Prenatal Diagnosis of Intraventricular Hemorrhage by Ultrasound – Case Report

J Phoon, S Tagore, SH Yeo

ABSTRACT

We describe two cases of antenatal diagnosis of fetal intraventricular hemorrhage. Both cases were diagnosed incidentally in the third trimester on a routine growth scan. Antenatal sonography revealed bilateral ventriculomegaly with no underlying malformation. All investigations were normal apart from positive antiplatelet antibody in one of the patients. Cardiotocography of both fetuses were normal during antenatal and intrapartum period. They were closely monitored till delivery. Both mothers and babies were well at discharge. One of the babies required ventriculoperitoneal shunt at 3 months of life.

Keywords: Prenatal, intracranial hemorrhage, intraventricular hemorrhage, fetal hemorrhage, ventriculomegaly

INTRODUCTION

Prenatal diagnosis of intracranial hemorrhage is a rare occurrence. Its true incidence has yet to be determined; although an estimate of 1 in 10,000 pregnancies has been suggested[1]. Factors predisposing to in utero intracranial hemorrhage include a variety of conditions. However, the etiology remains unclear in most cases. Intracranial hemorrhage is commonly associated with neonatal neurodevelopment delay and outcome including poor survival[2]. Due to possible significant adverse neonatal neurological outcome, the diagnosis has substantial impact on perinatal management with medicolegal implications.

Dr Shephali TAGORE
MBBS, MD, DRCOG, MRCOG
Consultant and Director, Obstetrics and Gynaecology International Medical Programme
Department of Maternal Fetal Medicine
KK Women's and Children's Hospital, Singapore

Prof Seow Heong George YEO
MBBS, FRCOG, FAMS
Head and Senior Consultant
Department of Maternal Fetal Medicine
KK Women's and Children's Hospital, Singapore

Corresponding Author:
Dr Jessie Wai Leng PHOON
MBChB, MRCOG, M.Med
Associate Consultant
Department of Reproductive Medicine
KK Women's and Children's Hospital, Singapore
Email: jess_phoon@yahoo.com
Telephone: +65-8123 8956

CASE REPORTS

CASE ONE

A 32 year old Gravida 4 Para 3 booked at 6 weeks of pregnancy. Her obstetric history included 3 previous uneventful spontaneous vaginal deliveries. A first trimester Down syndrome screening was low risk. A detailed fetal anomaly scan at 20 weeks gestation was unremarkable with satisfactory growth except for isolated right choroid plexus cyst of 5.8 x 3.5 mm in size with no history of pre-eclampsia or diabetes mellitus and no significant past medical history.

Fetal intraventricular hemorrhage was detected at routine growth scan at 32 weeks gestation. Ultrasound revealed bilateral ventriculomegaly whereby left posterior horn of lateral ventricle measured 16.8 mm, right posterior horn of lateral ventricle 20.2 mm (Figure 1), left anterior horn of lateral ventricle 22.1 mm and right anterior horn of lateral ventricle 15.4 mm (Figure 2). Ultrasonographic pictures were suggestive of porencephalic cyst in the anterior horn of left ventricle. There was no other underlying malformation. Fetal growth and liquor volume were within normal limits. A cardiotocograph

trace done was of a reassuring pattern (Figure 3). All maternal investigations were normal apart from positive antiplatelet antibody. However, there was no maternal thrombocytopenia. She had no previous history of blood transfusion. Infection screen for cytomegalovirus, toxoplasmosis and parvovirus were negative. The patient was referred to a neonatologist and neurosurgeon for further counseling of fetal intraventricular hemorrhage.

She was counseled on mode of delivery in view of suspected intraventricular hemorrhage. She was counseled on possible further fetal bleeding during a vaginal delivery. She decided and had an uneventful spontaneous vaginal delivery at ~39 weeks gestation. Cardiotocograph trace of fetus was reassuring throughout intrapartum period (Figure 4). The baby boy was delivered weighing 2968g with Apgar 9 (1st min) and 9 (5th min). Initial postnatal review of the baby was normal in terms of tone and reflexes except for a large, bulging anterior fontanelle with no widening of scalp sutures. There were no seizures or abnormal movements seen. Cranial ultrasound and MRI concluded a large left porencephalic cyst communicating with the ipsilateral lateral ventricle, associated with mild hydrocephalus. The baby's platelet count was within normal range. The baby was discharged home stable on day 8 of life. A repeat cranial ultrasound showed stable findings before discharge. Physiotherapy was initiated. Subsequent neonatologist and neurosurgery follow-ups were arranged. At 3 months of life, a left ventriculoperitoneal shunt was inserted as the baby was symptomatic of increasing cyst size with midline shift. At the 11th month of life, the baby had mild right hemiparesis with increased tone of right upper and lower limbs. The baby had established good feeding, normal cognitive/ hearing and eyes coordination. There were no seizures.

