Polyhydramnios as a Sonographic Marker of Fetal Anomaly Risk, Genetic Etiology, and Perinatal Outcomes: A Scoping Review

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Abstract. Polyhydramnios is an excess of amniotic fluid that may indicate maternal disease, fetal anomalies, genetic etiology, or increased perinatal risk. This scoping review mapped evidence on definitions, etiologies, fetal anomalies, genetic findings, and perinatal outcomes associated with polyhydramnios. Using a Population-Concept-Context framework, 18 open-access articles published between 2002 and 2024 were charted and synthesized thematically. The evidence covered diagnostic thresholds and severity, physiological mechanisms, maternal and fetal etiologies, structural anomalies, outcomes in idiopathic cases, and the role of genetic testing. Polyhydramnios should therefore be interpreted as a sonographic risk signal that prompts severity-based evaluation, targeted ultrasonography, selective genetic counseling, and proportionate delivery and neonatal planning.

Keywords: amniotic fluid, fetal anomalies, perinatal outcomes, polyhydramnios, scoping review.

1 Introduction

Amniotic fluid provides a medium that supports pulmonary development, fetal mobility, mechanical protection, and umbilical cord stability. In obstetric practice, an increased amniotic fluid volume is generally defined as polyhydramnios when the deepest vertical pocket (DVP) is at least 8 cm or the amniotic fluid index (AFI) is at least 24 cm [1, 2]. These thresholds are important because variations in definitions across studies may produce apparently inconsistent incidence rates, severity categories, and reported risks.

Physiologically, amniotic fluid volume reflects a balance between fetal urine production and pulmonary secretion on one side and fetal swallowing and intramembranous absorption on the other. Disturbance of either side of this balance can lead to fluid accumulation. Maternal diabetes may increase fetal diuresis, whereas gastrointestinal obstruction or neurological impairment may reduce fetal swallowing. Polyhydramnios should therefore be understood as a clinical warning sign. Ultrasonography reveals excess fluid, but the main clinical questions are what causes

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the excess fluid, whether additional fetal findings are present, and what degree of risk should be anticipated.

Many mild cases are idiopathic. Nevertheless, current evidence indicates that idiopathic polyhydramnios may still be associated with specific obstetric and neonatal outcomes [3]. A recent meta-analysis reported increased risks of several outcomes, including low Apgar scores, stillbirth, and perinatal mortality, although the certainty of evidence for some outcomes remained low and heterogeneity was substantial [4].

2 Methods

This manuscript used a scoping review design because the primary objective was to map concepts, types of evidence, and knowledge gaps rather than to calculate a pooled effect estimate. The review was organized as an academic scoping review comprising the research question, selection criteria, data charting, evidence mapping, thematic synthesis, limitations, and conclusions.

The research question was formulated using the Population-Concept-Context (PCC) framework. The Population comprised pregnant women with singleton pregnancies complicated by polyhydramnios and included populations from both high-income countries and low- and middle-income countries (LMICs) to reflect diverse care settings. The Concept included AFI/DVP definitions and severity, maternal-fetal etiology, structural fetal anomalies, genetic findings such as chromosomal microarray analysis (CMA) and copy number variants (CNVs), and perinatal outcomes. The Context covered antenatal care, fetal medicine, delivery, and neonatal evaluation. The core question was: how is polyhydramnios associated with fetal anomalies, genetic etiology, and perinatal outcomes, and how can this evidence be translated into pregnancy risk stratification?

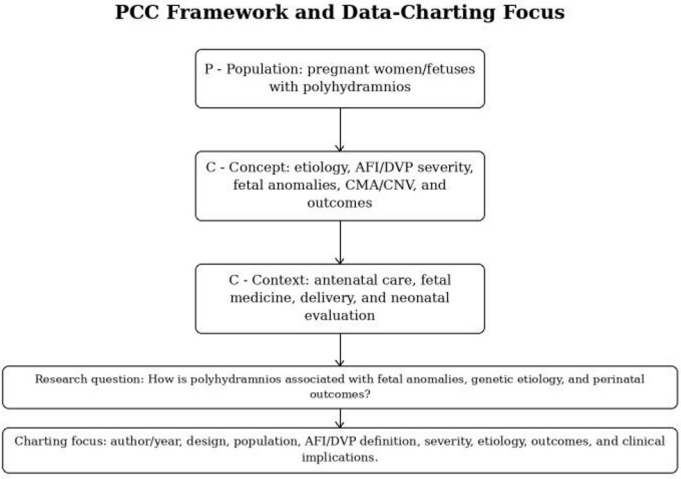


Fig. 1. PCC framework and data-charting focus.

