Molar Pregnancy with Viable Foetus Progressed to Term – Case Report

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INTRODUCTION

The incidence of mole with coexisting normal foetus is 1 in 100,000 pregnancies.[1] Partial hydatidiform mole differs from complete mole by its focal distribution, its slower transformation, the presence of an embryo/foetus and most often with the triploid karyotype (Figure 1).

Diagnosis could be established by ultrasound which most often reveals greatly enlarged placenta relative to the size of uterine cavity, cystic spaces within the placenta, an amniotic cavity either empty/amorphous foetal echoes, well formed but growth retarded foetus either dead or alive.[2]

We describe a case of molar change in a placenta that was associated with a normal foetus and how the pregnancy progressed.

Case Report

A 25 year old primigravida who conceived spontaneously on folic acid supplementation had uneventful first trimester with no hyperemesis or vaginal bleeding. Viability scan was done at 7–8 weeks gestational age showed a single gesta-

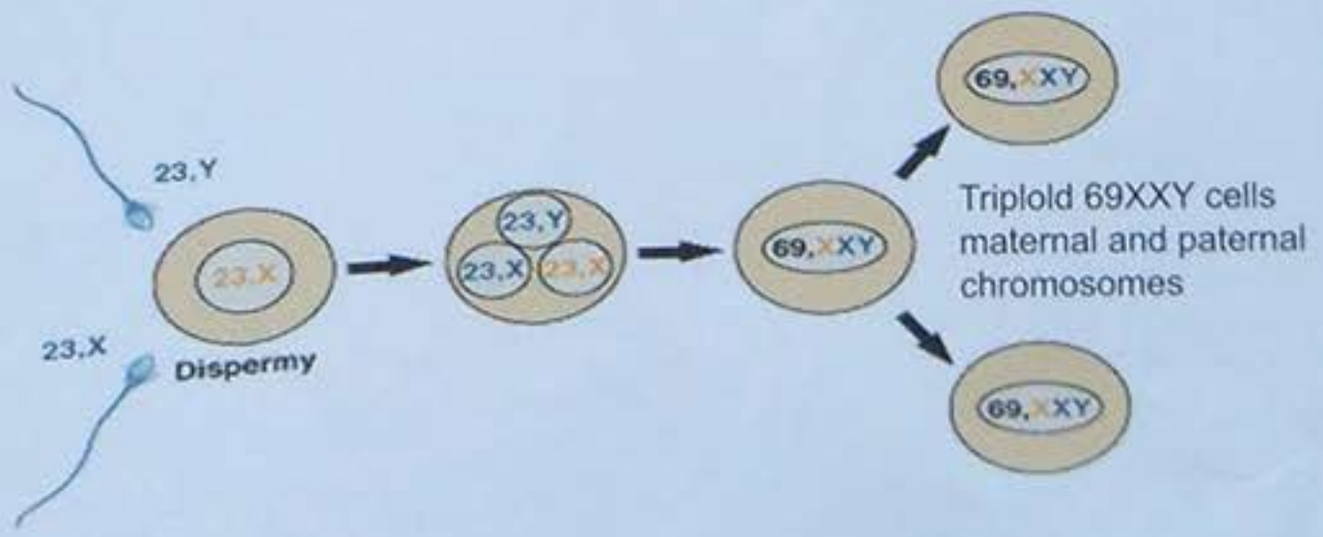

Figure 1. Formation of partial mole

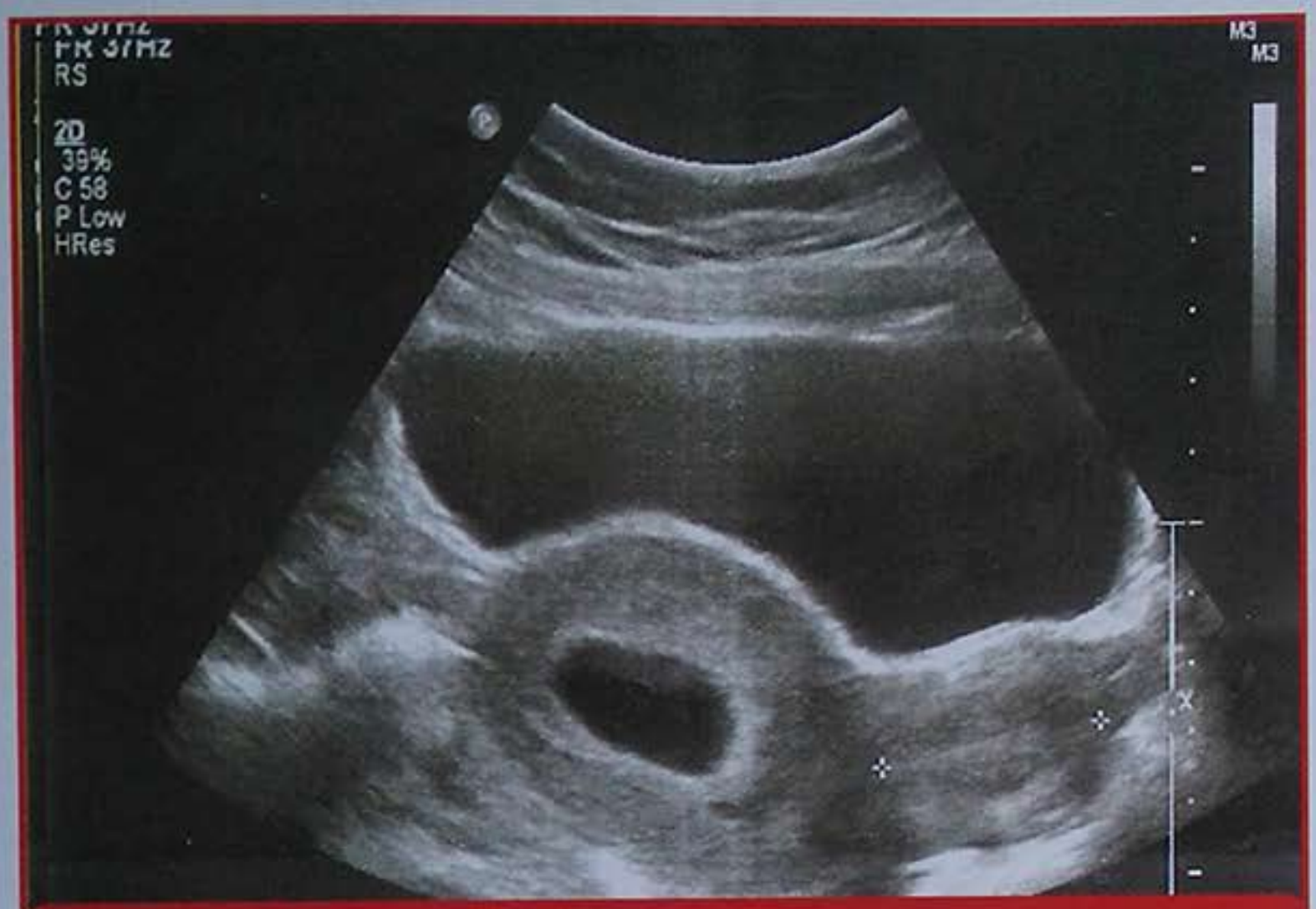

Figure 2. Viability scan

tional sac, CRL 1.1 cms, corresponding to 7.3 weeks good cardiac activity, perigestational sac haemorrhage of size 1.4 cms seen just above the internal os (Figure 2). The routine ANC profile was within normal limits.

At 12+ weeks NT scan was done which showed molar tissue at the right fundal end of uterine cavity of the volume 48 cc ,with a healthy live foetus of 12.5 weeks gestational age NT was 1.4 mm, nasal bone was visible gross morphology of the foetus appeared normal, liquor was adequate (Figure 3).

