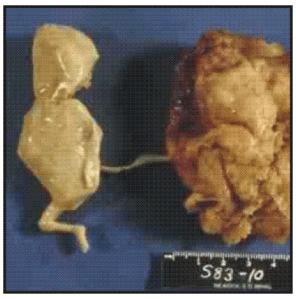
CASE REPORT PROF-922

FETUS PAPYRACEOUS;
DEMISE OF ONE TWIN IN SECOND TRIMESTER WITH SUCCESSFUL OUTCOME OF SECOND TWIN AT TERM



DR. MISBAH AKBAR, MBBS
Registrar
Shaikh Zayed Medical Complex,
Lahore.

DR. MUHAMMAD IKRAM MBBS, MCPS, FCPS
Senior Registrar
Shaikh Zayed Medical Complex,
Lahore.

DR. WASIM TALIB MBBS, MCPS, FCPS
Assistant Professor
Lahore Medical College,
Lahore.

Dr. Roohi Saeed MBBS, DA, MCPS, FRCOG
Associate Professor
Shaikh Zayed Medical Complex,
Lahore.

Prof. M. Saeed MBBS, MD, FRCOG
Professor & Head of Department
Shaikh Zayed Medical Complex,
Lahore.
obgyszh@yahoo.com

CASE HISTORY

Twenty years old primigravida was registered as a case of twin gestation at 20 weeks and advised usual baseline antenatal work up as a routine. The baseline/initial scan revealed one alive issue consistent with the gestational age of 23 weeks with one intrauterine death at gestational age of 18 weeks.

Expectant management with regular follow up and monitoring was planned for the patient which included frequent antenatal visits, ultrasonographic surveillance and monitoring of coagulation profile, after proper conunselling and discussion with the patient.

Patient had normal coagulation profile throughout her antenatal period as suggested by normal values of PT, APTT, FDPs, and serum fibribogen at monthly intervals. She had three ultrasound scans done at 29, 34 and 36 weeks of gestation, last scan showed element of mild IUGR. Placenta was fundal and post crew wall and a dead fetus near the anterior wall of uterus with cephalic presentation alongside the internal os was also noted.

After a smooth antenatal course, patient was admitted at 37+5 weeks of gestation with breech presentation for elective LSCS due to high risk pregnancy and primibreech with mild IUGR. Patient delivered 2.2 kg female as extended breech by elective LSCS alongwith a fetus papyraceous weighing 200g.

It was a monochorionic and diamniotic pregnancy. Mother and baby were discharged on fifth post operative day in a healthy state after an uneventful post operative course. To date, follow up of the baby has been perfectly satisfactory.

DISCUSSION

Fetus papyraceous occurs when one fetus dies later in pregnancy i.e. in second trimester and thereafter, but the pregnancy continues occurring in about 1 in 184 of twin pregnancies (0.54%)[1]. Thus it is relatively uncommon and has been shown to be associated with increased risk of morbidity and mortality for surviving twin.

In general, chorionicity rather than zygosity determines the risk of mortality and morbidity[2]. Perinatal mortality of monochorionic twin pregnancies is double that of dichorionic twin pregnancies. Prevalence of monochorionicity in single IUD in twin gestation is 50-70%,[3,4]

Attributable causes that have been cited in the aetiology of IUD include TTS, velamentous cord insertion, true cord knot, cord stricture, placental insufficiency, IUGR and congenital anomalies. Cord complications have been found in 30%, congenital anomalies have been present in 25% of cases[4,5] birth weight discordance has been responsible in 11-12% of cases.

Single fetal death in twin pregnancies exposes mother to the risk of DIC[6], where breach between maternal and fetal circulation allows passage of tissue thromboplastin from dead fetus and its placenta into maternal circulation leading to hemostasis impairment depending on the intensity of stimulus, incidence being 25%[7]

Effects of fetal death on surviving twin includes:

Structural abnormalities like Neural Tube Defects (NTDs), Optic nerve hypoplasia" hypoxic ischemic lesions of white matter, microcephaly, post haemorrhagic hydrocephalus, bilateral renal cortical necrosis, unilateral absence of kidney, GIT atresia, gastroschisis, hemifacial microsomia and aplasia cutis. But the risk of cerebral impairment being the greatest i.e. 1 in 5.[4] In monochorionic pregnancies death of one twin confers risk of cerebral damage in co twin of about 25% and of death in a further 25%. The situation is entirely different in dichorionic pregnancies, in which remaining twin will be spared following death of its co-twin[8].

Chronically elevated α-fetoprotein levels leads to unreliable biochemical screening results for both structural and chromosomal abnormalities[9].

In about 90% cases, there is precipitation of pre term labour and thus risk of prematurity[5].

Possibility of dystocia is there where fetus papyraceous lies transversely in pelvis below presenting part of surviving twin[10].

The proposed mechanism to explain the effects of fetal death on surviving twin is either genetic weaknesses shared by the twins triggering both fetal and developmental defects or unknown knock on effects of death of first fetus are there on the progress of the other. Observed survival difference due to chorionicity is because of frequency of vascular connections in monochorionic placentas (85-98%) leading to vascular disruption injury by haemodynamic fluctuation and trans-chorionic embolisation and coagulopathy[11].

Antenatal death of one twin in late second or third trimester poses an important management dilemma in obstetrics. Important aspects of management are[12]

Counselling and support..
Individualized management plan.
Management in a tertiary care centre with competent neonatal support.
Information on chorionicity.
Ultrasonographic evaluation of fetal anomalies and close fetal surveillance.
Steroid prophylaxis in case of preterm delivery.

Conservative management until 37 weeks unless intervention due to other obstetrical indications is required.

Aiming vaginal delivery if not contraindicated otherwise.

Postmortem examination of still born and histological examination of placenta (though of debatable value).
Paediatric assessment and long term follow up.

Regarding the time of delivery in monochorionic pregnancies it should be delayed for as long as possible to see if evidence of structural brain damage develops. In dichoronic pregnancies, the issues are entirely different and the timing of delivery must depend on gestational age and any underlying condition complicating the pregnancy[7].

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