



Utility Of Placental Examination In Unraveling Intrauterine Infection – Foetal Autopsy Analysis

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BACKGROUND

- The integrity of the foetal-placental-maternal unit is the key to the prevention of intrauterine infection(1).
 - In most instances, infections leave “footprints” in placenta either directly or indirectly.
 - Histopathologic examination of Fetal autopsy including the placenta enables initial diagnosis of infection and subsequent correlation with adverse foetal outcomes(2).
- Here, we present a case series where placental pathology played a vital role in diagnosis of intrauterine infections and offer insight into mechanism of fetal injury and outcome.**

MATERIALS & METHODS

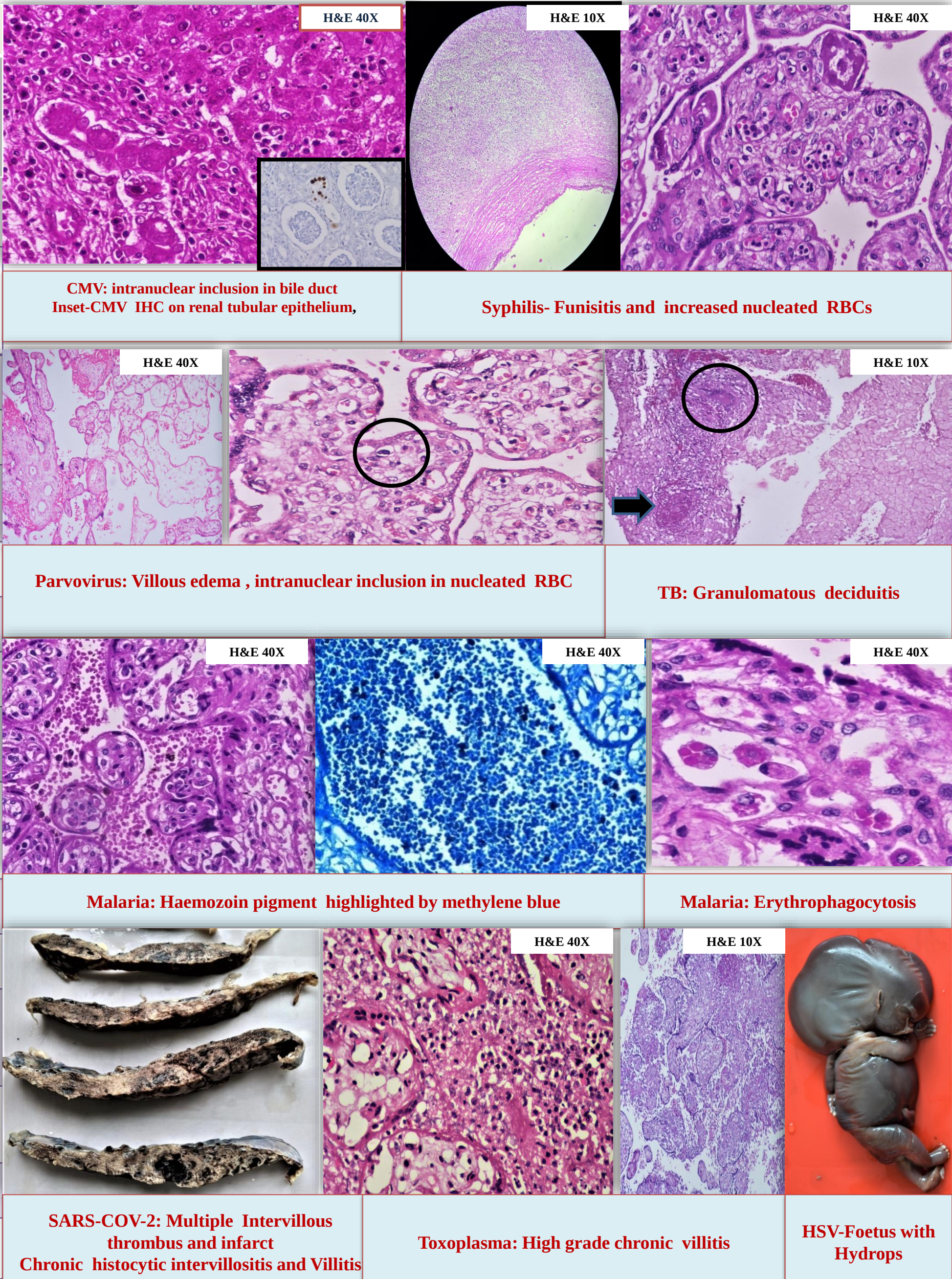
- Among the 621 fetuses and placentas analyzed over a period of 10 years, 41 case (including 2 twins) were identified with intrauterine infections.
- Photography, foetogram and standard autopsy protocol were followed after getting the informed consent.
- The lab parameters and pathological findings were analyzed.