2.1 Search Strategy and Data Charting

Initial sources were obtained from previously compiled polyhydramnios documents and supplemented by targeted searches of PubMed/PMC, ScienceDirect, SpringerLink, MDPI/BMC, and other relevant journal sources. Searches were restricted to open-access articles published from 2002 to 2024. Search terms included polyhydramnios, idiopathic polyhydramnios, fetal anomalies, congenital anomalies, chromosomal microarray, copy number variant, perinatal outcomes, neonatal outcomes, and prenatal diagnosis. Only English-language articles were included.

Data from the final 18 articles were extracted into a charting matrix containing author/year, source type, population, polyhydramnios definition or focus, reported outcomes, and clinical implications. Because the study designs were heterogeneous, synthesis was thematic and descriptive.

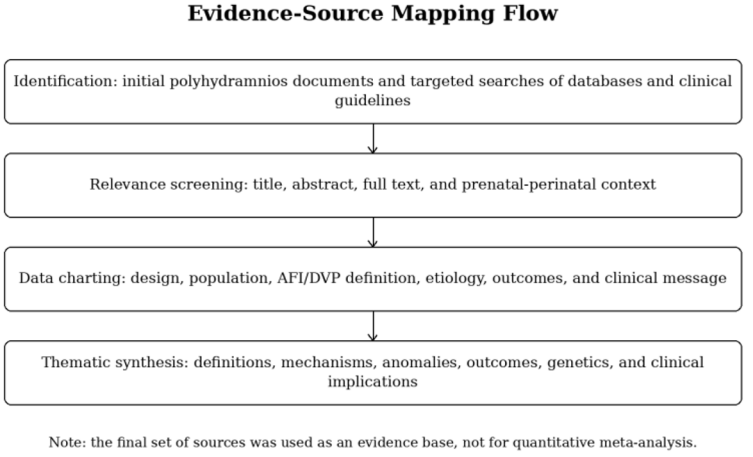


Fig. 2. Evidence-source mapping from identification to thematic synthesis.

2.2 Inclusion and Exclusion Criteria

Articles were included when they: (1) addressed polyhydramnios as a primary topic or as a separately analyzed variable; (2) reported mappable clinical data such as AFI/DVP definitions, severity, maternal-fetal etiology, structural fetal anomalies, prenatal genetic findings, or obstetric and neonatal outcomes; (3) examined women with singleton pregnancies from high-income or LMIC settings; (4) were original research articles or synthesis articles relevant to the research question; and (5) were available in full open-access English text and published between 2002 and 2024.

Exclusion criteria were: (1) opinions or commentaries without clinical data, articles without direct relevance to polyhydramnios, or sources that did not describe the population or outcomes; (2) sources addressing only oligohydramnios or other amniotic fluid disorders without separately mappable polyhydramnios data; (3) duplicate

findings or sources that only repeated recommendations; and (4) articles unavailable in full text or published outside the prespecified period.

3 Results

The mapped evidence showed that the clinical significance of polyhydramnios varies according to severity, gestational age at detection, isolated versus non-isolated status, and the presence or absence of maternal diabetes, structural anomalies, soft markers, or genetic abnormalities.

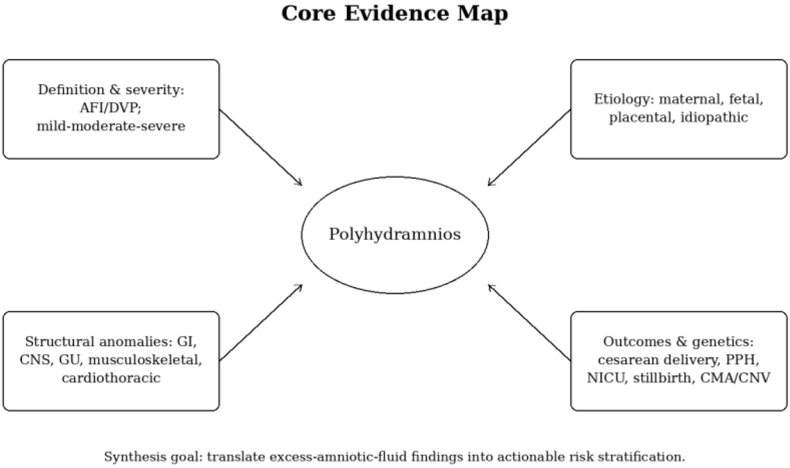


Fig. 3. Core evidence map used for the narrative synthesis.

