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Severe Neonatal Anemia Caused by Fetomaternal Hemorrhage: A Case Report and Literature Review

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Case Report

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Abstract

Background

Fetomaternal hemorrhage(FMH) is a rare pregnancy disease with a high perinatal mortality rate and a propensity for delayed diagnosis. We should raise knowledge about this disorder in order to enhance the prognosis of perinatal infants.

Case presentation:

This article retrospectively analyzes clinical data from a case of severe neonatal anemia and severe asphyxia caused by FMH, summarizes the diagnostic and treatment approach, and reviews related literature.

Conclusions

The possibility of FMH diagnosis should be considered for pregnant women with decreased fetal movement and aberrant fetal heart rate monitoring in the third trimester, such as sinusoidal curve and fetal distress. The resuscitation effect of newborns is frequently weak, and the degree of pallor and asphyxia is inconsistent.

1. Background

Fetomaternal hemorrhage(FMH) refers to a clinical syndrome in which a certain amount of fetal blood enters the maternal circulation through damaged placental villi, causing varying degrees of fetal blood loss. It is a rare pregnancy disease with an incidence rate of approximately 0.1%-0.3% and a perinatal mortality rate of 33%-50% [1]. The onset of this condition is often insidious but may progress rapidly. Massive blood loss can cause severe anemia, intrauterine distress, and even stillbirth in fetuses. Severe neonatal anemia caused by FMH is rare. This article introduces a case with severe neonatal anemia and severe asphyxia caused by FMH. The aim of this article is to improve comprehension and diagnostic proficiency concerning FMH.

2. Case presentation

A 12-minute-old male patient was admitted to our hospital due to severe asphyxia. The patient with a 2890g birthweight was delivered at 39^{+4} weeks gestation by cesarean section. During the operative procedure, the amniotic fluid was found to be contaminated with meconium, while both the placenta and umbilical cord appeared normal. The newborn presented with pallor, bradycardia, absent crying, decreased muscle tone, and no spontaneous respiration. The resuscitation measures were given immediately including tracheal intubation, artificial positive pressure ventilation, chest compressions, and 1:10000 adrenaline given by trachea. Apgar scores recorded at 1, 5, and 10 minute were 1, 5, and 7 respectively. Following resuscitation, the child exhibited poor responsiveness, low muscle tone, irregular breathing, and pale skin. The patient was transferred to our neonatal intensive care unit.

Maternal history

A 28-year-old woman(para 1 and gravida 1) was admitted to our hospital because of intermittent tightness and swelling in the lower abdomen for 5 hours. The mother had hypercholesterolemia during pregnancy. An ultrasound revealed that the amniotic fluid index was 5.0 cm on the day before delivery, which indicates oligohydramnios. Fetal heart monitoring revealed significantly reduced baseline variability. Due to fetal intrauterine distress, we executed an urgent cesarean section, resulting in the delivery of a markedly pale male infant.

The blood gas analysis of the pediatric patient revealed: pH = 7.108, PCO2 = 24.9 mmHg, pO2 = 87mmHg, BE = − 21.7mmol/L, Lac=16mmol/L. Metabolic acidosis is considered to be related to perinatal asphyxia. Both the neonatal and maternal blood types were confirmed as Rh D type O. The complete blood count for the newborn reveals leukocytes of 18.97×10^9/L, a red blood cell count of 1.44×10^{12}/L, a hemoglobin level of 5.5g/dL, a platelet count of 265×10^9/L, a neutrophil count of 50.4×10^9/L, the reticulocyte count of 0.328× 10^12/L. Total bilirubin was 23.28umol/L. peripheral blood smears showed red blood cell size was uneven, with visible polychromatic red blood cells, oval red blood cells, and occasionally fragmented red blood cells. Liver function and myocardial enzymes were generally normal. The renal function showed a bicarbonate concentration of 8 mmol/L. CRP was 1mg/L. Blood culture test, TORCH and direct antiglobulin test were negative. The lactate dehydrogenase level was 751U/L. The chest X-ray and cranial MRI were normal. Cardiac ultrasound showed a slightly larger proportion of the right heart, a small amount of tricuspid regurgitation, pulmonary hypertension (68mmhg), and a small amount of pericardial effusion. The maternal hemoglobin levels before and after childbirth are 134 g/L and 94g/L respectively. This may be related to blood loss during cesarean section.