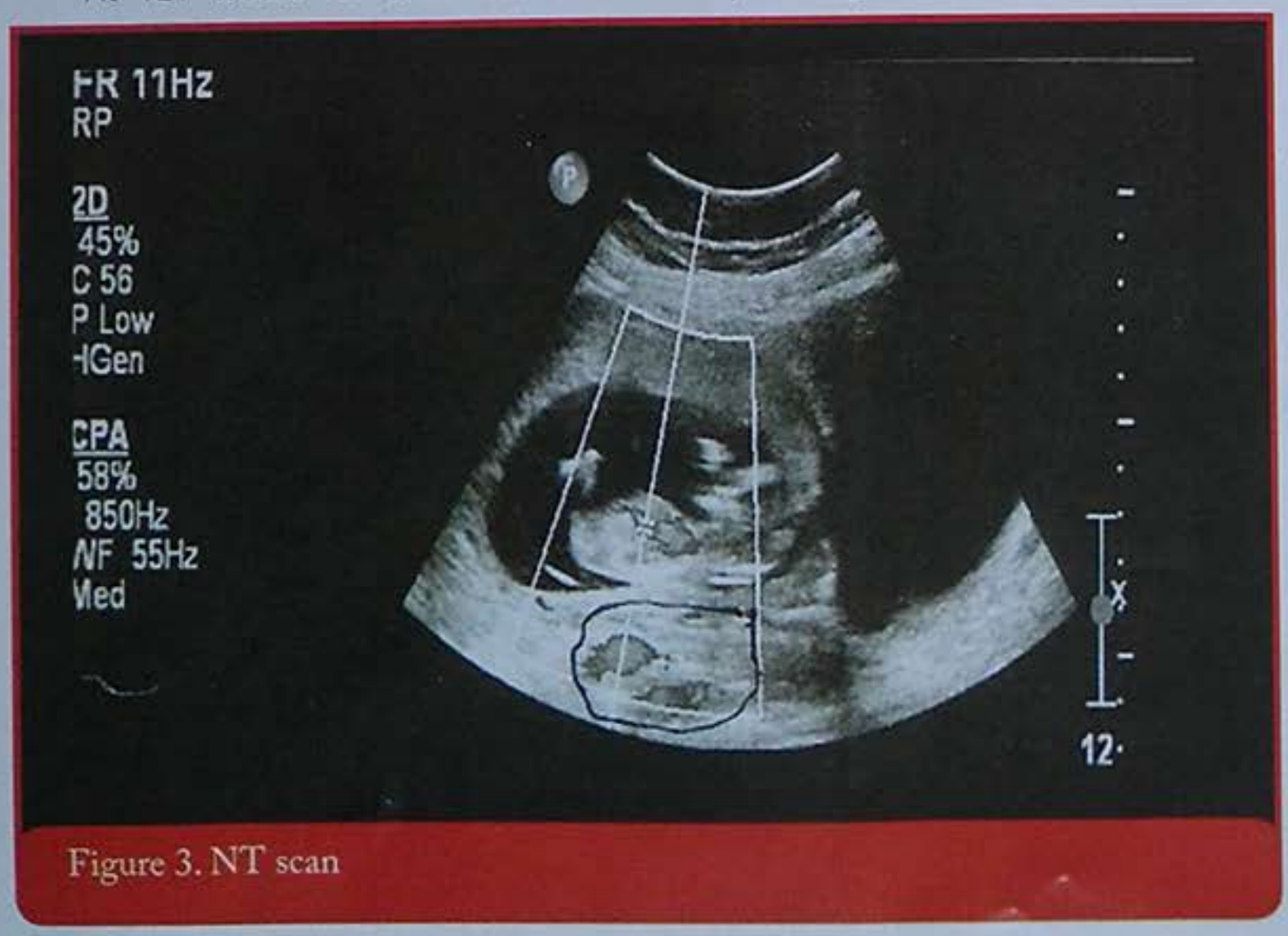

Figure 3. NT scan

Mother was doing well. She had no hyperemesis and no bleeding per vaginum. Blood pressure was within normal limits. She had no proteinuria and her blood sugar levels were within normal limits.

She was referred to a Foetal medicine centre for further evaluation. The couple were counseled regarding the ongoing gestation with the molar transformation of a part of the placenta (Figure 4) and was advised a chorionic villus sampling to determine the karyotype of the foetus. Initial FISH report was normal karyotype the follow up culture after 3 weeks also was normal.

