Eduvest – Journal of Universal Studies
Volume 6 Number 6, June, 2026
p- ISSN 2775-3735- e-ISSN 2775-3727

A Rare Case of Limb Body Wall Complex Associated with Environmental Mercury Exposure and Maternal Herpes Infection

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Keywords
limb body wall complex (LBWC); congenital anomaly; prenatal ultrasonography; antenatal screening; HSV infection.

ABSTRACT
Limb body wall complex (LBWC) is a rare and fatal congenital anomaly involving severe defects of the body wall, spine, limbs, and umbilical cord. This study aims to report a rare case of LBWC associated with environmental mercury exposure and maternal herpes infection, to review the literature on LBWC pathogenesis and risk factors, and to generate hypotheses for future etiological research. Early prenatal diagnosis is crucial; however, cases may be detected late in settings with limited antenatal care. We report a 37-year-old multiparous woman in whom LBWC was diagnosed by ultrasonography at 24–25 weeks' gestation. Imaging revealed a large abdominal wall defect with exposed intestinal loops, spinal deformity, limb dysplasia, and a short umbilical cord — findings that were confirmed postnatally. Maternal assessment identified active HSV-1/2 infection and residence near gold-mining activities, suggesting possible environmental mercury exposure. LBWC remains a devastating and fatal congenital condition. This case emphasizes the critical role of early antenatal screening and highlights the need for further research exploring possible associations between LBWC, maternal infections, and environmental exposures, particularly mercury exposure in gold-mining areas.

INTRODUCTION

Limb body wall complex (LBWC), also known as body stalk syndrome, is a rare and uniformly fatal poly-malformative syndrome with a reported frequency of between 1 per 14,000 and 1 per 31,000 pregnancies in large epidemiological studies (Adeleke et al., 2021; Haddout et al., 2022; Narayan et al., 2024; Ye et al., 2023), and approximately 0.32 per 100,000 births (Bhat et al., 2016; Chikkannaiah et al., 2013; D'Souza et al., 2004; Prasun et al., 2008). It is characterized by major defects in the fetal abdominal or thoracic wall, visceral herniation, significant scoliosis or spina bifida, limb deformities, craniofacial deformities, and umbilical cord abnormalities including a short or absent umbilical cord (Haddout et al., 2022; Van Allen et al., 1987; Ye et al., 2023). The diagnosis of LBWC can be established based on any three of the following features: (1) encephalocele or anencephaly with facial clefts; (2) thoraco- and/or abdominoschisis; and (3) limb deformities (Narayan et al., 2024; Van Allen et al., 1987). Given

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its fatal nature, prompt prenatal diagnosis is crucial in order to provide parents with timely counseling regarding pregnancy management (Bhat et al., 2016; Narayan et al., 2024). With its distinctive features, prenatal and antenatal ultrasonography can be helpful in obtaining an early diagnosis (Haddout et al., 2022; Narayan et al., 2024; Ye et al., 2023); however, reports on early pregnancy ultrasound screening for LBWC remain relatively few (Ye et al., 2023). We report a case of LBWC diagnosed by antenatal ultrasound (USG) at 37 weeks' gestation and confirmed after delivery.

The research urgency of this study stems from several critical factors. First, LBWC is a uniformly fatal condition, and early prenatal diagnosis is essential for informed pregnancy management decisions. Delayed diagnosis — as occurred in this case due to limited antenatal care access — has profound implications for maternal psychological well-being and pregnancy outcomes. Second, artisanal small-scale gold mining is expanding globally, with an estimated 15–20 million miners across more than 70 countries, including significant operations in Indonesia (Lombok, Kalimantan, and Sulawesi). Mercury, used in gold amalgamation, is released into the environment, contaminating water, soil, and the food chain, thereby exposing surrounding communities. Understanding whether such exposure contributes to LBWC or other severe congenital anomalies carries significant public health implications. Third, maternal HSV infection is common, with seroprevalence rates of 50–90% for HSV-1 and 10–30% for HSV-2 among women of reproductive age. If active maternal HSV infection during early gestation contributes to LBWC pathogenesis, this represents a potentially preventable risk factor through antiviral therapy or vaccination. Fourth, without case reports documenting these potential associations, researchers and clinicians lack the impetus for systematic epidemiological investigation (Murad & Wang, 2017; Nambiema et al., 2021; Page et al., 2016).