AIMS & OBJECTIVES

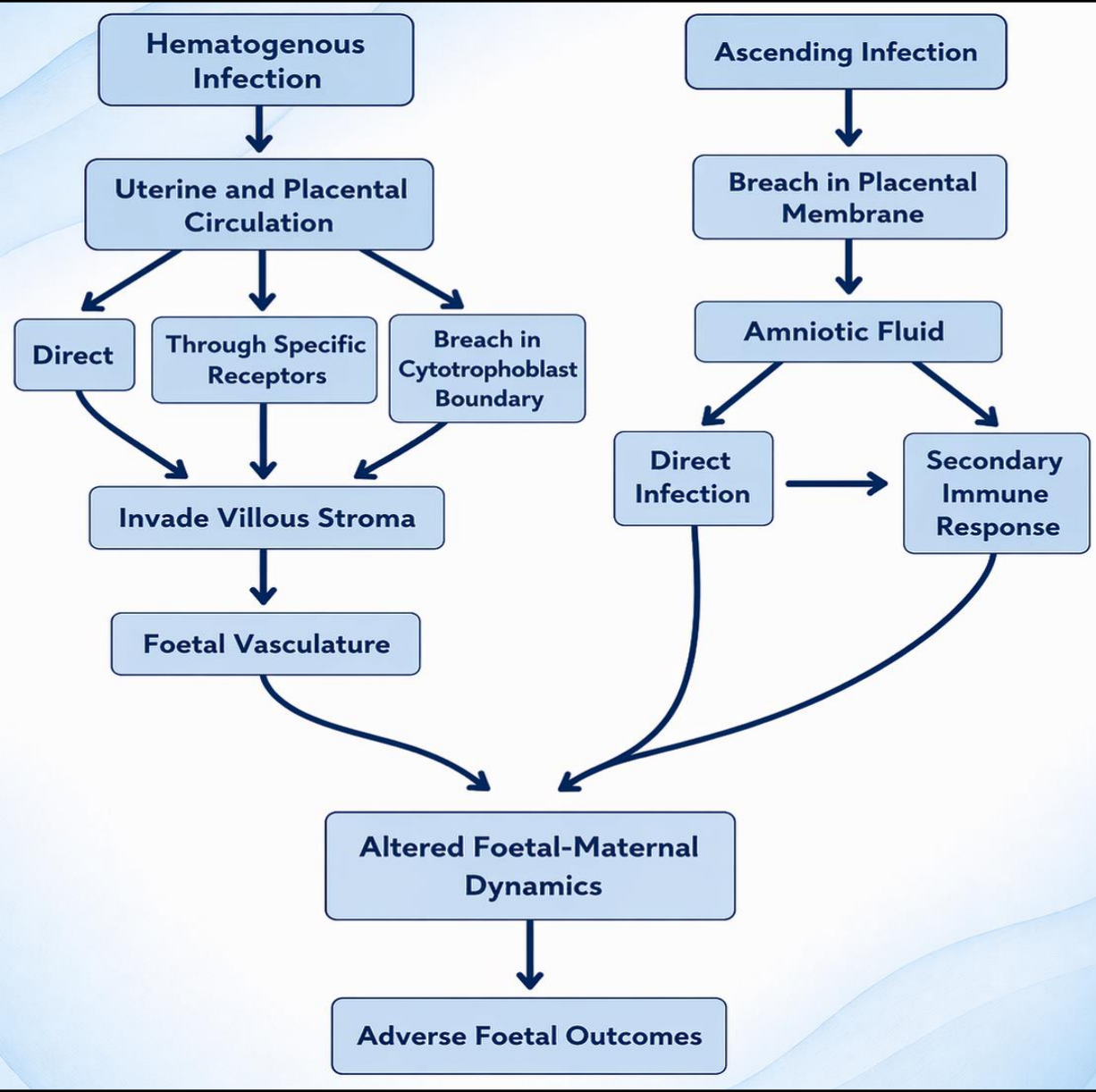
- To determine the utility of placental examination in specific intrauterine infections.
- To analyze foetal and placental findings in these infections, emphasizing their crucial role in accurate diagnosis and clinical correlation.

