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RESEARCH ARTICLE

NON-IMMUNE HYDROPS FETALIS SECONDARY TO PARVOVIRUS B19 INFECTION WITH FAVORABLE PERINATAL OUTCOME: CASE REPORT AND REVIEW OF THE LITERATURE

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Abstract

Parvovirus B19 infection is an uncommon yet feared cause of fetal complications, particularly anemia and non-immune hydrops. We present the case of a 23-year-old woman who acquired a primary infection during pregnancy; her fetus developed severe anemia that required two intrauterine transfusions, culminating in a favorable neonatal outcome. A literature review is included to contextualize the diagnostic and therapeutic challenges.

Introduction: -

Parvovirus B19 is a DNA virus belonging to the family Parvoviridae. The virus was first identified in 1975 in a blood-donor bag labeled “B19” [1]. It is transmitted principally via respiratory secretions, blood products, or transplacentally. Maternal infection during pregnancy is often asymptomatic, yet it can lead to severe fetal complications, notably profound anemia, non-immune hydrops fetalis, and even intrauterine fetal death (IUFD) [2].

Case Report

A 23-year-old gravida 2 para 1 woman (blood type A Rh-positive) with an unremarkable medical history and a husband of blood type O Rh-positive was referred at 23 weeks’ gestation for suspected fetal hydrops. Obstetric ultrasonography revealed massive fetal ascites and an elevated middle cerebral artery peak systolic velocity of 1.63 multiples of the median (MoM), consistent with moderate-to-severe fetal anemia. Maternal serology demonstrated parvovirus B19–specific IgM antibodies, confirming a recent infection. A first intrauterine transfusion (IUT) of 30 mL was performed, resulting in clear sonographic improvement; a second 20 mL IUT was administered at 27 weeks’ gestation. The subsequent course was uneventful, and at 37 + 3 weeks an elective Caesarean section delivered a growth-appropriate neonate (birth weight 2.9 kg; Apgar scores 10/10 at 1 and 5 minutes). Six-month follow-up showed normal growth and development.Table 1 summarises the transfusion parameters and hematological response.

Discussion:-

Parvovirus B19 infection is common, with a sero prevalence estimated at 50 %–75 % in adults, yeti toften remain sunder diagnosed during pregnancy. It is transmitted chiefly via respiratory secretions, blood products, or transplacentally, and affects roughly 1 %–5 % of pregnant women, with a vertical transmission risk to the fetus of 17

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%–33 % [3]. Duringepidemicperiods, thisrisk can beeven higher, particularly in professionally exposed women such as teachers or mothers of youngchildren, in whom seroconversion rates mayreach 16 % [4].

Ery the main fectiosum, or fifth disease, is the typical presentation in children, characterised by moderate fever, malaise, and the pathognomonic “slapped-cheek” facial rash, followed by a maculo papular eruption on the trunk and limbs. In adults, especially pregnant women, infection is often a symptomatic (in 30 %–50 % of cases) but may present with an influenza-like syndrome, a “megalo-erythema”–type rash, and, above all, arthralgia of the hands, wrists, or kneesthat can bedisabling. Rare manifestations includemyocarditis and heartfailure, aplasticcrisis in the context of chronicanaemia, and complications in immunocompromisedindividuals [5].

Duringpregnancy, parvovirus B19 infection generallydoes not compromise maternalhealth, yetit can have significantfetalconsequences. The vertical transmission rate isestimated at 17 %–33 %, with the highestriskbetween 9 and 20 weeks of gestation [6]. Most infectedfetuses, however, recoverspontaneouslywithoutsequelae.