Figure 3 summarizes four evidence domains that most strongly influence the interpretation of polyhydramnios: definition and severity, etiology, structural anomalies, and outcomes and genetics. This structure formed the basis of the charting matrix, allowing findings from diverse study designs to be interpreted within a consistent clinical framework [5, 6].

Table 1. Summary matrix of major evidence sources and clinical implications.

Source	Type	Main Finding	Clinical Implication
Liu et al., 2024	Retrospective genetic study	Six hundred singleton pregnancies with persistent polyhydramnios underwent CMA; outcomes were compared by isolated versus non-isolated status.	CMA is most informative when accompanying findings are present; isolated cases with negative CMA tend to have better outcomes.

Source	Type	Main Finding	Clinical Implication
Vanda et al., 2022	Prospective cohort	Idiopathic polyhydramnios was compared with normal pregnancies; cesarean delivery, postpartum hemorrhage, and neonatal respiratory distress were increased.	The label “idiopathic” does not automatically mean risk-free.
Pasquini et al., 2022	Retrospective cohort	Mild idiopathic polyhydramnios was associated with increased cesarean delivery; severe neonatal outcomes were not consistently increased.	Risk in mild cases should be interpreted proportionately rather than equated with severe disease.
Pagan et al., 2023	Retrospective cohort	Mild idiopathic polyhydramnios was evaluated for intrauterine fetal demise and pregnancy outcomes.	Supports follow-up discussions even for apparently isolated mild cases.
Gürel et al., 2024	Postnatal analysis	New postnatal diagnoses were more frequent in moderate-to-severe cases, including abnormalities not identified prenatally.	Neonatal evaluation is important, particularly in persistent or severe cases.
Lerner et al., 2024	Multicenter cohort	Labor induction in term isolated polyhydramnios was compared with a low-risk group.	Induction decisions should be individualized rather than based on the diagnostic label alone.
Bansal et al., 2018	Prospective observational study	The frequency of congenital anomalies increased with polyhydramnios severity; gastrointestinal anomalies predominated.	AFI severity can help determine the urgency of targeted ultrasonography.
Mohsin et al., 2023	Diagnostic cross-sectional study	Polyhydramnios was assessed as an indicator for detecting fetal anomalies on ultrasonography.	Moderate-to-severe cases warrant more comprehensive anatomic evaluation.
Parmar & Parmar, 2019	Observational AFI study	Abnormal AFI was associated with variations in pregnancy outcomes and perinatal morbidity.	AFI definitions and measurement should be applied consistently.
Özdemir et al., 2022	Maternal-fetal observational study	Maternal-fetal characteristics, etiology, and outcomes of polyhydramnios were clinically mapped.	Etiology is heterogeneous; evaluation should not stop at the label “excess fluid.”