The novelty of this research lies in five aspects. First, this is the first case report of LBWC in a patient residing near an artisanal gold-mining area with documented environmental mercury exposure risk. Second, this is the first case report linking active maternal HSV-1/2 infection to LBWC. Third, this case describes LBWC in the Indonesian context (Sekotong, Lombok), where no previous LBWC cases have been reported. Fourth, this case provides detailed documentation of maternal environmental and infectious risk factors alongside comprehensive ultrasonographic and post-delivery photographic confirmation. Fifth, this case integrates mechanistic explanations from the toxicological literature (mercury-induced oxidative stress and DNA methylation changes) and virological literature (HSV teratogenicity and limb dysplasia) to propose plausible pathogenic pathways for LBWC, generating testable hypotheses for future research.

The purpose of this study is to report a rare case of limb body wall complex associated with environmental mercury exposure and maternal herpes infection, to review the literature on LBWC pathogenesis and risk factors, and to generate hypotheses for future etiological research. The research contribution is threefold: clinically, by raising awareness among obstetricians and ultrasonographers in high-risk areas — particularly gold-mining regions — about LBWC and the importance of early antenatal screening; from a public health perspective, by highlighting the need for environmental health policies to reduce mercury exposure in pregnant women and for maternal HSV screening and treatment; and from a research

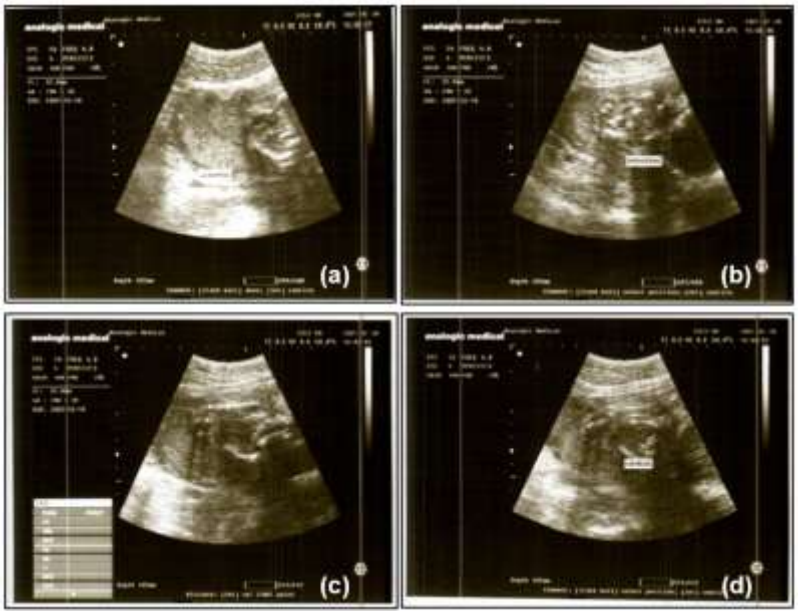
perspective, by providing a foundation for future epidemiological studies examining associations between LBWC, environmental mercury exposure, and maternal infections.

CASE REPORT

A 37-year-old multiparous woman presented for her first ultrasonography at 24-25 weeks' gestation on 5 years of her marriage. On presentation, she reported absence of fetal movement and fetal kicks since the previous day without abdominal pain and had no history of active vaginal bleeding. She had not previously consulted an obstetrician and had received antenatal care only from a midwife and also forget first day menstruation on her last period. There is no known consanguinity or family history of fetal malformations. On further history taking, additional social and environmental factors were identified: the patient resides near an illegal gold-mining site in Sekotong Lombok with a suspected risk of mercury and arsenic exposure. In addition, a local water study in her area previously reported suspected micro plastic contamination.

