedema. Due to the signs of heart failure in the mother, emergency caesarean section was performed at week 27 of gestation. The newborn had an Apgar score of 5 and the mother developed myocarditis during the postpartum period[25,26].

There are reports saying that a potential cause of mirror syndrome is **Rh incompatibility**. Rustamov described the case of an RhD-negative pregnant patient who at week 26 developed headaches, moderate oedema and vomiting; in addition, foetal hypokinesia, elevated blood pressure, proteinuria and an increased urea level were observed. Foetal ultrasound revealed oligohydramnios, oedema and pericardial effusion, while vascular flow rates were abnormal as assessed with Doppler ultrasound. After a few days of observation and treatment and deteriorating laboratory parameters in the mother, intrauterine transfusion was performed. The woman developed severe pulmonary oedema and decreased haemoglobin and platelet levels; progressing mirror syndrome was diagnosed. Emergency caesarean section was performed; the neonate died one hour after the end of pregnancy. The mother's clinical condition improved considerably within a few days after delivery; laboratory parameters returned to normal within a week[27].

Medical literature also contains reports of mirror syndrome in **twin pregnancies**. Chai et al. analysed the occurrence of mirror syndrome in **diamniotic monochorionic (DM) twin pregnancies**, complicated with twin to twin transfusion syndrome (TTTS). Among 103 analysed pregnancies complicated with TTTS, in 19 cases Quintero stage 4 (out of 5) TTTS was diagnosed, which is characterised by the sequence polyhydramnios/oligohydramnios, abnormal Doppler ultrasound results, hydrops fetalis and absence of urine in the urinary bladder of the donor. In 5 patients with stage 4 TTTS mirror syndrome occurred; the foetal age at the time was 19.4–28.2 weeks of gestation. All women developed oedema; arterial hypertension and oliguria were also observed. Based on laboratory tests, anaemia, hypoproteinaemia, proteinuria and elevated liver enzyme levels were found. Maternal complications were observed such as postpartum haemorrhage, pulmonary oedema and congestive heart failure[28,29].

Mirror syndrome has also been reported for **diamniotic dichorionic (DD) twin pregnancies**. At week 23 a patient pregnant with DD twins developed arterial hypertension and bilateral massive leg oedema. In one of the foetuses, ultrasound scan revealed hydrops fetalis, polyhydramnios, hypoplastic left heart syndrome (HLHS), brachycephaly with hypomineralisation, abnormal gyration of the cerebral cortex, bilateral femoral fractures and placentomegaly. The other foetus had a normal anatomy and amniotic fluid volume. A decision was made to perform selective termination of the sick foetus. After the procedure, the woman's manifestations resolved and her parameters stabilised. At week 27 the pregnancy was ended with caesarean section due to premature rupture of membranes and breech presentation. The newborn weighed 734 g (10th percentile) and had an Apgar score of 8 in the first minute of life. The child stayed at an intensive care unit for 3 months after birth[30].

A less typical case of mirror syndrome was described by Giacobbe et al. At week 37 of pregnancy, the patient developed leg oedema and **massive vulval oedema**, mild anaemia, hypoalbuminaemia and proteinuria. Foetal ultrasound

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did not reveal any oedema; however, polyhydramnios and placentomegaly were present. The pregnancy was ended with caesarean section due to increasing proteinuria and the lack of possibility of natural delivery due to massive vulval and anal oedema. During the postoperative period the patient developed arterial hypertension (managed with pharmacotherapy); the neonate had an Apgar score of 7 in the first minute of life. The mother's manifestations resolved within 7 days after delivery[6].

Lee and Hwang reported a case of mirror syndrome in a 28 weeks pregnant patient. Cordocentesis revealed **acute megakaryoblastic leukaemia** and Down's syndrome in the foetus. The pregnancy was ended with caesarean section at week 31 due to respiratory problems in the pregnant woman. After delivery, the patient's mirror syndrome manifestations resolved. The child underwent surgery for duodenal atresia. The leukocyte level returned to normal and no immature blastic cells were observed; the patient did not develop myelodysplastic syndrome or acute leukaemia[1].

A case of Ballantyne syndrome (leg oedema, feet pain and headache) was also reported for a 26 weeks pregnant patient in whose child congenital high airway obstruction syndrome (**CHAOS**) was diagnosed *in utero* in the form of subglottic laryngeal stenosis. After caesarean section a stillborn neonate was delivered. Additional observed pathologies included the presence of fluid in the abdominal cavity, club feet, a pelvic right kidney, appendix located in the left upper quadrant near the spleen, pulmonary enlargement and the lack of the right middle lung lobe; abdominal circumference upon birth was 36 cm. This is probably the only case of concomitant mirror syndrome and CHAOS syndrome reported to date[9].