Source	Type	Main Finding	Clinical Implication
Sagi-Dain et al., 2021	Retrospective CMA study	Seven hundred forty-two singleton pregnancies were analyzed for the association between polyhydramnios severity and CMA findings.	Genetic diagnostic yield is influenced by severity and accompanying findings.
Wu et al., 2022	CMA and karyotyping study	CMA/karyotyping identified genetic variants, particularly in non-isolated cases.	Supports selecting appropriate cases for genetic testing.
Lin et al., 2024	Retrospective genetic study	Isolated polyhydramnios cases undergoing amniocentesis were evaluated for the value of genetic testing.	Pretest counseling is important because yield is lower than in non-isolated cases.
Tsakmaki et al., 2024	Systematic review/meta-analysis	Apparently isolated polyhydramnios was analyzed for genomic abnormalities and the role of confirmed fetal phenotype.	Genomic findings may be identified, but interpretation depends on phenotype.
Kechagias et al., 2024	Systematic review/meta-analysis	Idiopathic polyhydramnios was associated with several maternal and neonatal outcomes; heterogeneity was high.	Follow-up should be proportionate and risk-based.
Kornacki et al., 2017	Severity-based observational study	Congenital anomaly frequency increased with severity; gastrointestinal anomalies were common.	Severity is a practical signal to search for structural anomalies.
Huri et al., 2023	Clinical review	Amniotic fluid disorders were linked to prenatal management and neonatal outcomes.	Connects prenatal findings with the need for neonatal evaluation.
Dashe et al., 2002	Anomaly prevalence study	Polyhydramnios was evaluated in relation to anomaly prevalence and sonographic detection.	Polyhydramnios has long been recognized as a clue prompting investigation for fetal anomalies.

Table 1 indicates that the strongest evidence for more intensive follow-up occurs in non-isolated, persistent, or moderate-to-severe cases. Truly isolated mild cases still require initial evaluation and follow-up, but monitoring intensity should be adapted to the clinical context to avoid overmanagement [5, 6].

4 Thematic Synthesis

4.1 Definition and Severity of Polyhydramnios

The most commonly used definitions are AFI ≥24 cm or DVP ≥8 cm. In clinical practice, polyhydramnios is classified as mild, moderate, or severe. This classification

is useful because the risks of structural anomalies, maternal symptoms, malpresentation, preterm birth, and referral increase with greater severity.

Table 2. Severity classification of polyhydramnios and clinical implications.

Severity	General Criteria	Clinical Meaning
Mild	AFI 24-29.9 cm or DVP 8-11 cm	Often idiopathic; baseline evaluation remains necessary.
Moderate	AFI 30-34.9 cm or DVP 12-15 cm	Risk of secondary causes increases; closer follow-up is required.
Severe	AFI ≥35 cm or DVP ≥16 cm	Higher risk of anomalies and complications; consider tertiary referral.

Table 2 emphasizes that severity is not merely a descriptive label but a graded risk marker. The higher the AFI/DVP, the greater the probability of identifying a secondary cause or structural anomaly, lowering the threshold for further evaluation and referral in moderate and severe cases [7, 8].

4.2 Physiological Mechanisms

The basic mechanism of polyhydramnios is fluid production that exceeds absorption or fetal swallowing capacity. In maternal diabetes, fetal hyperglycemia increases diuresis. Gastrointestinal obstruction or neurological disorders impair swallowing. In anemia, infection, or placental abnormalities, hemodynamic changes may also alter amniotic fluid volume.

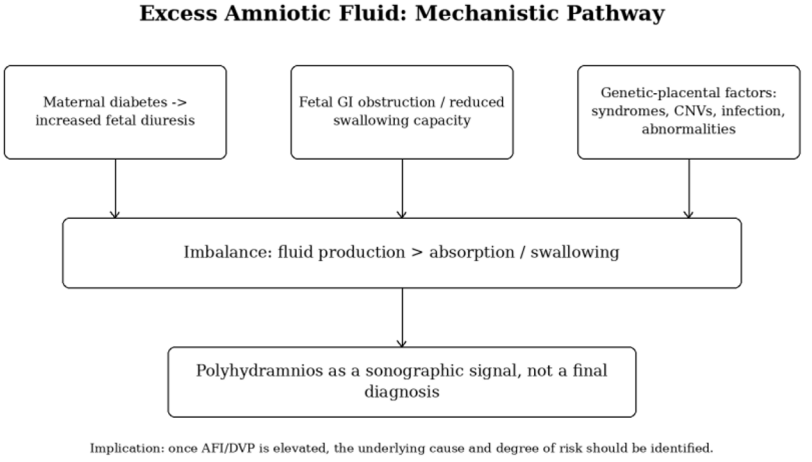


Fig. 4. Mechanistic pathway from polyhydramnios etiologies to the clinical signal.

4.3 Maternal-Fetal Etiology

Etiologies of polyhydramnios can be grouped as maternal, structural fetal, functional fetal, genetic, and idiopathic. The principal maternal factor is pregestational or gestational diabetes. Fetal factors include esophageal or duodenal obstruction, central

nervous system disorders, neuromuscular disorders, craniofacial anomalies, fetal anemia, congenital infection, and genetic abnormalities. Placental causes are less common but should still be considered in selected contexts.