RESULTS

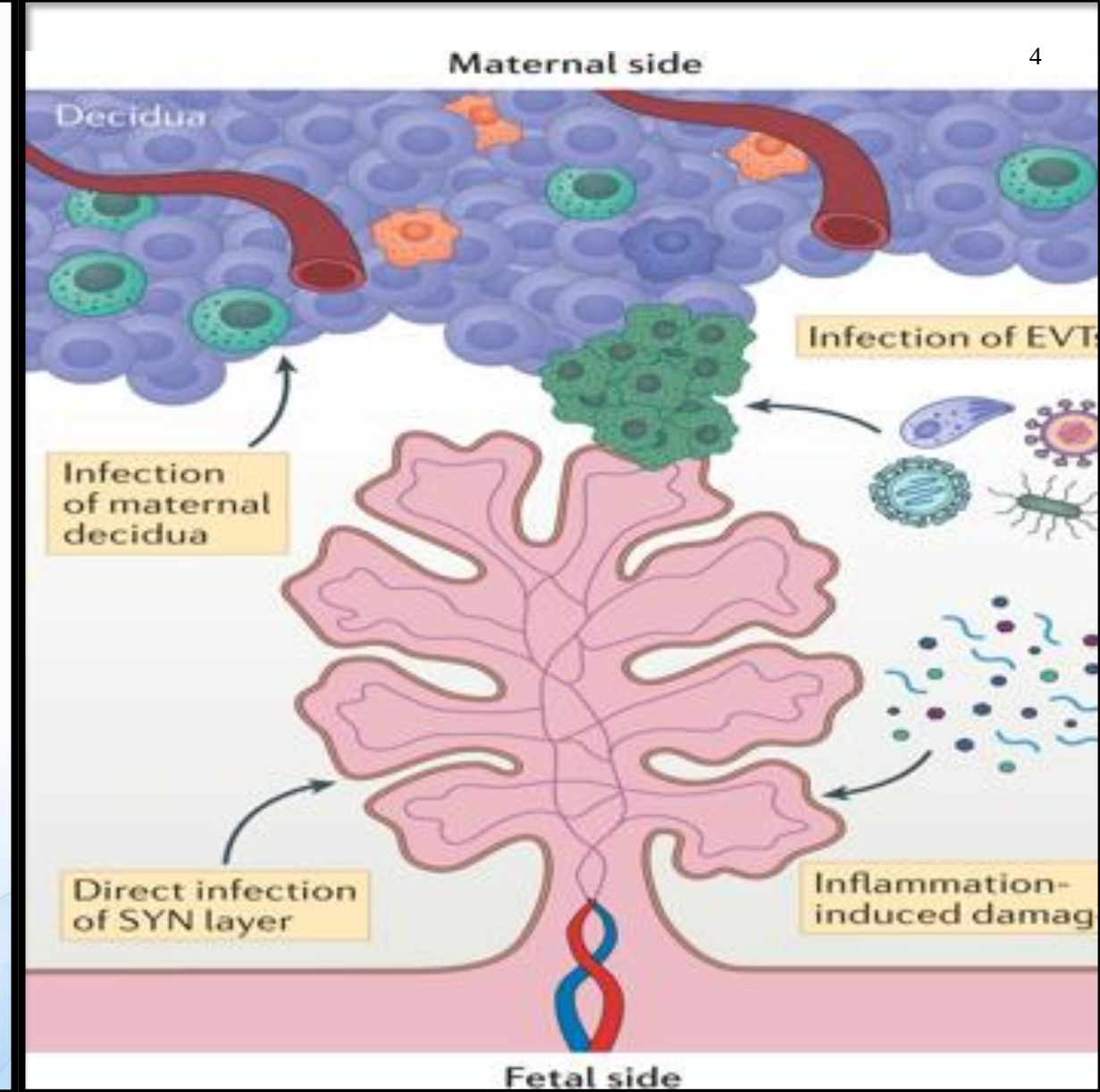
S. NO	TYPE OF INFECTION/ ORGANISM		AGE OF GESTATION	MATERNAL LAB PARAMETER	COMMON FOETAL FINDINGS	COMMON PLACENTAL FINDINGS
1	VIRAL (19)	PARVO (4)	20-34 WK	RT-PCR +	Hydrops fetalis	Marked villous oedema, intranuclear inclusion in nucleated RBCs, foetal malperfusion.
		SARS-COV2 (3)	19- 34 WK	RT-PCR+	Intraventricular haemorrhage	High grade Villitis, CHIV, maternal and foetal vascular malperfusion.
		CMV(5)	17-22 WK	IGG +	Hepatosplenomegaly, viral inclusions in most of the organs(brain, lung, liver, Kidney	Foetal-maternal malperfusion Lymphohistiocytic Villitis and Intravillous inclusions.
		HIV(2)	30-35 WK	ELISA +	Neural tube defects, hydronephrosis	Mild lymphohistiocytic villitis,chorangiosis
		DENGUE(2)	21 WK	NS1 ANTIGEN POSITIVE	Intraparenchymal hemorrhage in liver, spleen and prominent megakaryocytes	Sub amniotic hemorrhage.
		HSV(1)	34WK	PCR IG M +	Hydrops fetalis with cardiomegaly	Placentomegaly, villous stromal cells with intranuclear inclusions.
		VARICELLA (1)	32WK	BLISTERS AROUND THE OS	Foetal growth restriction	Plasma Cell deciduitis and Chronic chorionitis.
		ZIKA(1)	28+3 WK	SUSPECTED	Microcephaly,craniosynostosis,ventriculomegaly, dysgenesis of corpus callosum and neuronal migration disorder with lymphohistiocytic infiltration	Villous edema with Avascular villi.
2	BACTERIAL (19)	SYPHILIS(7)	34-40 WK	VDRL-reactive	Hydrops foetalis, Cardiomegaly and hepatosplenomegaly	Acute chorioamnionitis, funisitis,foetal and maternal vascular malperfusion.
		TB(5)	22-30 WK	AFB++	Hydrops foetalis	Cord with strictures, foetal vascular malperfusion, granulomas.
		LISTERIA (6)	20 WK	ELEVATED CRP, LEUCOCYTOSIS	Hepatic microabscesses	Acute villitis, intervillitis, abscess.
		NOCARDIA (1)	22WK	WEAK AFB +	Pulmonary hypoplasia with intra-alveolar filamentous colonies	Decidual necrosis, decidual vasculopathy, Foetal vascular malperfusion.
3	PARASITIC /PROTOZOA (3)	MALARIA (1)	23 WK	THICK AND THIN SMEAR	Facial dysmorphism, Ventriculomegaly	Villitis with erythrophagocytosis, haemozoin pigment.
		TOXOPLASMA (2)	30WK	IGM- +, IG G PCR	Hydrocephalus, Hepatosplenomegaly	Lymphohistiocytic Villitis, plasma cell Villitis.