Hemoglobin electrophoresis of the pregnant woman: Hemoglobin acetate membrane electrophoresis: No H band was found, and hemoglobin A2: 0.026 (normal); Anti alkaline hemoglobin test: 0.049 (elevated, higher than the upper limit of 2%). K-B test was positive(figure 1). Pathological examination report of placenta: Localized congestion and scattered calcifications were visible in the placental tissue.

3. Treatment and follow-up

After admission, the treatment included ventilator assisted breathing, emergency blood transfusion to correct anemia, correction of acidosis, prevention of bleeding with vitamin K1, prevention of infection, correction of electrolyte imbalance, nutritional support. The blood transfusion was divided into three sessions, totaling 114 ml administered. After 3 days, the anemia condition gradually improved, with the red blood cell count increasing to 5.42×10^{12}/L and

hemoglobin increasing to 172g/L. Follow up half a month after discharge, there was no neurological sequelae. Follow up after 1 year and 2 months of discharge, the child's head circumference, height, and weight development were normal.

4. Literature review

We performed a comprehensive review of the existing literatures from 2021 up to 2026. A search on PubMed databases was performed to identify all studies that reported FMH. In this literature search, 'Fetomaternal Transfusion' was used as the subject term. (Transfusion Fetomaternal)OR (Transfusions Fetomaternal) OR (Hemorrhage Fetomaternal) OR (Fetomaternal Hemorrhage) OR (Fetomaternal Hemorrhages) OR (Hemorrhages Fetomaternal) was used as a free term. Inclusion criteria: (1) Case reports, case series, and retrospective cohort studies on FMH published between 2021 and 2026. (2) At least some of the clinical data in the literature can be extracted, including gestational age, maternal clinical symptoms, high-risk factors, neonatal hemoglobin, Apgar score, clinical outcomes, FMH diagnostic methods, and placental pathology. Exclusion criteria: (1)Non English literature; (2)The full text cannot be obtained; (3) Serious lack of literature and data; (4) Animal experiments, reviews, expert reviews, and letters without original case data will be excluded; (5) Insufficient diagnostic evidence of FMH. The clinical characteristics, maternal pregnancy status, diagnostic methods, complications, and prognosis of pediatric patients were fully examined in 24 articles with 30 cases(Table 1 and Fig. 2).

Table 1
Review of literature about Fetomaternal hemorrhage

No.	Author	Year	GA (w)	Maternal symptom	CTG pattern	Risk factors	Apgar score (1/5/10min)	Hemoglobin (g/dL)	Complications and outcomes	Diagn metho
1	Zheng Y et al.[2]	2023	37^{+5}	decreased fetal movement	loss of baseline variability	none	6–7	2.6	normal	HbF↑, AFP↑
2	Zheng Y et al. [2]	2023	36	none	recurrent variable decelerations with minimal variability	fetal growth restriction	9–9	5.2	normal	KBT
3	Derme M et al. [3]	2025	38^{+4}	decreased fetal movement	sinusoidal waves	none	5–6	4.3	Normal	MCA-I KBT
4	Farmer M et al. [4]	2024	35^{+6}	decreased fetal movement	NM	NM	NM	2.4	PPHN, discharged unevenfully	KBT
5	Sagawa K et al. [5]	2021	36	none	severe variable deceleration	dichorionic twin pairs with fetal growth restriction in one twin	3/7	5.7	NM	HbF↑
6	Smet C et al. [6]	2022	38^{+1}	decreased fetal movement	sinusoidal waves	none	IJ2/4/5j	3.1	refractory hypoxemia, PPHN,Transferred to a hypothermia center	HbF↑
7	Smet C et al. [6]	2022	29	decreased fetal movement	reduced variability	none	0/3/4	1.8	severe intraventricular hemorrhage, death	HbF↑
8	Smet C et al. [6]	2022	35^{+1}	decreased fetal movement	reduced variability, late decelerations	none	3/7/7	3.9	white matter lesions with neurological impairment	HbF↑
9	Smet C et al. [6]	2022	37^{+3}	None	reduced variability, late decelerations	none	8/8/9	4.8	discharged unevenfully	HbF↑
10	Liao S et al. [7]	2023	33^{+5}	decrease fetal movement	sinusoidal waves	none	2-7-8	2.8	normal	MCA-I AFP, H
11	Liao S et al. [7]	2023	33^{+5}	decrease fetal movement	sinusoidal waves	none	2-7-8	2.4	normal	MCA-I AFP, H
12	Gică N et al. [8]	2021	37^{+2}	decrease fetal movement	tachycardia, low variability, and chemoreceptor-type decelerations, absent	none	4–6	3.6	normal	MCA-I HbF
13	Li P et al. [9]	2024	36^{+4}	decrease fetal movement	reduced baseline variability	gestational diabetes mellitus and undifferentiated connective tissue disease	1-5-6	4.1	lung consolidation, neonatal pneumonia, and right pleural effusion, discharged unevenfully	NM
14	Lawrence AS et al. [10]	2026	34^{+3}	decrease fetal movement	baseline tachycardia, absent accelerations, decreased variability and provoked decelerations, sinusoidal waves	conceived by assisted reproductive technology, thrombocytopenia	2-3-7	2	right-sided grade I intraventricular haemorrhage, discharged unevenfully	MCA-I HbF↑