Preoperative physical examination revealed the patient in good general condition, fully conscious, and hemodynamically stable. On general status were on normal limit, obstetric status uterus fundus two fingers above umbilical regio, contractions are not detectable yet. The initial ultrasound examination (28 July 2025) showed gestational ages 24 weeks 2 days with estimated delivery date 15/11/2025. It also showed absence of the fetal abdominal wall with exposed intestinal loops visualized within the uterine cavity (Figure 1 b). Laboratory testing (30 July 2025) revealed positive IgG for anti-Toxoplasma and anti-cytomegalovirus (CMV), indicating prior exposure. The patient also tested positive for anti-HSV-1/2 IgM, indicating an ongoing infection with HSV-1 and/or HSV-2. At cesarean section, a neonate was delivered with no spontaneous respirations or crying and had eviscerated abdominal organs without any membranous covering.

USG (28 July 2025)



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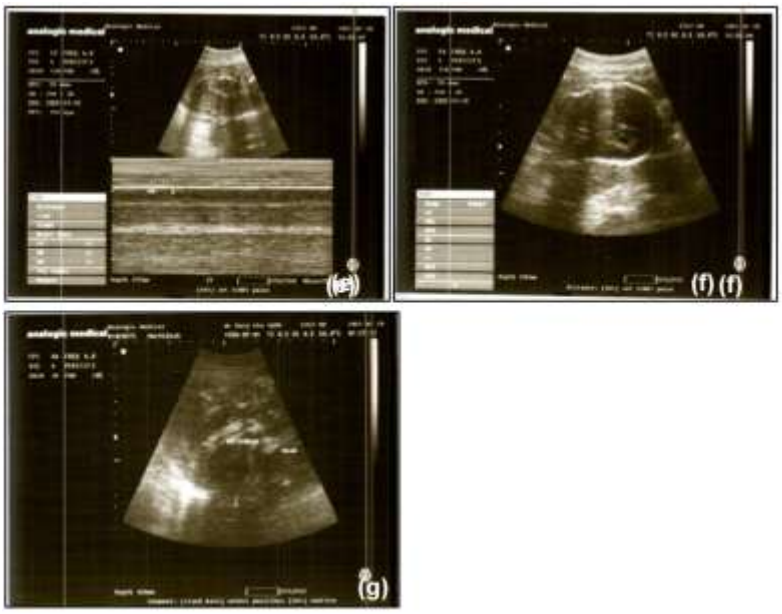

Figure 1 (a) placenta fundus corpus anterior, (b) exposed intestinal loops visualized within the uterine cavity, (c) length of femur, (d) cardiac, (e) Fetal Heart Rate 144 bpm (f) head circumtance (g) vertebrae

Figure 2. Post-delivery image (a) showing eviscerated abdominal organs without membranous covering (red arrow), hypoplastic right lower limb (blue arrow), a malrotated left lower limb with deformed foot (blue arrow), and short umbilical cord (yellow arrow). Furthermore, a curvature of the spine is abnormal. Fetus size (b) showing the fetus' size appeared similar to a kidney tray.

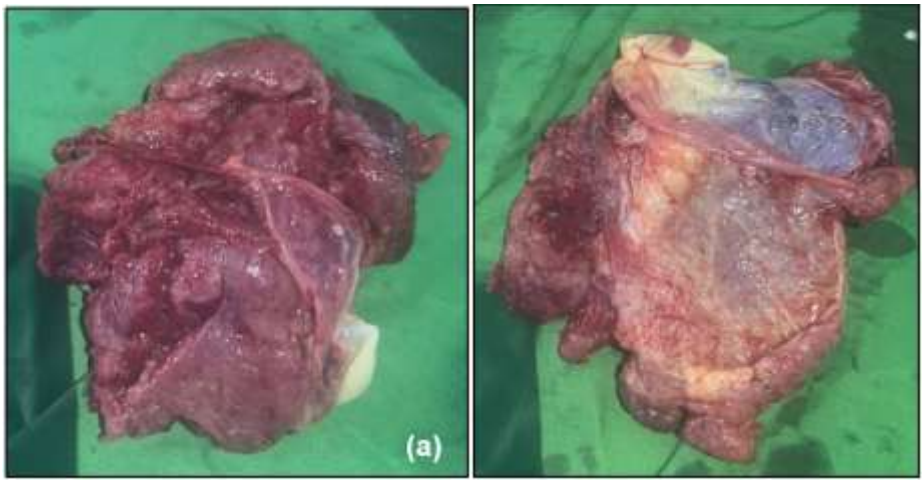

Figure 3. Post-delivery placenta: (a) inner (maternal) surface demonstrating complete cotyledons; (b) outer (fetal) surface of the placenta.