The principal complications are:

1. **Spontaneousmiscarriage:** the riskis 13 % when infection occursbefore 20 weeks’ gestation, versus only 0.5 % thereafter. This differencemaybeattributable to multivisceral lesions, even in the absence of anemia or hydrops. [6]

2. **Non-immune fetalhydrops:**thisis the mostovert manifestation of congenital infection. The riskdepends on the gestationalage at the time of infection:
   - Lessthan 5–10 % if infection occursbefore 12 weeks’ gestation
   - About 5 % between 13 and 20 weeks’ gestation
   - Lessthan 1 % after 20 weeks’ gestation [7]

The mechanisms involved include severe fetal anemia resulting from infection of erythroid precursors, compounded by the short lifespan of fetal red blood cells during hepatic hematopoiesis. Additional causes comprise fetal viral myocarditis, which can progress to heart failure, as well as hepatic involvement attributable either to direct hepatocyte destruction or to indirect toxicity from iron (hemosiderin) deposition. [7]

Diagnosis relies on a combination of ultra sound f indings—ascites, edema, effusions, placentomegaly—together with Doppler assessment of the middle cerebralartery (MCA) peak systolic velocity, where a value > 1.5 multiples of the median (MoM) is indicative of moderate-to-severeanemia, and detection of maternal anti-parvovirus B19 IgM antibodies. Fetal confirmation can beobtained by PCR on eitherfetalblood or amnioticfluid [3][5][7].

Management iscentered on intrauterine transfusion (IUT), whichrapidly corrects the anemia and markedly improves prognosis. Reported perinatal survival rates after IUT range from 67 % to 85 %, while spontaneousre gression of hydropsoccurs in approximately 30 %–34 % of cases whenanemiaisonlymoderate [7]. In our patient, two transfusions were required and achieved both clinical and ultra sonographic improvement, culminating in a favorable outcome. Long-term follow-up of affected infants has revealed no significant neuro developmenta lsequelae, provided that intervention istimely.

At present, no licensed vaccine against parvovirus B19 exists. Preventive measures there fore depend on strict hygiene practices and avoidance of high-riskexposures in pregnantwomen. Serologic screening at the onset of pregnancy may be considered for women at particularly high risk of exposure, although routine testingis not yetuniversallyrecommended [5].

Conclusion:-

Parvovirus B19 infection during pregnancy is generally benign but can lead to serious complications, such as fetal anemia or hydrops. Diagnosis is based on serologic testing and middle cerebral artery (MCA) Doppler assessment. Intrauterine transfusion (IUT) markedly improves the prognosis when anemia is confirmed. Appropriate and timely management helps ensure a favorable fetal outcome.