Table 3. Etiologic groups that should be assessed in polyhydramnios.

Etiologic Group	Clinical Examples to Investigate
Maternal	Gestational/pregestational diabetes, alloimmunization, infection.
Structural fetal	Gastrointestinal obstruction, CNS anomalies, craniofacial anomalies, musculoskeletal anomalies.
Functional fetal	Swallowing impairment, neuromuscular disorders, fetal anemia.
Genetic	Aneuploidy, pathogenic CNVs, selected syndromes, particularly when the finding is non-isolated.
Idiopathic	No cause identified after initial evaluation; continued follow-up is still required according to context.

This grouping provides a systematic diagnostic framework so that investigation of the underlying cause does not stop at a single possibility, especially because some etiologies, such as maternal diabetes, are modifiable [9].

4.4 Structural Fetal Anomalies

Polyhydramnios may be an early clue prompting assessment for structural fetal anomalies. The most relevant systems include the gastrointestinal tract, central nervous system, genitourinary system, musculoskeletal system, cardiothoracic structures, and craniofacial region. Observational studies consistently show that anomalies are more frequently detected at higher severity, making the degree of polyhydramnios a practical guide to the urgency of further evaluation [8, 7, 10].

Table 4. Targeted ultrasound checklist in polyhydramnios.

Target Ultrasound Area	Clinical Rationale
Face, palate, and neck	Swallowing impairment or upper obstruction may be subtle.
CNS and spine	Neurological disorders may impair the swallowing reflex.
Gastrointestinal tract	Atresia or obstruction may reduce fluid absorption through swallowing.
Heart and thorax	Hemodynamic abnormalities may be associated with hydrops or anemia.
Extremities and fetal movement	Neuromuscular disorders may present as abnormal movement or tone.
Placenta and umbilical cord	Assess placental masses, hydrops, or other abnormal signs.

This structured approach increases the likelihood of identifying subtle anomalies, such as gastrointestinal obstruction or neuromuscular disorders, that may be missed on routine examination [10, 11].

4.5 Outcomes in Idiopathic Polyhydramnios

One important finding is that an idiopathic label does not automatically imply an absence of risk. A recent meta-analysis reported that idiopathic polyhydramnios may

be associated with preterm birth, labor induction, placental abruption, shoulder dystocia, cesarean delivery, postpartum hemorrhage, low Apgar scores, stillbirth, and perinatal mortality, although the certainty of evidence for several outcomes remained low and heterogeneous [4, 3].

Prospective and retrospective cohorts do not always show identical patterns. In some studies, the most prominent increases involved obstetric outcomes such as cesarean delivery and postpartum hemorrhage [12]. In others, attention shifted to neonatal respiratory distress, NICU admission, or new postnatal diagnoses [3, 13]. These differences indicate that outcomes should be interpreted according to severity, population, and the quality of prenatal evaluation.

4.6 Genetic Dimension: CMA and CNV

Genetic testing is most relevant when polyhydramnios is non-isolated, for example when accompanied by structural anomalies, soft markers, fetal growth abnormalities, or a relevant family history [14, 15]. Chromosomal microarray analysis (CMA) can detect copy number variants (CNVs) that are not visible on conventional karyotyping. However, CMA can also identify variants of uncertain significance, making pre- and post-test counseling essential [16, 6].

4.7 Clinical Risk Stratification

Clinical risk stratification integrates three elements: AFI/DVP severity, isolated versus non-isolated status, and maternal-fetal context. Truly isolated mild cases can be managed proportionately. By contrast, moderate-to-severe cases or those accompanied by anomalies require more intensive evaluation and delivery planning with neonatal support.