She was on regular antenatal visits. Anomaly scan at 21–22 weeks showed no gross anomaly in the foetus the persistence of molar changes of placenta in the fundus. Uterine artery Doppler was normal follow up with growth scans at 28 and 32 weeks showed adequate interval growth and adequate liquor. Pregnancy progressed uneventfully.

Term scan showed a single live 34–35 weeks gestational age grossly normal appearing baby weighing 2400 g. in cephalic presentation, good biophysical profile and a still persisting molar change at the fundus.

At 36 weeks of gestation, her blood pressures were found high with proteinuria 1+. She was started on alpha methyl dopa 250 mg thrice daily, with which her blood pressures remained under control. Her PET profile remained within normal limits. At 39 weeks labour was induced with Dinoprostone gel in view of pre-eclampsia. As labour progressed timely

amniotomy was done and blood stained liquor was drained, suggestive of abruptio placenta, examination revealed face presentation.

A decision for emergency LSCS was taken. Live girl baby was extracted with good APGARS, and birth weight of 2.9 g, placenta and membranes were delivered in toto. A retroplacental clot of about 200 g was seen, hydropic changes in a part of the placenta was also noted (Figure 5). It was sent for histopathological examination

On histopathological examination, grossly placenta measured 15X15X3 cms, weighing 620 g. Insertion of cord was peripheral. Membranes were complete, cord measured 60 cms, cut surface showed 3 vessels and the hydropic part of placenta measured 11X8X1.5 cm (Figure 6). On its surface, it reveals multiple vesicles filled with clear fluid.

On microscopic examination, revealed enlarged villi with hydropic degeneration of the stroma, most villi showed fluid filled cisterns (Figure 7), trophoblastic layer showed focal florid hyperplasia, many villi were necrotic. It was reported as molar transformation (Figure 8).

Post operative period was uneventful, her blood pressures remained normal, did not require any antihypertensives. She was followed up with serial Hcg.[3] Weeks post partum her β hcg was 21.73 m IU/ml, 6 weeks post partum β hcg 8.2 m IU/ml, at 9 weeks postpartum β hcg 2 m IU/ml. Contraceptive options were discussed, she opted barrier method.