Zhang et al. reported a rare case of mirror syndrome caused by the presence of **haemoglobin Barts** in the foetus, a variety of α-thalassemia which involves impaired production of haemoglobin α chains. In this pathology, tetrameric γ chains (haemoglobin Barts) are present, which originate due to a defect in all four globin chains. As a result, there is little circulating haemoglobin, which leads to hydrops fetalis and death. The neonate was born by natural delivery at week 31 of gestation using obstetrical forceps and died soon after birth[31,32].

In a study by Braun et al., which included 56 cases of mirror syndrome, the most common manifestations in the mother were: weight gain and oedema (89.3% of patients), elevated arterial pressure (60.7%), anaemia and haemodilution (46.4%), albuminuria and proteinuria (42.9%), elevated levels of uric acid and creatinine (25%), moderately increased levels of liver enzymes (19.6%), oliguria (16.1%), headache and vision disturbances (14.3%); severe maternal complications, including pulmonary oedema, occurred in 21.4% of patients[33].

PATHOGENESIS

The aetiology and pathogenesis of mirror syndrome remains unexplained and there is a wide variety of clinical signs. There are theories explaining the pathomechanism of the syndrome, but none of them is completely satisfactory and further research is necessary to account for it[14,34].

It is suggested that the cause of Ballantyne syndrome may be placental abnormality. Some studies suggest that placentomegaly caused by an increased secretion of human chorionic gonadotropin (hCG) may lead to placental ischaemia. As a compensatory mechanism, placental perfusion increases, which may cause as much as a tenfold increase in placental production of renin; as a result, aldosterone production in the mother increases leading to fluid retention in her body. In addition, some studies suggest that an increased hCG level may occur in cases of mirror syndrome associated with chorioangioma[1].

Recent studies suggest that the primary mechanism for mirror syndrome is the lack of balance between angiogenic and antiangiogenic factors in pregnant patients[30]. It has been demonstrated that the levels of angiogenic and antiangiogenic factors circulating in the maternal blood such as soluble fms-like tyrosine kinase-1 (sFlt-1), also called soluble vascular endothelial growth factor receptor-1 (sVEGFR-1), and soluble endoglin (sENG) are elevated in mirror syndrome. These factors may be responsible for endothelial dysfunction, which may be the cause of Ballantyne syndrome. In addition, hydrops fetalis is often associated with villous oedema, which may reduce the intervillous space, causing a decrease in the flow of blood through the villi and, consequently, reduced oxygen exchange. Hypoxia in trophoblast villi results in an increased production and release of sFlt-1 to the circulation of the mother, leading to damage in her endothelium. Exacerbated villous oedema can increase the production of the antiangiogenic factors mentioned above[1,28].

Another hypothesis assumes that mirror syndrome is the result of an increase in oxidative stress in the placenta caused (as in preeclampsia) by abnormal trophoblast invasion. This leads to hypoxia with a subsequent ischaemia–reperfusion reaction. A second theory explaining an increase in oxidative stress assumes that the remains of necrotic or apoptotic cells (their increased numbers are observed in placentas with a large mass, e.g. in multifoetal pregnancies or molar pregnancies) cause elevated levels of proinflammatory factors, which also results in an increase in oxidative stress[14].

In mirror syndrome, elevated levels of vasopressin and atrial natriuretic peptide (ANP) have been observed in the maternal blood, which may cause an increased volume of circulating blood, haemodilution and dilutional anaemia[28].

In a study on 40 normal pregnancies and 4 pregnancies complicated with mirror syndrome Espinoza et al. demonstrated a statistically significant increase in plasma sVEGFR-1 level in mothers with mirror syndrome compared to the control group which included patients with spontaneous premature deliveries; the groups were matched for gestational age. In addition, all patients with mirror syndrome

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were within >95th percentile for sVEGFR-1 concentration for a given gestational age. Histopathological examination of the patients' placentas was also performed. In the group with mirror syndrome, immature villi, oedematous lesions, an increased number of syncytial nodes, elevated fibrin concentration inside the villi and multifocal calcification of villi were observed; in the control group no such abnormalities were demonstrated[35]. Llurba et al. reported the case of a patient with mirror syndrome with an elevated level of sFlt-1 in the blood and a decreased level of free placental growth factor (PlGF). Following treatment and resolution of symptoms, sFlt-1 and PlGF levels returned to normal and were comparable to those in the control group. It was also noted that the concentrations of these factors were similar in patients with preeclampsia[34].