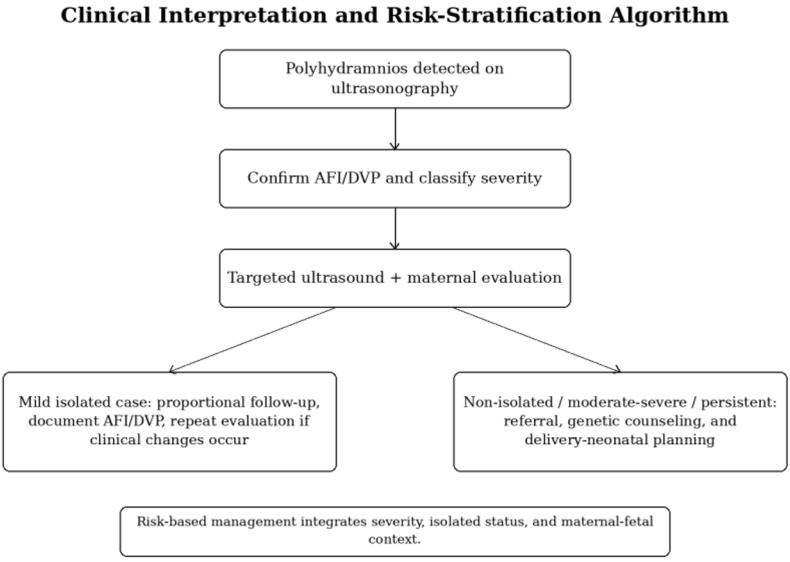


Fig. 5. Clinical interpretation and risk-stratification algorithm.

5 Discussion

The evidence map shows that polyhydramnios has two clinical faces. On one hand, many mild cases are idiopathic and do not inevitably result in adverse outcomes [17, 18]. On the other hand, excess amniotic fluid may be the entry point for diagnosing structural anomalies, maternal disorders, infection, anemia, or genetic abnormalities [5, 10]. The central clinical challenge is to distinguish cases that can be monitored from those requiring referral and in-depth evaluation.

A rational clinical approach is stepwise. First, confirm the definition and severity using AFI or DVP. Second, investigate the most likely causes, including diabetes, anatomical anomalies, hydrops/anemia, infection, and maternal history. Third, determine whether the case is truly isolated. Fourth, consider genetic counseling and CMA in the presence of structural anomalies, soft markers, moderate-to-severe polyhydramnios, or persistent polyhydramnios.

For mild idiopathic cases, the main message from the mapped evidence is balance. Not every patient should be managed as a severe case, yet some studies still show increased obstetric or neonatal risks in selected contexts. Initial evaluation should be comprehensive, documentation should be explicit, and follow-up should be adjusted to clinical changes.

6 Limitations

This manuscript is a scoping review based on evidence mapping for academic purposes, rather than a systematic review with a registered protocol and a fully reproducible comprehensive database search. Definitions of polyhydramnios, severity categories, study populations, and reported outcomes varied considerably across articles. The findings are therefore best interpreted as a conceptual evidence map and clinical direction rather than as final quantitative risk estimates.

7 Conclusion

Polyhydramnios is an important sonographic signal that may lead to the identification of maternal etiologies, structural fetal anomalies, genetic abnormalities, and adverse perinatal outcomes. AFI/DVP severity supports risk stratification, but clinical decisions should also consider whether the finding is isolated, persistent, or accompanied by other abnormalities. The most appropriate approach is to confirm the diagnosis, perform targeted ultrasonography, evaluate maternal-fetal etiologies, consider genetic counseling in selected cases, and develop delivery and neonatal plans proportionate to the level of risk.

8 Recommendations

1. Explicit documentation: Clinicians should document the AFI or DVP value and severity category clearly. Clear documentation prevents ambiguity between mild, moderate, and severe cases and provides a basis for determining monitoring intensity.
2. Targeted ultrasonography: Each diagnosis of polyhydramnios should ideally be followed by targeted ultrasonography because excess fluid may be an early sign of a fetal structural anomaly that was not apparent on routine examination.
3. Systematic maternal evaluation: Maternal diabetes and other maternal etiologies should be assessed systematically because diabetes is among the most common pathological causes and is potentially modifiable.
4. Genetic counseling: Genetic counseling and consideration of chromosomal microarray analysis should be offered for non-isolated cases or moderate-to-severe polyhydramnios because genetic diagnostic yield becomes more meaningful when additional findings are present.
5. Risk-based delivery planning: Delivery planning should be adapted to the level of risk and the availability of neonatal facilities because severe cases have a higher likelihood of anomalies and perinatal complications and may require multidisciplinary support.

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