Abbreviations: gestational age(GA), cardiotocography(CTG), not mentioned(NM), Alpha-fetoprotein (AFP), persistent pulmonary hypertension(PPHN), middle cerebral artery–peak systolic velocity (MCA-PSV) ,Fetal hemoglobin (HbF), hematocrit(Ht), kleihauer betke test (KBT),

No.	Author	Year	GA (w)	Maternal symptom	CTG pattern	Risk factors	Apgar score (1/5/10min)	Hemoglobin (g/dL)	Complications and outcomes	Diagn metho
15	Monteiro S et al. [11]	2021	36^{+6}	decreased fetal movement	a possible isolated deceleration	none	NM	NM	fetal death in-utero.	KBT
16	Takano N et al. [12]	2024	35	none	sinusoidal waves	fetal growth retardation	1–5	3.7	normal	MCA-I HbF↑
17	Bubmann J et al. [13]	2026	35^{+1}	none	decreased fetal heart rate, unable to be measured	cervical insufficiency progressive growth discordance pregnancy-induced hypertension	0-0-0	2.2	death	HbF↑
18	Schepisi C et al. [14]	2023	NM	none	fetal intrauterine distress	short cervix	0-0-0	2.1	death	autop invest
19	Liggett CJ et al. [15]	2023	NM	none	recurrent variable fetal heart rate decelerations	none	4-6-7	0.127 L/L(Ht)	normal	KBT
20	Song S et al. [16]	2021	31^{+4}	anasarca and started to feel chest distress, palpitation, lethargy, and oliguria	none	fetal hydrops hypothyroidism,severe ascites,polyhydramnios	2-5-5	5.8	severe seroperitoneum mild hydrothorax, delayed development of the nervous system	AFP, HbF↑I PSV
21	Hao X et al. [17]	2025	38	none	none	hepatitis B virus carrier status	9–10	8.5	normal	HbF↑
22	Jin Y et al. [18]	2025	40	decreased fetal movement	severe variable decelerations	none	9–9	NM	normal	HbF↑
23	Liu T et al. [19]	2026	34^{+1}	decreased fetal movement	normal	none	6-8-8	4.5	death	MCA-I
24	Lv Z et al. [20]	2024	40^{+2}	decreased fetal movement	severe variable decelerations with absent fetal heart rate variability	none	8-8-8	4.7	normal	HbF↑
25	Signore F et al. [21]	2021	40	decreased fetal movement	reduced variability, deceleration and intermittent sinusoidal foetal heart	NM	3–4	3.5	death	HbF↑
26	Gao Y et al. [22]	2026	32^{+1}	decreased fetal movement	inadequate heart rate acceleration	gestational diabetes, polyhydramnios	6-7-9	5.9	normal	AFP, K HbF↑
27	Zeng W et al. [23]	2026	37^{+3}	none	sinusoidal fetal heart rate pattern	fetal growth restriction	1-/-/	2.8	normal	HbF↑
28	Zeng W et al. [23]	2026	37^{+3}	none	sinusoidal fetal heart rate pattern	fetal growth restriction	2-5-7	2.9	normal	HbF↑
29	Wang L et al. [24]	2025	24^{+2}	decreased fetal movement	disappearance of fetal heart rate variation	none	3–3	2.2	death	MCA-I AFP
30	Sun Y et al. [25]	2025	38^{+5}	decreased fetal movement	normal	fetal distress	8-8-8	4.6	normal	MCA-I HbF↑