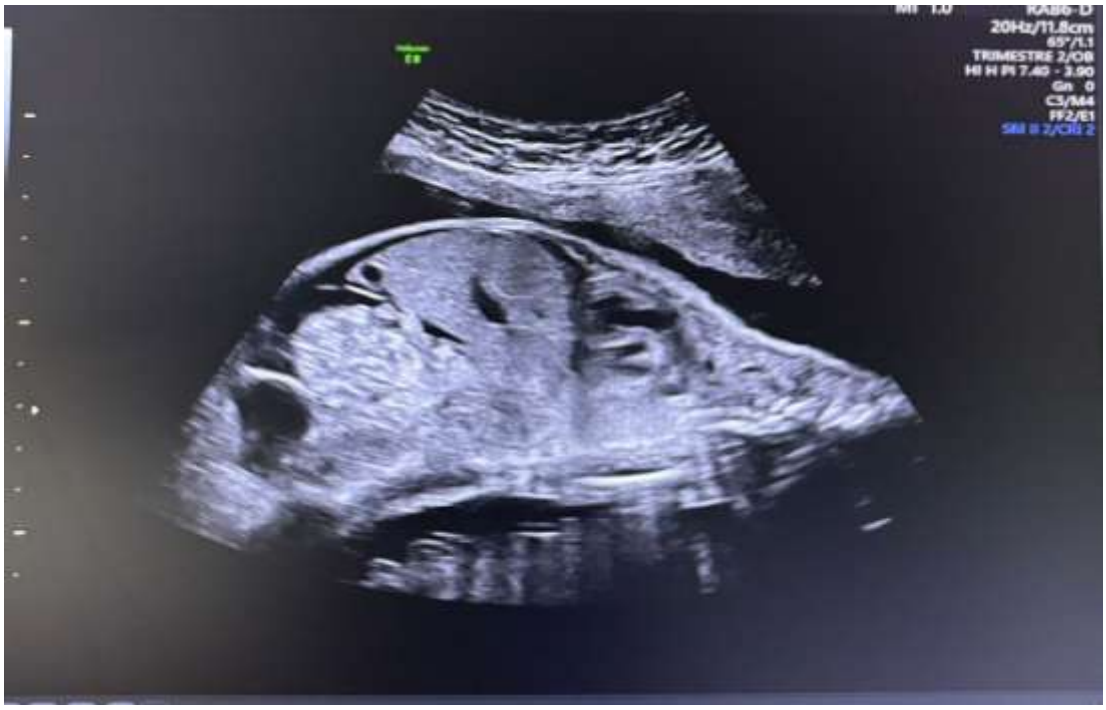

Figure 1:-Ultrasound image at 23 weeks of gestation showing fetal ascites.

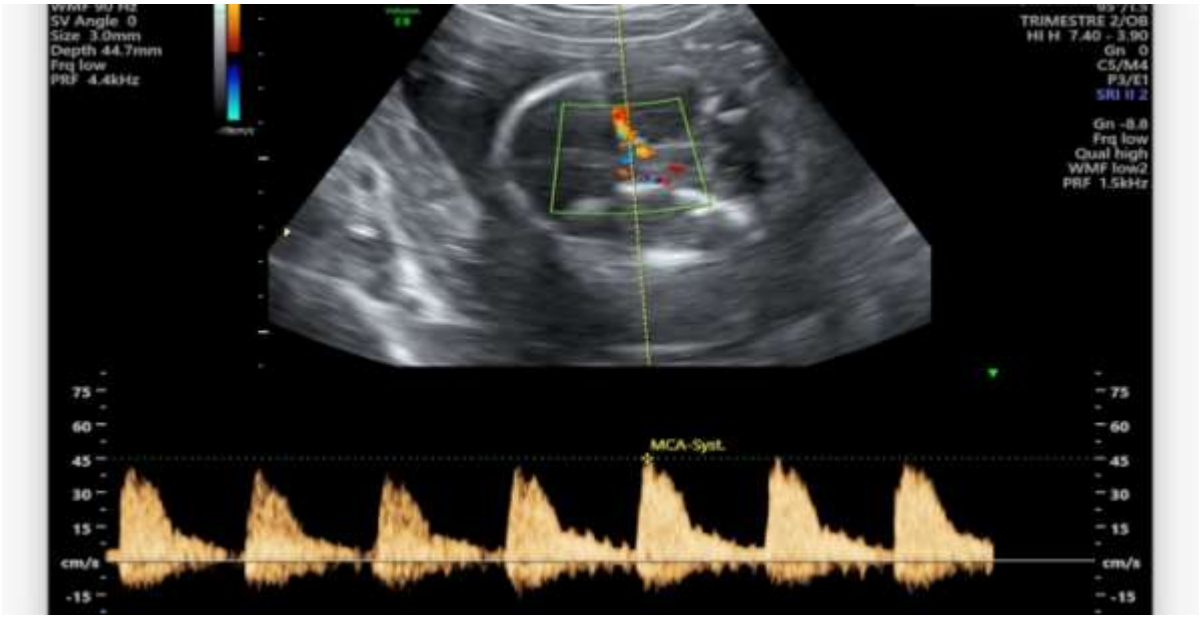

Figure 2:Middle cerebral artery Doppler velocimetry at 23 weeks gestation showing a peak systolic velocity of 1.6 MoM.

	Age gestationnel	Hb et Hte du sang fœtal en Pré transfusionnel		Volume transfusé	Hb et Hte du sang fœtal en post transfusionnel	
		Hb g/dl	Hte%		Hb g/dl	Hte%
1 TIU	23SA	10.3	31.5	30cc	12.8	39.1
2 TIU	27SA+6jr	8	23	25cc	13.4	53

Figure 3:Summary of the twointrauterine transfusions performed.

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