CASE TWO

This is a 35 year old woman Gravida 1 Para 0 booked at 7 weeks gestation. First trimester Down syndrome screening was low risk. Antenatal bloods were normal. Detailed fetal anomaly scan at ~20 weeks was unremarkable with growth parameters appropriate for gestation. Maternal blood pressure was within normal limits throughout pregnancy.

A routine growth scan at 28 weeks gestation revealed bilateral lateral ventricular dilatation. The right posterior horn of lateral ventricle measured 15.3 mm (Figure 5) and the left posterior horn of lateral ventricle measured 24.0 mm (Figure 6). The third and fourth ventricles were unaffected. Ultrasonography also revealed small granular deposits on ventricular wall and choroid plexus suggestive of recent intraventricular hemorrhage. TORCH screen and platelet antibody screen were negative. Her platelet count was normal. There was no underlying malformation and fetal growth parameters/liquor volume was within normal limits. The patient was referred to a neonatologist and a neurosurgeon for further counseling with regards to suspected fetal intraventricular hemorrhage.

A follow-up growth scan at 34 weeks gestation showed worsening dilatation of left posterior horn of lateral ventricle 53.0 mm while the right posterior horn of lateral ventricle 18.0 mm. Fetal head circumference was at 97th% for the gestation. Fetal abdominal circumference and liquor volume were normal. Cardiotocograph trace was reassuring.

In view of worsening fetal hydrocephalus, an elective caesarean section was performed at 36+3 weeks gestation after completing a course of antenatal corticosteroids. This decision was made after a multidisciplinary discussion (high risk consult) involving an obstetrician, neonatologist and neurosurgeon in our hospital. The baby girl was delivered weighing 2820g with Apgar 9 (1st min) and 9 (5th min). Initial postnatal review was normal with full range of movements of all limbs. Postnatal ultrasound and MRI scans confirmed bilateral ventriculomegaly with no midline shift. Baby was discharged well on day 4 of life. Physiotherapy was initiated. Chromosomal analysis showed normal karyotype of 46XX. No thrombocytopenia was evident in the baby. All hematological investigations (vonWillebrand factor, ristocetin, factor VIII/IX/XI/XIII, corrected PTT) were normal. The baby remained stable clinically with good head control at 6 months follow-up. Scans showed stable findings, thus the baby was managed conservatively. Postnatal review at 9 months revealed the baby's tone was normal. Subsequent review at 11 months revealed normal limb tone with no obvious neurological deficit. MRI scan also showed reduction in size of ventriculomegaly.

DISCUSSION

Antenatal detection of intracranial hemorrhage is a rare occurrence. Predisposing maternal conditions include alloimmune and idiopathic thrombocytopenia, von Willebrand's disease, specific medications (warfarin) or illicit drug (cocaine) abuse, seizures, severe abdominal trauma inflicting subsequent fetal injury, amniocentesis, cholestasis of pregnancy and febrile disease. Predisposing fetal conditions include fetal coagulation disorders, hemorrhage into various congenital tumors, twin-

twin transfusion, demise of a co-twin, or fetomaternal hemorrhage[3]. However, the cause is not identified in most cases. Most lesions occur sporadically and apart from the cases in which a specific fetomaternal condition may be identified, the risk of recurrence is low.

Maternal antiplatelet antibody in case one may have been the possible contributing factor. Neonatal alloimmune thrombocytopenia which commonly results in intracranial hemorrhage is associated with maternal antiplatelet antibody[4]. The absence of maternal and neonatal thrombocytopenia raised the question of the significance of presence of maternal antiplatelet antibodies in our case. Several reports have supported this finding. Scott JR et al.[5] has reported that although the concentration of antiplatelet antibody in maternal serum frequently reflected the severity of neonatal thrombocytopenia, a number of exceptions to this observation limited the clinical usefulness of the test for individual patients. The finding of anti-IbIX autoantibody (a specific maternal antiplatelet antibody) in control women who gave birth to neonates without thrombocytopenia raises the question of the incidence and clinical significance of such antibodies which could be natural autoantibodies as well as associated with compensated thrombocytolysis or with overt immune thrombocytopenia[6]. The significance of positive maternal antiplatelet antibody (which was not subtyped) in patient 1 is unclear as there was no neonatal thrombocytopenia.