Abbreviations: gestational age(GA), cardiotocography(CTG), not mentioned(NM), Alpha-fetoprotein (AFP), persistent pulmonary hypertension(PPHN), middle cerebral artery–peak systolic velocity (MCA-PSV) ,Fetal hemoglobin (HbF), hematocrit(Ht), kleihauer betke test (KBT),

5. Discussion and conclusions

Fetomaternal hemorrhage(FMH) is rare and has a high mortality rate. At present, the specific pathogenesis of the disease is not clear. It may be caused by various high-risk factors that lead to placental barrier damage, villus dysplasia or villus degeneration. FMH can occur at any stage of pregnancy. It often occurs during the third trimester or at the time of delivery. This is consistent with our literature review results. In our literature analysis, the smallest gestational age is 24 weeks, and the largest gestational age is 40 weeks. It is reported in the literature that 82% patients are idiopathic, while 18% are related to the following high-risk factors [26, 27]. During our literature analysis, 50% of medical records did not show any abnormalities during prenatal check-ups. The reported value is lower than the data, which is due to the small sample size. The related high-risk factors of FMH include: (1) umbilical cord and placental factors: such as single umbilical artery, placenta previa, placental abruption or implantation, vascular previa, placental chorioangioma or choriocarcinoma, umbilical vein thrombosis, etc; (2) Maternal factors: such as smoking, multiple births, maternal trauma, gestational hypertension, fetal maternal blood group incompatibility, autoimmune diseases, infections, etc; (3) Fetal factors, such as fetal malformation, twin transfusion syndrome, stillbirth, etc; (4) Iatrogenic injuries, such as amniocentesis, umbilical cord puncture, oxytocin induced labor, cesarean section, artificial placental dissection, etc. In addition, the strong contraction of the uterus during labor can damage the villi too. Although these risk factors are common in clinical practice, but FMH rarely occurs in these pregnant women. And, these high-risk factors are often absent in severe FMH. The condition of neonates with comorbidities is not more serious than that of other neonates. There are no special high-risk factors except oligohydramnios in this case. It is considered that the chorionic villi are damaged due to the intense contraction of the uterus in the third trimester of pregnancy, however, the exact reasons require further investigation.

The clinical manifestations of FMH are mostly nonspecific, which is related to fetal blood loss and blood loss speed. There are different definitions of massive blood transfusion. It varies from 20 to 150ml. When the blood loss reaches 80-150ml, the incidence of FMH is about 0.02%-0.1%[28]. Acute massive blood loss can lead to fetal hypovolemic shock, heart failure, and even death. It can cause sequelae such as neurological damage, cerebral palsy, and persistent pulmonary hypertension, and so on. It is reported that the incidence of neurological damage is between 4% and 18%[9]. Besides, there is report of 109 neonates with FMT suffering from neonatal mortality or long-term neurological morbidity that can be as high as 38%-50%[29]. In our literature review, there were 7 deaths (23%) and 5 cases with complications (16%). In the patient with chronic blood loss, the vascular resistance of all fetal tissues except placenta increases, and the increase of hydrostatic pressure will lead to tissue edema, fetal edema and fetal growth restriction. In this case, the patient suffers severe asphyxia, severe anemia, pericardial effusion, pulmonary hypertension, and a slightly larger proportion of right heart after birth, which is considered to be related to acute massive intrauterine blood loss. The clinical signs of FMH in pregnant women typically included decreased or absent fetal movement, sinusoidal waves suggested by fetal heart rate monitoring, and fetal edema. However, typical clinical manifestations are uncommon and fetal heart rate monitoring can display reduced baseline variation of fetal heart rate, late deceleration, and irregular waveform. This is consistent with our literature review. 83% of cases experienced abnormal fetal heart rate monitoring. 63% of pregnant women have symptoms of reduced fetal movement before delivery. And one pregnant woman suffered chest distress, palpitation, lethargy, and oliguria symptom. It is related to the hemolytic transfusion reaction caused by fetal blood entering the mother's body. Thus, when there are high-risk factors of FMH, pregnant women have decreased fetal movement and abnormal fetal heart rate monitors, it is important to be aware of the possibility of FMH. The placental pathological examination of FMH patients may be normal. It also can display placental pallor, placental microthrombosis, placental interstitial or postplacental hemorrhage, placental villus edema, local chorionic plate and intervillous hematoma formation, intravascular and intervillous nucleated red blood cells, etc. [30]. It may also be associated with chorioangioma and placental choriocarcinoma [31, 32]. Besides, there is the expression of vascular endothelial growth factor, CD34, and CD31 in capillary endothelial cells [2]. The pathological examination of the placenta in this report had no specificity for the diagnosis of FMH. In this literature review, 13 medical records described placental pathology, including 2 cases with pale placental surface, 1 case with placental edema, 5 cases with choriocarcinoma, 1 case with choriocarcinoma accompanied by chronic choriitis, 1 case with choriocarcinoma accompanied by hemorrhagic necrosis of placental villi, 1 case with focal villous edema, 1 case with placental chorangioma, and 1 case with chorionic septal cyst. This is consistent with the reported literature description.