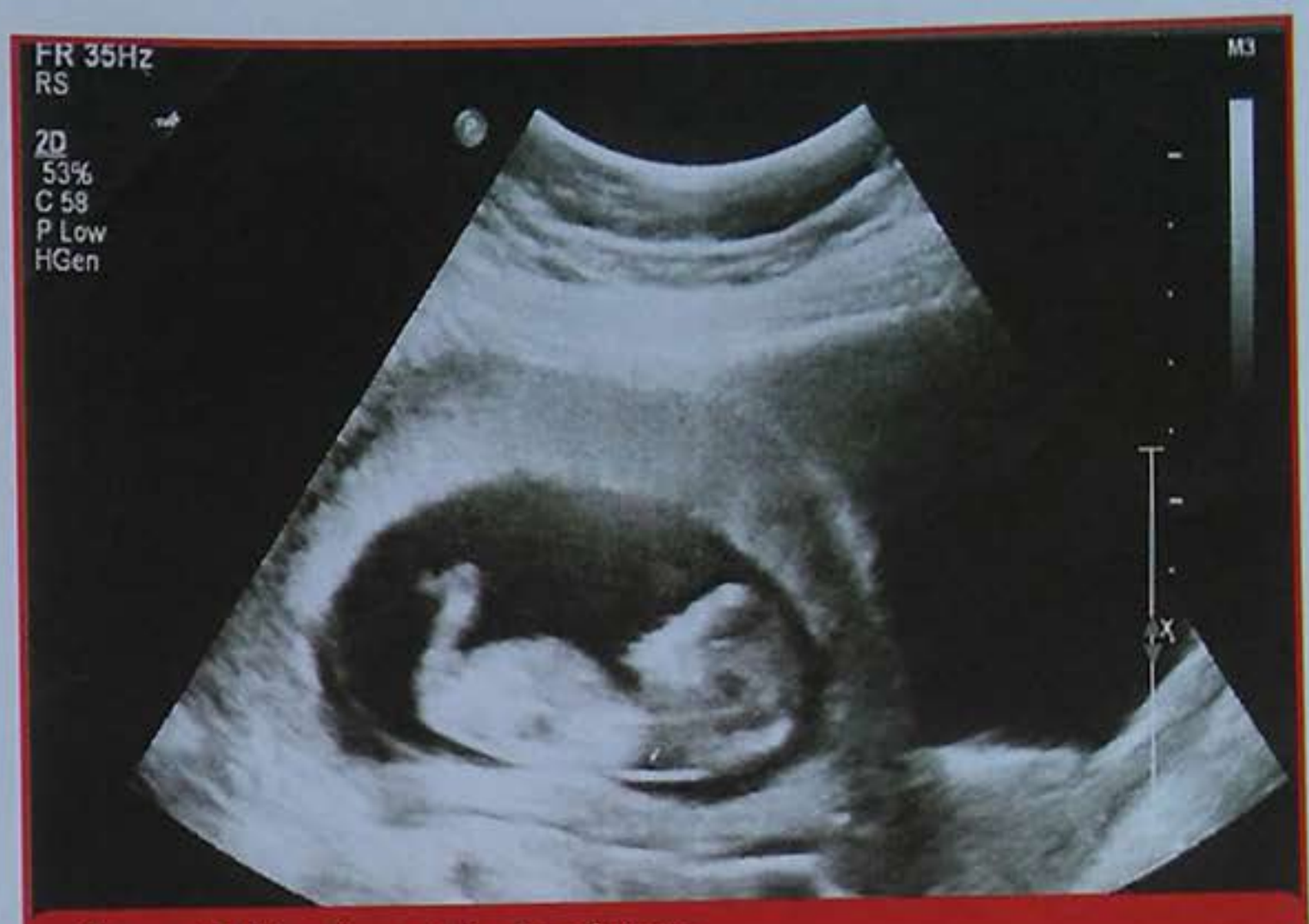

Figure 4. Molar tissue at fundus of uterus

DISCUSSION

The formation of moles is complex and it is not easily divisible into so called partial and complete moles. Rather individual genetic study is needed to make an accurate diagnosis because macroscopic or microscopic examination alone fails to assess the complexity of these entities.[4] When a foetus is present in conjunction with partial mole, it generally exhibits the stigmata of triploidy, including growth restriction, multiple congenital malformations.[5] This is compatible with both