RESULT AND DISCUSSION

Limb body wall complex (LBWC) was originally defined by Van Allen et al. (1987) as the presence of at least two of three anomalies—exencephaly or encephalocele with facial clefts, thoracoschisis and/or abdominoschisis, and limb defects (Adeleke et al., 2021). More recent literature classifies LBWC into two main types—Type I with craniofacial defects and Type II with thoracoschisis or gastroschisis (Russo et al., 1993; Ye et al., 2023). By contrast, Sahinoglu et al. further refined this into a three-type system by separating defects above and below the umbilicus, with Type III denoting infraumbilical defects and an intact thoracic wall (Sahinoglu et al., 2007). The etiology of LBWC remains uncertain. To date, no specific environmental teratogen or genetic abnormality has been clearly linked to its development (Adeleke et al., 2021; Van Allen et al., 1987).

There are three main pathogenic mechanisms for LBWC that have been proposed in the literature (Hartwig et al., 1989; Kumtepe et al., 2003; Van Allen et al., 1987; Ye et al., 2023): (1) embryonic dysplasia leading to abnormal folding of the trilaminar embryonic disc during the first four weeks of development, (2) amniotic rupture leading to amniotic band sequence, and (3) embryonic vascular disruption resulting in impaired blood flow. In our case, the findings fulfilled the diagnostic criteria for LBWC and are most consistent with an embryonic dysplasia mechanism. Abnormal transverse folding of the embryonic disc likely resulted in gastroschisis, allowing the abdominal contents to protrude into an enlarged amniotic sac that inserted peripherally into the placental chorionic plate, leading to an absent or markedly shortened umbilical cord (Figure 5). Compression of the herniated abdominal viscera appears to have interfered with symmetrical development of the spine and thoracic cavity, resulting in spinal scoliosis. In addition, impaired spinal rotation and incomplete pelvic closure may explain the congenital limb deformities observed in this patient (Hartwig et al., 1989; Ye et al., 2023).

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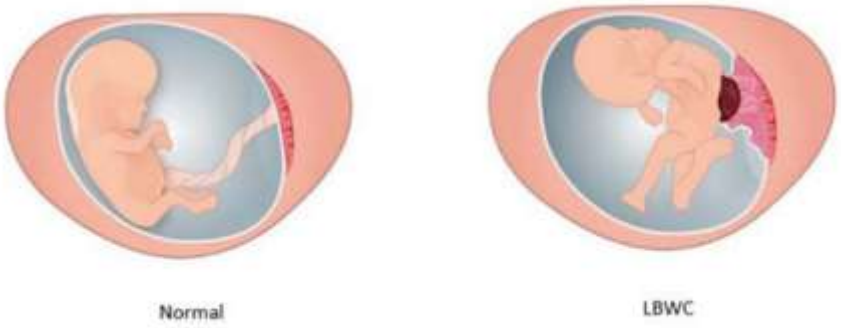

Figure 4. Comparison between a normally developing fetus (left) and a fetus with limb body wall complex (LBWC) (right), showing an extremely short or absent umbilical cord and evisceration of the abdominal contents.14

Early first-trimester ultrasound (11–13^{+6} weeks of gestation) is regarded as the safest and most reliable approach for the prenatal diagnosis of LBWC and for counseling regarding possible medical termination of pregnancy (Adeleke et al., 2021; Ye et al., 2023). Fetal age is often difficult to estimate from ultrasound findings due to a persistently flexed or hyperextended fetal posture; consequently, gestational age is determined using menstrual dating instead (Ye et al., 2023). Some authors consider abnormal spinal curvature to be the most characteristic ultrasonographic feature of LBWC in early gestation (Becker et al., 2000). In general, the sonographic basis for diagnosing LBWC includes the presence of a large abdominal or thoracoabdominal wall defect with visceral herniation, a short or absent umbilical cord, and associated spinal abnormalities (Jayaram et al., 2018; Mulita et al., 2020). The case series of 18 LBWC cases reported by Cai-Hong et al., in which thoracoschisis or gastroschisis were the most frequent findings, is consistent with the findings in the present patient. Furthermore, the umbilical cord was frequently difficult to visualize or appeared markedly shortened (Adeleke et al., 2021; Ye et al., 2023), as was likewise observed in this case (Figure 3). In addition, all fetuses had developmental abnormalities of the spine, including kyphosis, torsion, or scoliosis of varying degrees (Ye et al., 2023).