MIRROR SYNDROME OR PREECLAMPSIA?

Currently, mirror syndrome is considered to be a possible variety of preeclampsia. Both syndromes involve oedema, elevated arterial pressure and proteinuria; the placenta probably plays a key role in their aetiology and pathogenesis. Differentiating between mirror syndrome and preeclampsia may be difficult and the only difference lies in foetal oedema, which is only present in mirror syndrome. In addition, haematocrit and haemoglobin levels may be helpful in differential diagnosis: they are decreased in mirror syndrome (haemodilution, increased blood volume associated with elevated concentrations of vasopressin and ANP, oedema, dilutional anaemia) and increased in preeclampsia (haemoconcentration). In preeclampsia, a decreased platelet count is often observed, while in mirror syndrome it is typically within normal limits, which may also be helpful in the differential diagnosis between mirror syndrome and HELLP syndrome, whose name stands for haemolysis, elevated liver enzymes and, relevant to the comparison, low platelet count. In addition, preeclampsia is more common at later stages of pregnancy (it occurs rarely before week 24) than mirror syndrome (the majority of cases reported in the literature develop before week 30 of gestation)[18,28,30,33].

PROGNOSIS

Literature data show that mirror syndrome is potentially life-threatening and is associated with increased foetal mortality and maternal morbidity[5,28]. As previously mentioned, in mirror syndrome, prognosis for the foetus is unfavourable and many cases end with intrauterine death. The treatment of hydrops fetalis, regardless of its cause, often leads to the resolution of manifestations in the mother, and, at the same time, better prognosis for the foetus[34].

In a literature review of 56 cases of mirror syndrome conducted by Braun et al., intrauterine deaths and stillbirths occurred in 35.7% of cases[33]. Brochot et al. noted that the prognosis for a foetus with hydrops due to B19V infection complicated with mirror syndrome in the mother is worse than if there are no signs of mirror syndrome in the mother[20].

In the previously mentioned study by Chai et al. on diamniotic monochorionic pregnancies, in 5 cases complicated with TTTS and mirror syndrome, despite treatment the overall perinatal survival rate after 28 days was 28.57% (2 out of 7 foetuses that were alive *in utero* survived following selective termination of one of the foetuses or amnioreduction) and only 1 neonate survived beyond 18 months after selective termination of the other foetus[28]. There has also been a report of complete resolution of mirror syndrome in a mother of dichorionic diamniotic twins within 2 weeks of intrauterine death of a foetus with hydrops[36]. The signs of mirror syndrome resolve relatively quickly after treatment of hydrops fetalis, pregnancy termination or delivery[12]. In the previously mentioned study by Braun et al., the mean time of symptom resolution in the mothers was 8.9 days[33].

TREATMENT

The treatment of choice for mirror syndrome manifestations in the mother is the treatment of oedema in the foetus, as the aetiology of the condition suggests. If hydrops fetalis cannot be treated, maternal complications can be managed by ending the pregnancy. If it is possible to treat hydrops fetalis, there is a chance for a subsequent spontaneous maternal recovery[20].

The most common methods for the treatment of hydrops fetalis include: intrauterine transfusions for cases of anaemia, amnioreduction for cases of polyhydramnios, laser coagulation of blood vessels for cases of twin pregnancy or chorioangioma, injection of alcohol or other substances into the tumour in cases of chorioangioma, bed rest, reduction of fluid intake, dietary salt restriction, blood transfusion in the mother and drain insertion into the foetal pleural cavities if fluid is present. Depending on the causes and manifestations of the syndrome, the following medicines have been used to treat the condition: reserpine, acetazolamide, iron, vitamin B_{12}, hydrochlorothiazide, calcium lactate, spironolactone, adrenocorticotropic hormone (ACTH), methylprednisolone, diuretics, methyldopa, dexamethasone, nicardipine, labetalol, albumins, digoxin, magnesium sulphate, methyldopamine, flecainide, digitalis and phenobarbital, among others[12,18,33].

Conflict of interest

The authors do not report any financial or personal affiliations to persons or organisations that could adversely affect the content of or claim to have rights to this publication.

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