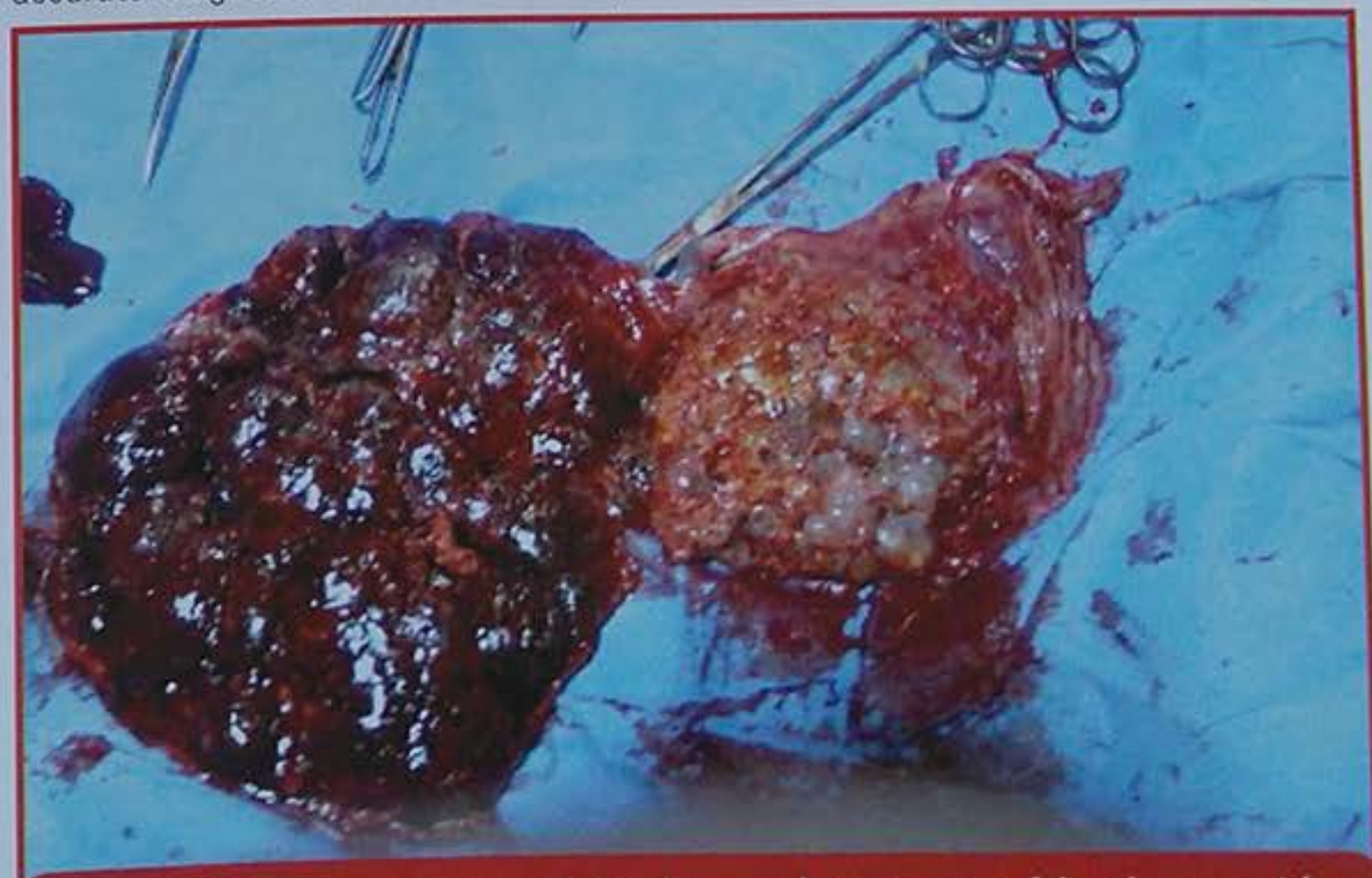

Figure 5. Gross examination of the placenta showing part of the placenta with molar changes

Figure 6. Part of the placenta with molar changes

foetal and placental development but not long term survival. Cytogenetically partial moles usually have triploid karyotype with the extra haploid set of chromosomes of paternal derivation. Whereas complete moles have a diploid karyotype that is entirely of paternal origin.[6]

Hydatidiform mole with a coexisting foetus can be established by the partial mole syndrome or by a twin pregnancy