Cantrell's pentalogy represents an important differential diagnosis of LBWC due to substantial overlap in prenatal ultrasonographic findings. Cantrell's pentalogy is classically characterized by the presence of omphalocele, ectopia cordis, lower sternal defects, anterior diaphragmatic defects, and pericardial abnormalities, with omphalocele and ectopia cordis considered its hallmark features (Chen et al., 2007). However, spinal malformations and limb developmental anomalies are not components of Cantrell's pentalogy and therefore serve as critical distinguishing features from LBWC (Stein et al., 2009; Ye et al., 2023). In the present case, the identification of significant spinal deformities and limb abnormalities strongly supports the diagnosis of LBWC.

From a maternal risk perspective, the mother in this case tested positive for HSV-1, as indicated by reactive anti-HSV-1/2 IgM serology. Maternal HSV infection may result in vertical transmission, leading to neonatal herpes within the first 28 days of life (Carolyn & Zane, 2007; Clarke et al., 2024; Fleming et al., 1997; Nahmias et al., 1990; Stanberry et al., 1999). HSV-1 and HSV-2 can be transmitted to the fetus via the transplacental route; however, transmission through contact with a virus-shedding lesion in the genital tract is a far more common mode of vertical transmission. Both HSV-1 and HSV-2 are neurotropic and lie

dormant in the dorsal root ganglion following primary infection (Clarke et al., 2024). Adverse outcomes of maternal infection include teratogenic effects resulting in congenital anomalies in approximately 3% of live births, as well as fetal growth restriction, miscarriage, stillbirth, neonatal death, prematurity, and increased maternal morbidity (Carolyn & Zane, 2007; Clarke et al., 2024; Fleming et al., 1997; Nahmias et al., 1990; Stanberry et al., 1999). Upon reaching the fetus, these viruses are highly teratogenic and are associated with a classic triad of manifestations involving the skin (aplasia cutis, scarring, and erosions), central nervous system (ventriculomegaly, microcephaly, and intracranial calcifications), and eyes (chorioretinitis and atrophy), as well as limb dysplasia (Clarke et al., 2024; Megli & Coyne, 2022). In the present case, the predominant manifestation was limb dysplasia.

The mother in this case resides in a settlement located in close proximity to gold-mining activities, raising concern for chronic environmental mercury exposure. Although the biological pathways linking prenatal mercury exposure to adverse pregnancy outcomes remain incompletely defined, several plausible mechanisms have been described. Mercury is a potent toxicant with particular relevance to fetal development and early life (Bose-O'Reilly et al., 2010). From an endocrine perspective, mercury has been described as an endocrine-disrupting agent that may interfere with normal reproductive hormonal regulation, including estrogenic and androgenic signaling (Rice et al., 2014). At the molecular level, the high toxicity of mercuric ions (Hg^{2+}) is largely attributed to their strong affinity for sulfhydryl (–SH) and thiol-containing groups in proteins, which can disrupt enzymatic function and redox homeostasis in maternal-fetal tissues (Rice et al., 2014). During pregnancy, mercury exposure has also been linked to oxidative stress — a pathophysiologic state that can contribute to endothelial injury and compromised placental development and perfusion (Al-Gubory et al., 2010; Burton & Jauniaux, 2004). Oxidative stress and placental dysfunction have, in turn, been associated with pregnancy complications including pregnancy loss and other adverse outcomes (Al-Gubory et al., 2010; Burton & Jauniaux, 2004; Gupta et al., 2007).