In both patients, the diagnosis was fortuitous when the fetal ventriculomegaly was seen on an ultrasound scan which was primarily intended for evaluation of growth parameters. Ultrasonography when used to detect intracranial hemorrhage has a reported sensitivity of 96% and specificity of 94%[7]. Advances in cranial ultrasonography have led to the improved visualization of hemorrhagic or ischemic lesions, with minimal risk. Ghi et al.[2] concluded that sonograms of antenatal intracranial hemorrhage were always diagnostic. The role of MRI in the evaluation of fetal cerebral anomalies is controversial. In our patient, the MRI brain confirmed the ultrasound findings and helped exclude other underlying brain abnormalities.

Intracranial hemorrhage has a broad spectrum of manifestations with diverse prognosis. Results of Ghi et al.[2] and the review of the literature suggest that prenatally diagnosed intracranial hemorrhages have a poor outcome. About 40% of fetuses die either in utero or within the first month of life. Among the survivors, less than half appear neuro-developmentally normal at short term follow-up. With this knowledge, it is paramount for early and accurate antenatal detection of fetal intracranial hemorrhage if present. The timely diagnosis of fetal intracranial hemorrhage has substantial impact in subsequent antepartum and intrapartum management. It might be useful in the counseling of parental expectations in preparation for a poor fetal outcome. Both of our patients were counseled at length in conjunction with neonatologists and neurosurgeons upon detection of the diagnosis. This allowed the patients to make informed choices with regards to decision of further peripartum care eg. mode of delivery. The early diagnosis also benefited the patients psychologically. This underscores the importance of routine growth scans in late second/third trimester.

Long-term neurodevelopmental outcomes for both of our babies have yet to be determined although there was no obvious neurological deficit at 11 months of life in one of the babies.

There is scarce literature regarding correlation of fetal intracranial hemorrhage and fetal heart rate tracings (cardiotocogram). Some authors suggest that abnormal cardiotocograms are commonly observed in fetuses with intracranial hemorrhage[8]. However, the cardiotocograms are normal in the antenatal period and intrapartum period in both our patients. This finding emphasizes that a normal cardiotocogram does not correlate with abnormality of this severity. This finding is supported by Tejani N et al[9]. Fetal heart rate patterns immediately preceding delivery are not predictive of fetal intracranial hemorrhage especially in the preterm and low birth weight group. Termination of pregnancy was not an option as both our cases have passed the legal age of termination (24 weeks gestation) in Singapore.

The optimal mode of delivery of an infant with sonographic evidence of intracranial haemorrhage is uncertain. There is no data to indicate that a caesarean delivery may ameliorate the outcome of those infants with less severe lesions[10]. Severe lesions usually have poor prognosis and conservative management may be offered. In our case series, one of the patients opted for spontaneous vaginal delivery while the other opted for an elective caesarean delivery in view of worsening hydrocephalus. Antenatal detection of a diagnosis with potential poor prognosis is important to allow realistic expectations thus, allowing patients to make informed decisions.

CONCLUSION

Due to the possible significant adverse neonatal neurological outcome, an accurate antenatal diagnosis of fetal intraventricular hemorrhage is paramount. An antenatal diagnosis will aid counseling with regards to perinatal management. In most if not all cases, intraventricular hemorrhage is commonly diagnosed postnatally leading to a blame of an intrapartum insult. Prenatal diagnosis has implications suggesting that neurological outcome may not necessarily be due solely to intrapartum events and management. With the current advent and availability of sonography, it is timely and important to consider the value and process of determining the need for routine second/third trimester fetal ultrasound as evident by our cases. This finding is of utmost importance in terms of the ever increasing number of medicolegal cases.