POSSIBLE MECHANISM OF TRANSMISSION AND IMPACT:



DISCUSSION



Distribution of Diagnosed Cases Based on Clinical and Histopathological Correlation

Total cases evaluated	Clinically diagnosed cases with specific histopathology findings	Histopathology only diagnosed cases (clinically unsuspected)	Clinico-pathologically confirmed cases
41	15	16	10

By examining the placenta histopathologically, pathologists can identify subtle signs of infection, such as inflammation, microorganisms, or tissue damage which is essential to investigate spontaneous abortion, hydrops, stillbirth, prematurity ,FGR ,congenital anomaly and a sick neonate.

SUMMARY AND CONCLUSION

- The cases presented here are incredibly rare. The diagnosis often missed due to low index of suspicion.
- Our study reinforces that standard autopsy including placentas should ideally be an essential part of fully investigating foetal loss , still births, congenital malformation and suspected/unsuspected infections.

ABBREVIATIONS

HIV-Human Immunodeficiency Virus; CMV-Cytomegalovirus ,HSV-Herpes Simplex Virus; SARS-CoV-Severe Acute Respiratory Syndrome-COVID, TB-Tuberculosis , CNS-Central Nervous System, FGR-Foetal Growth Restriction

References:

1. Horn LC, Röse I. Placental and foetal pathology in intrauterine viral infections. Intervirology. 1998;41(4-5):219-25
2. Roberts, D. (2017). Placental Infections. In R. Redline, T. Boyd, & D. Roberts (Eds.), *Placental and Gestational Pathology* (Diagnostic Pediatric Pathology, pp. 115-136). Cambridge: Cambridge University Press.
3. Benirschke K, Kaufmann P, Baergen RN. Pathology of the Human Placenta. 5th ed. Pathology. New York: Springer Science+Business Media, Inc.; 2006. p. 1–1050.
4. Megli, C.J., Coyne, C.B. Infections at the maternal–fetal interface: an overview of pathogenesis and defence. *Nat Rev Microbiol* 20, 67–82 (2022)

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