As outlined above, even relatively low-level prenatal mercury exposure efficiently crosses the placenta, accumulates in the fetus, and alters DNA methylation patterns in cord blood and placental tissue (Charkiewicz et al., 2025; Cohen et al., 2005; Dutta & Ruden, 2024; Ha et al., 2017; Król-Pakulska & Pakulski, 2017). Such exposure has been associated with impaired neurodevelopment and cognition, including delayed learning and memory, as well as structural brain injury that may culminate in cerebral and psychomotor impairment, with autopsy findings of cerebellar hypoplasia, abnormal cortical neuronal number, disturbed neuronal migration and lamination, and reduced total brain mass (Charkiewicz et al., 2025; Cohen et al., 2005). Moreover, epidemiological data from low- and middle-income countries indicate that mercury exposure during pregnancy is linked to higher rates of spontaneous abortion, stillbirth, preterm delivery, low birth weight, congenital anomalies, and low Apgar scores (Charkiewicz et al., 2025; Cohen et al., 2005). Supporting these mechanistic concerns, a large prospective cohort study in Northern Tanzania found that higher maternal total blood mercury concentrations during mid-pregnancy were associated with increased odds of stillbirth and other adverse birth outcomes (Nyanza et al., 2020). Taken together, these observations provide a biologically plausible framework in which chronic mercury exposure related to nearby gold-mining activities could have contributed to the adverse fetal outcome in this

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LBWC case, although a direct causal relationship cannot be established from a single observation and should be interpreted with caution.

Clinical Implications

This case highlights the need for strengthened preventive strategies to improve maternal and fetal outcomes in communities residing near artisanal and small-scale gold mining areas. Reducing mercury exposure requires coordinated environmental and occupational health policies, effective enforcement, and community engagement. Establishing local environmental health and safety committees in collaboration with village authorities and healthcare providers may facilitate culturally appropriate risk mitigation and awareness.

Preventive efforts should prioritize vulnerable populations, particularly pregnant women, through targeted education on mercury exposure, promotion of safe food consumption, and periodic biomonitoring. In parallel, mandatory training for healthcare workers is essential to enhance early detection, counseling, and management of environmental and infectious risks, including herpes simplex virus during pregnancy. Integrating environmental health screening and infection surveillance into routine antenatal care may contribute to improved reproductive outcomes in high-risk settings.

Limitations

Future research should focus on systematic investigations into potential associations between LBWC and environmental exposures, particularly in regions with artisanal and small-scale gold mining, such as Sekotong, Lombok. Population-based studies integrating detailed environmental assessments, biomonitoring of mercury and other heavy metals, and robust prenatal imaging data are needed. In addition, advanced toxicological and epigenetic studies examining mercury-induced molecular and developmental disruptions may provide further insight into plausible pathogenic pathways contributing to severe fetal malformations such as LBWC.

CONCLUSION

Limb body wall complex is a rare and uniformly fatal congenital anomaly characterized by severe defects of the body wall, spine, limbs, and umbilical cord. This case highlights the diagnostic challenges of LBWC when antenatal care is limited and first-trimester ultrasonography is not performed, resulting in delayed recognition until late gestation. The ultrasonographic findings of a large abdominal wall defect with exposed viscera, marked spinal deformity, limb dysplasia, and a short umbilical cord were consistent with established diagnostic criteria and were confirmed postnatally.

Although the precise etiology of LBWC remains unclear, the findings in this case are most compatible with an embryonic dysplasia mechanism occurring early in gestation. In addition, the presence of maternal risk factors — including active HSV infection and potential chronic environmental mercury exposure from nearby gold-mining activities — raises important considerations regarding multifactorial influences on adverse fetal outcomes. While a direct causal relationship cannot be established from a single case, these factors represent biologically plausible contributors that warrant further investigation.

This report underscores the critical importance of early and adequate antenatal surveillance, particularly first-trimester ultrasound screening, to allow prompt diagnosis, appropriate counseling, and timely decision-making regarding pregnancy management. Furthermore, it highlights the need for greater awareness of environmental and infectious risk factors among pregnant women living in high-risk settings. Future studies with larger cohorts are required to better elucidate the potential interactions between environmental exposures, maternal infections, and the pathogenesis of limb body wall complex.

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