Figure 1. Right posterior horn of lateral ventricle measuring 20.2 mm

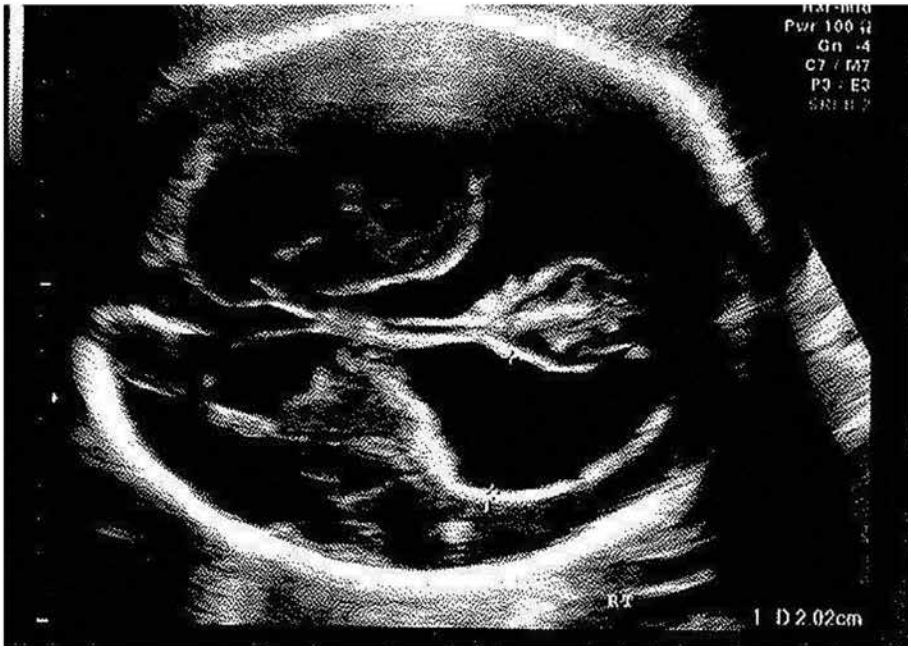


Figure 2. Right anterior horn of lateral ventricle measuring 15.4 mm

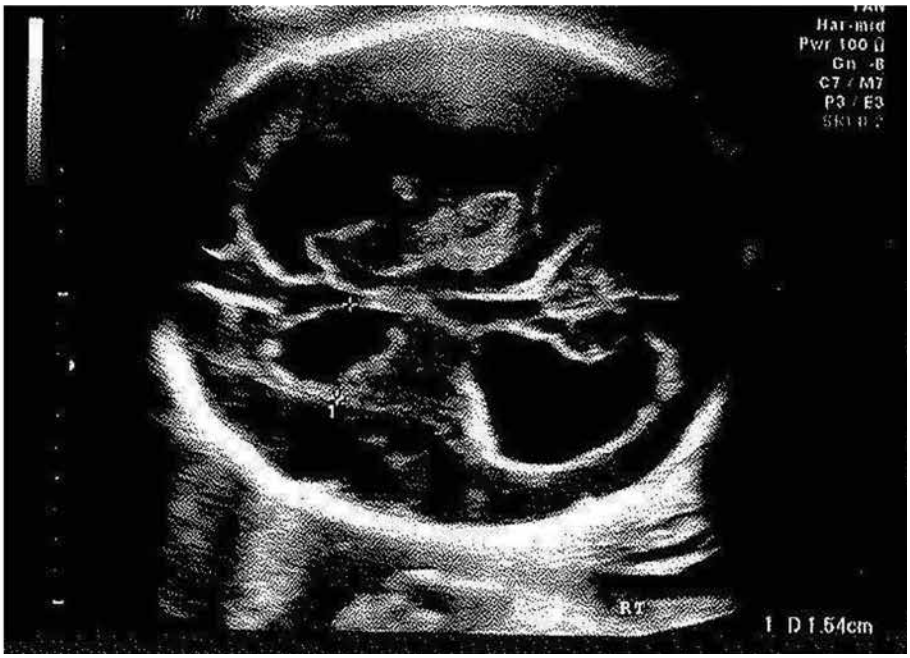