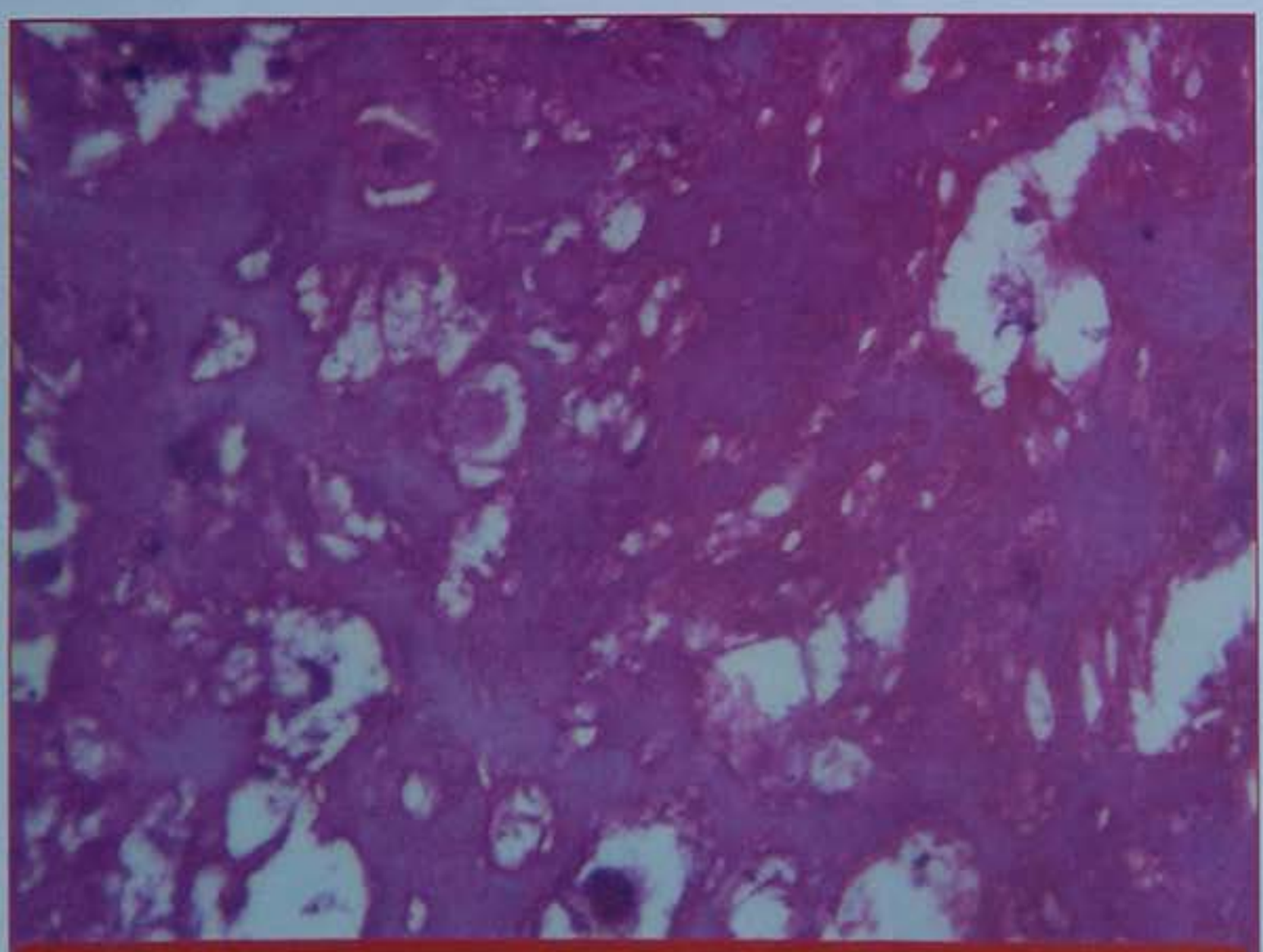

Figure 7. Microscopic examination

where the other conceptus has degenerated into a mole.[7] The theoretical explanation that it resulted from a twin dizygotic pregnancy in which one twin had developed normally and the other had degenerated into a complete mole. The difference between a partial and a complete mole cannot be firmly established by ultrasound because they both present with the same vesicular pattern.[8] In our case ultrasound revealed normal appearing foetus, but also a normal placenta connecting with sharply defined molar tissues. Since the normal placenta which was separate from molar tissues can be well defined, a complete mole pregnancy with a concurrent foetus can be diagnosed.

Risk of developing persistent gestational trophoblastic tumour should be kept in mind. The determining factor seems to be whether the molar component is partial or complete. Partial moles have a relatively low incidence of 4 percent of producing pGTT when compared to 20 percent risk in complete moles.[4] Careful surveillance for the pGTT is warranted.

Literature review have reported that pregnancies which continue beyond the 28th week, a surviving child may be expected in ~70% of pregnancies, the risk for intrauterine or neonatal death being ~30%. Persistent trophoblastic disease was reported in 9.1% of those who had continued.[9]

CONCLUSION

Molar pregnancy with a coexisting foetus progressing to term with good outcome is an extremely rare entity. They present

with varied complications. In few cases pregnancies may have to be terminated. Optimum management of these cases poses significant clinical dilemma to both patients and clinicians.