The common laboratory testing methods of FMH include kleihauer betke test (KBT), hemoglobin(HB) electrophoresis, maternal blood alpha fetoprotein(AFP) detection, and B-ultrasound detection of fetal middle cerebral artery systolic blood flow peak detection. KBT is currently considered the standard method of diagnosis. This method is highly sensitive but prone to false positive results. In addition, the AFP of pregnant women will gradually increase during pregnancy, and gradually decrease after reaching the peak at 32 weeks of gestation, mostly less than 200 ng/ml[33]. A retrospective study showed that the median AFP of FMH patients was 3958.5 ng/ml, ranging from 1606 to 14330 ng/ml [34]. It is an important significance in the diagnosis of FMH. However, the measurement of maternal blood AFP needs to be compared with the value before delivery. Due to the lack of specificity in the clinical manifestations of FMH, most FMH medical records are retrospective diagnosis. The AFP was not sent for examination because the prenatal AFP value was not obtained in this case. Therefore, the clinical application of this method is limited. In addition, the diagnosis of FMH can be assisted by HB electrophoresis detection, which is economical, rapid and reliable. Under normal condition, the fetal hemoglobin(HBF) content in maternal blood is < 2%, and when FMH occurs, the HBF content will increase. In addition, the method of ultrasound can assist the diagnosis. By ultrasound, it is possible to detect the peak systolic blood flow rise and end diastolic reentry of the middle cerebral artery, while also detecting the fetal coronary blood flow[35]. If the peak value of fetal middle cerebral artery blood flow is greater than 1.5 times the median value, the possibility of severe anemia is high [29]. However, it has also been reported that this method has a high false positive rate [36]. In this case, the KBT and HB electrophoresis are used. The KBT and HB electrophoresis are positive which indicates the diagnosis of FMH. In our literature review. MCA-PSV plays an important role in the early detection of FMH and is a key tool for early identification. However, only a few cases were diagnosed prenatally through ultrasound examination. Due to our insufficient understanding of FMH, we did not undergo ultrasound examination or prenatal diagnosis. The K-B test is still an accurate method for diagnosis. Hemoglobin electrophoresis and AFP are widely used methods.

For the treatment of FMH, fetuses with small gestational age should be treated with intrauterine blood transfusion. The indication of intrauterine blood transfusion for gestational week under 32w is hematocrit < 0.3. The maximum amount of blood transfusion is (gestational weeks − 20) × 10ml according to fetal hematocrit, weight and gestational age. The maximum amount of blood transfusion is not more than 30ml/kg each time. It is appropriate to use 5-6ml/kg each time. The hematocrit after blood transfusion is close to or more than 0.4 or fetal Hb 150g/l as the standard for the end of blood transfusion. Pregnancy should be terminated when the fetus is mature. If the gestational age is greater than 35 weeks, the pregnancy should be terminated by cesarean section in time after diagnosis.

Early diagnosis of FMH is challenging because of the non-specific characteristics of FMH. For pregnant women with reduced fetal movement and abnormal fetal heart rate monitoring in the third trimester, such as sinusoidal curve and fetal distress, the possibility of FMH diagnosis should be considered. Newborns can present with severe asphyxia, skin pallor, anemia, shock, heart failure, etc. The resuscitation effect of newborns is often poor, and the degree of pallor and asphyxia is inconsistent. Early diagnosis and treatment can reduce the mortality rate with this disease.

Abbreviations

Fetomaternal hemorrhage(FMH), hemoglobin(HB), Abbreviations: gestational age(GA), cardiotocography(CTG), not mentioned(NM), Alpha-fetoprotein (AFP), persistent pulmonary hypertension(PPHN), middle cerebral artery–peak systolic velocity (MCA-PSV) ,Fetal hemoglobin (HbF), hematocrit(Ht), kleihauer betke test (KBT),

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the patient family for publication of this case report.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

There is no conflict.