Figure 3. Antenatal cardiotocograph of patient 1

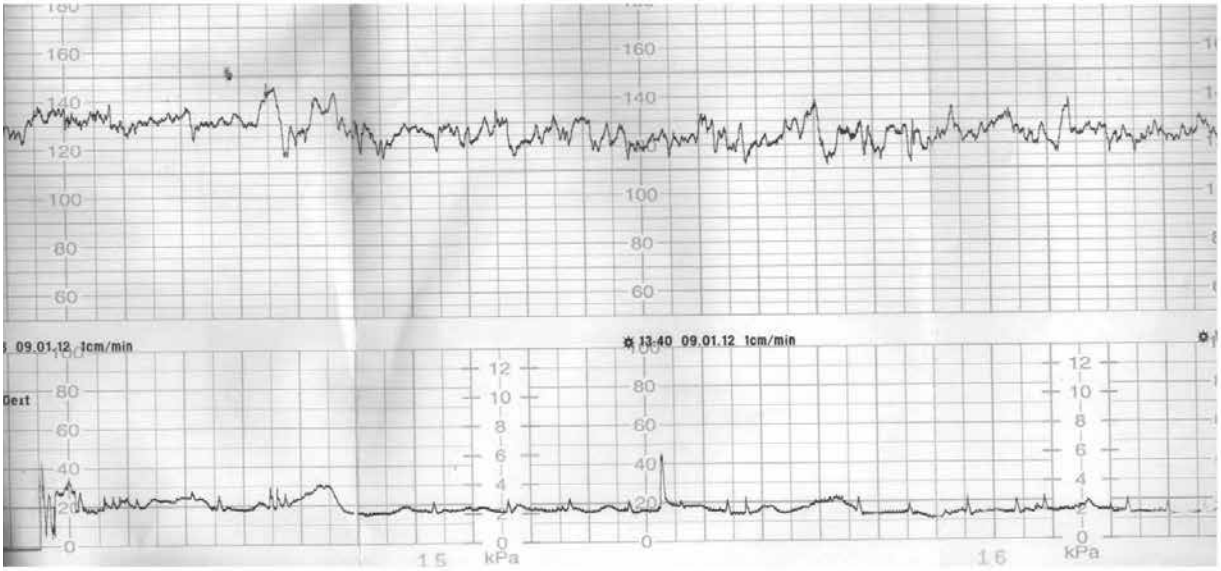


Figure 4. Intrapartum cardiotocograph of patient 1

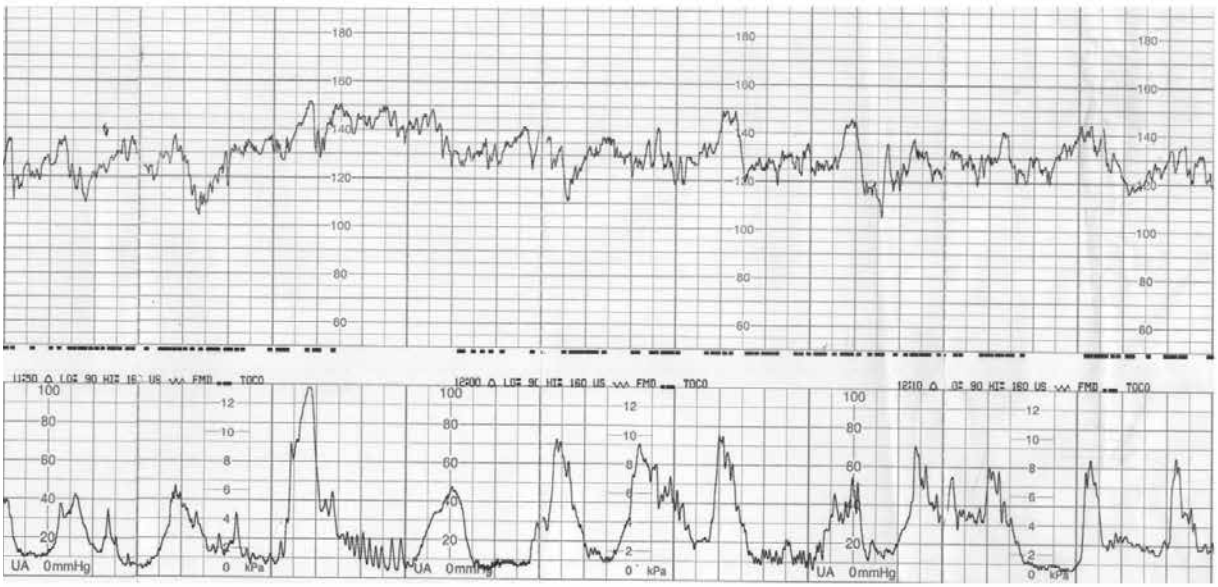


Figure 5. Right posterior horn of lateral ventricle measuring 15.3 mm