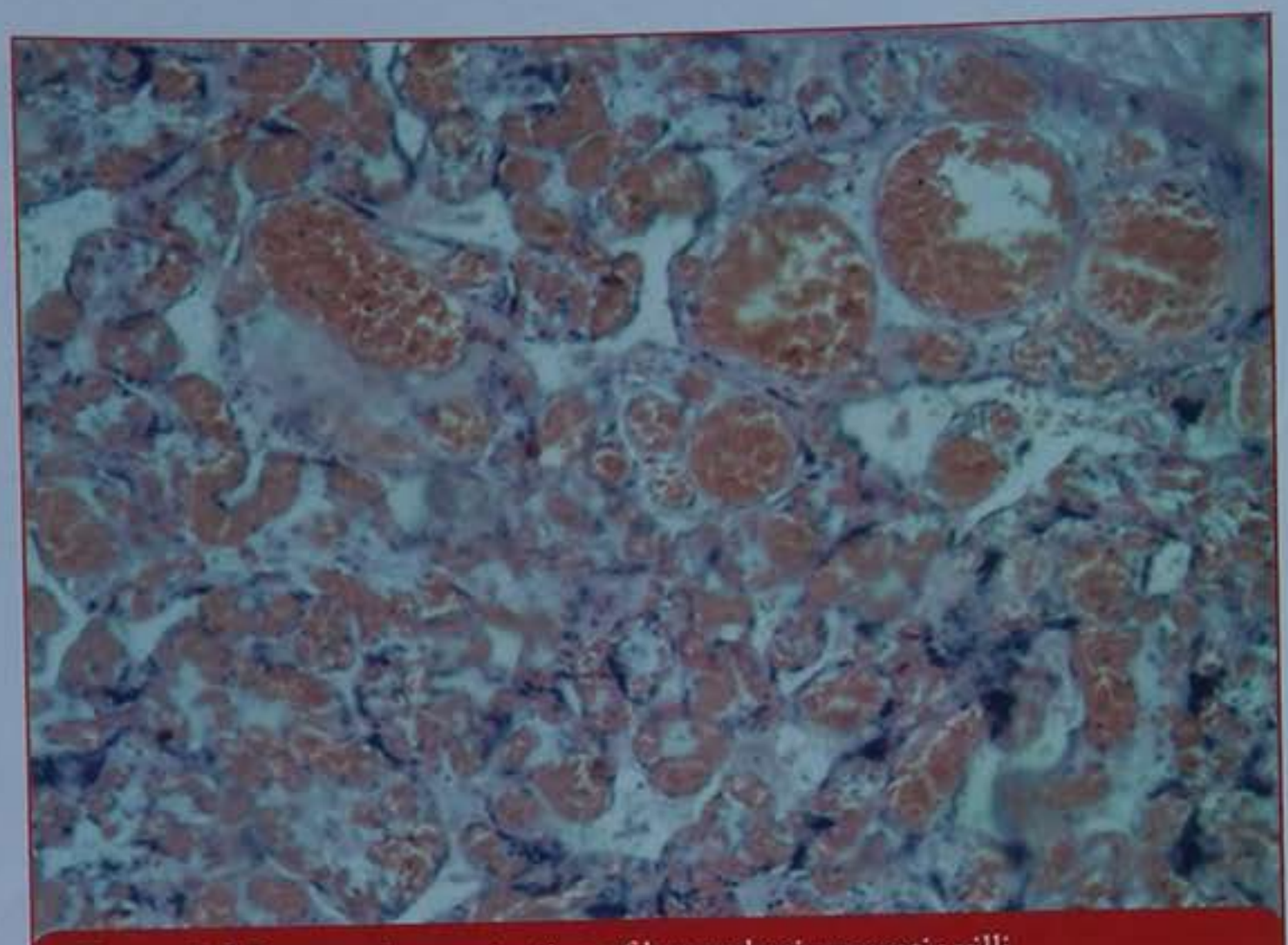

Figure 8. Microscopic examination of hyperplastic necrotic villi

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