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Authors' contributions

L and Q: data curation, methodology, writing-original draft. J: writing-review and editing

D：data curation, methodology, supervision, writing-review and editing. All authors read and approved the final manuscript.

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Figures

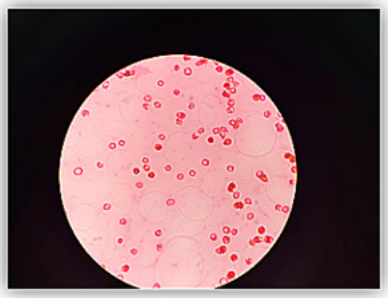


Figure 1

K-B test was positive

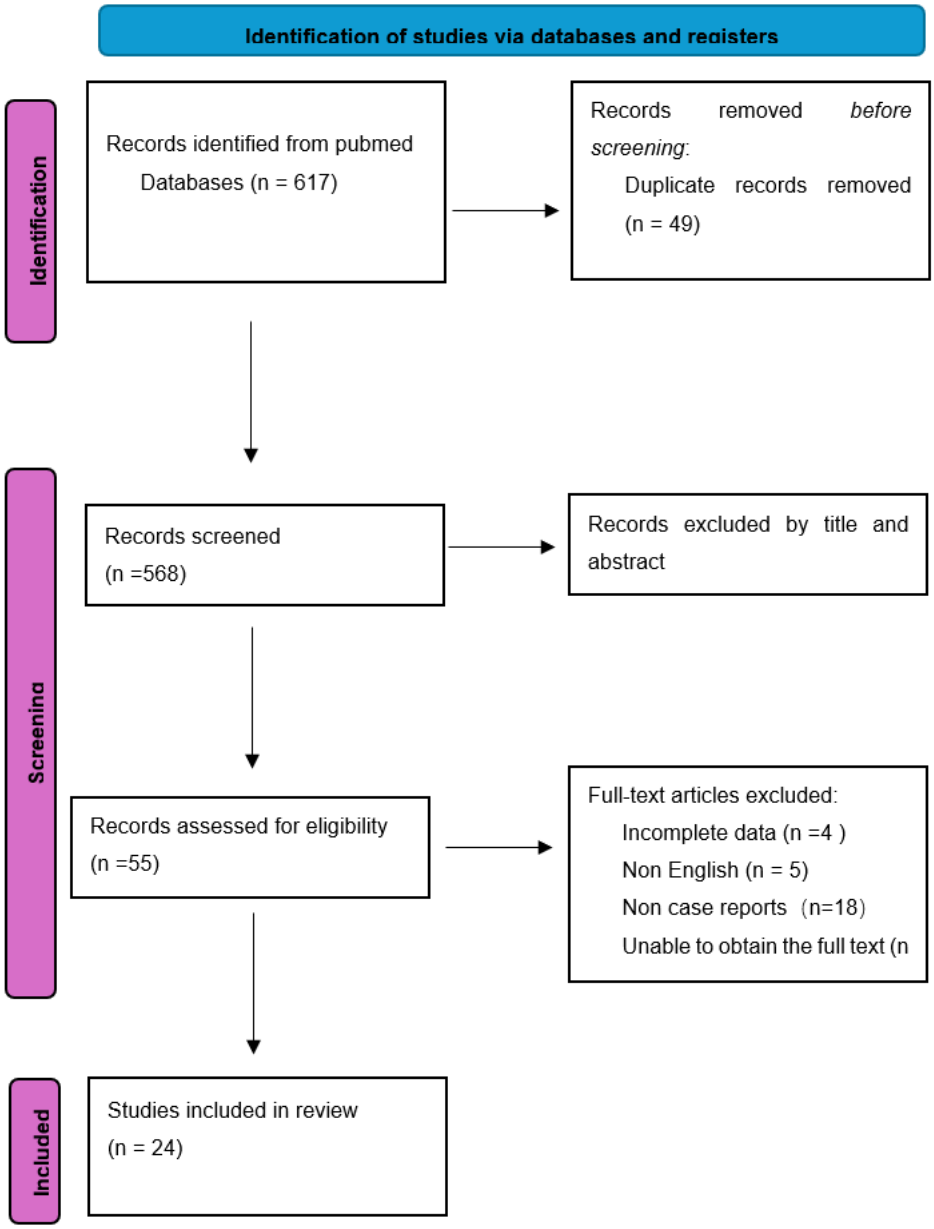


Figure 2

Literature Screening Flowchart