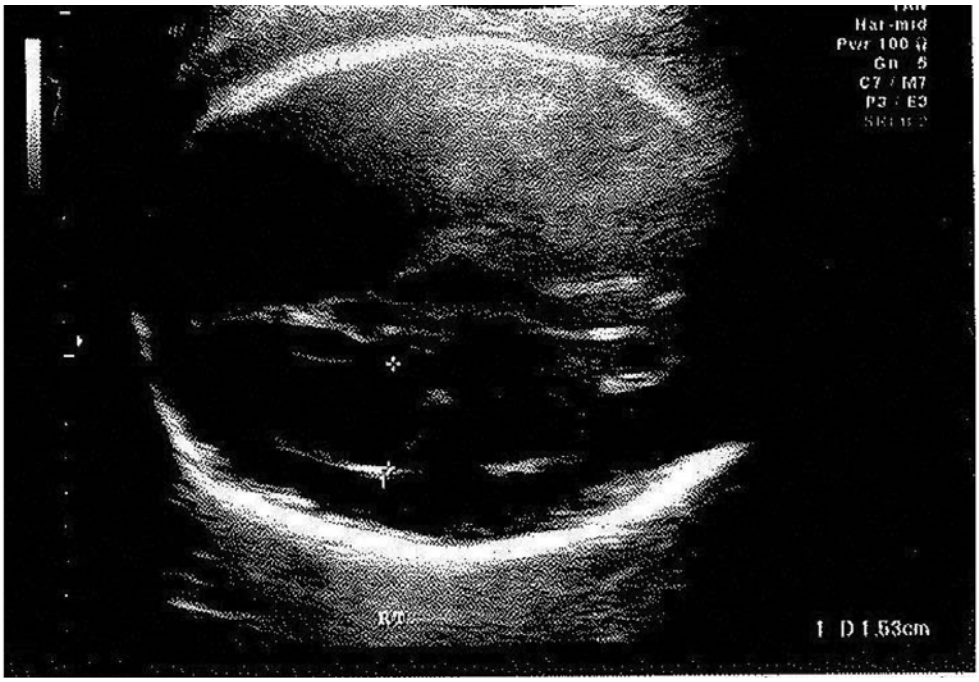
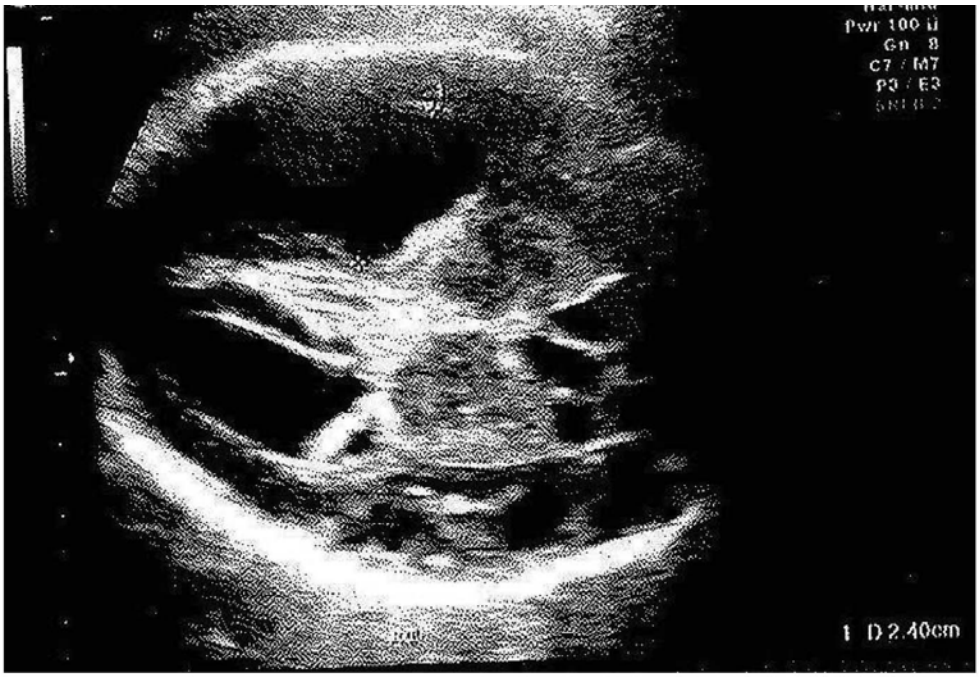


Figure 6. Left posterior horn of lateral ventricle measuring 24.0 mm



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