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Fetal intracranial hemorrhage: prenatal imaging findings and postnatal clinical outcome

Fetal intrakraniyal kanama: prenatal görüntüleme bulguları ve postnatal klinik sonuçlar

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ÖZ

Amaç: Fetal intrakraniyal kanamanın görüntüleme bulgularını ve postnatal klinik özelliklerini araştırmak.

Gereçler ve Yöntemler: Çalışmaya kurumumuzda 2018-2020 yılları arasında ultrasonografi ve manyetik rezonans görüntüleme ile tanı konulan fetal intrakranial kanama olguları dahil edildi. Maternal özellikler, ultrason ve manyetik rezonans görüntüleme bulguları ile postnatal sonuçlar hasta kayıtlarından elde edildi.

Bulgular: Tanı anında gestasyonel yaşı 21-35 hafta olan toplam 10 olgu tespit edildi. Sekiz fetusta intraventriküler kanama, bir fetusta serebellar kanama ve birinde hem intraventriküler hem de subdural kanama vardı. Olguların %60'ında tanımlanabilir bir risk faktörü saptandı. Ultrasonografi ile yedi fetusta intrakranial kanama tanısı konurken, üç fetusta diğer endikasyonlar için yapılan manyetik rezonans görüntüleme ile fetal intrakranial kanama saptandı. İki olguda gebelik sonlandırıldı, bir adet intrauterin fetal ölüm meydana geldi ve canlı doğan yedi olgu postnatal dönemde en az altı ay süreyle izlendi. Bunların %66,6'sında nörolojik bozukluk görüldü.

Sonuç: Manyetik rezonans görüntüleme fetal intrakranial kanamanın tanı ve karakterizasyonunda önemli bir rol oynar ve ultrasonografiyi tamamlayıcıdır. Bu nedenle prenatal tanıda ultrason ve manyetik rezonans görüntüleme birlikte kullanılmalıdır.

Anahtar Kelimeler: Fetal intrakraniyal kanama, fetal manyetik rezonans görüntüleme, intraventriküler kanama, prenatal tanı, ultrasonografi

ABSTRACT

Aim: To investigate imaging findings and postnatal clinical features of fetal intracranial hemorrhage.

Materials and Methods: Fetal intracranial hemorrhage cases detected in our institution between 2018 and 2020 by ultrasonography and magnetic resonance imaging were included. Maternal characteristics, ultrasonography and magnetic resonance imaging findings, and postnatal outcomes were noted.

Results: A total of 10 cases with a gestational age of 21-35 weeks at the time of diagnosis were detected. Eight cases had an intraventricular hemorrhage, one case had a cerebellar hemorrhage, and one case had both intraventricular and subdural hemorrhage. An identifiable risk factor was detected in 60% of the cases. While intracranial hemorrhage was diagnosed by ultrasonography in seven cases, in 3 cases it was detected by magnetic resonance imaging that was performed for indications other than intracranial hemorrhage. In two cases pregnancy was terminated, one intrauterine fetal death occurred and seven cases born alive were followed up for at least six months in the postnatal period. Among these, the neurological disorder was observed in 66.6%.

Conclusion: Fetal magnetic resonance imaging plays an important role in the diagnosis and characterization of intracranial hemorrhage and is complementary to ultrasonography. Hence we recommend using ultrasonography and magnetic resonance imaging together in its diagnosis.

Keywords: Fetal intracranial hemorrhage, fetal magnetic resonance imaging, intraventricular hemorrhage, prenatal diagnosis, ultrasonography

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INTRODUCTION

The incidence of fetal intracranial hemorrhage (ICH) has been reported to be approximately 0.5-1 / 1000 (1-3). With the development of vascular connections between the germinal matrix and the subependymal venous network, fetal ICH mostly occurs after 20 weeks of gestation (1,4). The ICH can be accurately identified and categorized by prenatal ultrasonography (USG)ic evaluation and magnetic resonance imaging (MRI). Hemorrhage may be seen at brain parenchyma, cerebellum, periventricular, subependymal, and subdural regions, and more commonly at intraventricular, and periventricular regions (2, 5). When ICH occurs in the critical stage in which brain development continues, such as the neonatal period (6), it can cause significant negative neurodevelopmental consequences. To demonstrate that ICH has occurred prior to labor, not due to birth injury, may prevent medicolegal action (7).

The aim of our study is to determine the ultrasonographic findings and magnetic resonance imaging features, etiological factors, and perinatal outcomes of fetal intracranial hemorrhage.

MATERIALS AND METHODS

This is a retrospective observational study conducted at the Perinatology Clinic of Health Sciences University Etlik Zübeyde Hanım Training and Research Hospital between January 2018 and January 2020. Ethical approval was obtained before the study was performed. Fetal ICH cases were detected during routine or targeted prenatal sonographic examinations were included. In all cases, fetal neurosonography was performed with Voluson E6 ultrasound device, 3-5 MHz transabdominal probes, and 5-10 MHz transvaginal probes when necessary, according to ISUOG guidelines (8). The diagnosis of ICH was considered in the presence of ventriculomegaly with thick, irregularly shaped choroid plexus that has heterogenous echogenicity, hyperechogenic ventricular walls, intraventricular hyperechogenic clots, the presence of parenchymal hyperechogenic avascular mass, increased periventricular white matter echogenicity or porencephaly. Subdural hemorrhage was detected as space-occupying hypoechoic lesions compressing ipsilateral cerebral structures.

The classification system recommended in the literature was used to define fetal ICHs (9). The grading is as follows: Grade 1: Limited to subependymal matrix, Grade 2: Extension into the lateral ventricle with normal size or ventriculomegaly with less than a diameter of 15 mm in ventricular atrium, Grade 3: With ventriculomegaly exceeding 15 mm in the lateral ventricular atrium with blood clots in one or both lateral ventricles, and Grade 4: The presence of hemorrhagic infarction in periventricular white matter, including Grade 1-3 hemorrhage. In each case fetal MRI (following ultrasonography (USG) examination) and follow up USG (1-3 sonograms) were performed. A 1.5 Tesla scanner (Siemens Magnetom AERA 1.5 T) was used for MRI. All patients were examined at supine or lateral decubitus position after 4 hours of fasting. The MRI protocol included, single shot fast spin echo T2, balanced SSFP and fast gradient echo T1 weighted sequences in axial, coronal and sagittal planes. Maternal records were reviewed to search for personal and family history, results of serological studies, and blood coagulation tests, search for maternal drug use, history of trauma, cytomegalovirus, rubella, toxoplasmosis, or herpes simplex infections which may have a role in the etiology of ICH. Blood coagulation parameters and platelet count of the newborn were also evaluated. Postnatal neurological outcome of the cases were searched through medical records and/or phone calls with their parents. The presence of cerebral palsy, epilepsy, and delayed psychomotor development were evaluated as adverse neurodevelopment. The localization and degree of fetal ICH according to sonographic and MRI findings were compared with postpartum neurodevelopmental results at least six months.

RESULTS

A total of 10 fetal ICH cases were detected. Maternal age range was 20-40 (average 27.7). Gestational age at the time of diagnosis was between 21-35 weeks (average gestational age was 25.0 weeks). In three cases pathologies additional to ICH were detected; one case had Dandy Walker malformation, one case had rhombencephalosynapsis and absence of cavum septum pellicidum, and one case had vermian hypoplasia with accompanying corpus callosum agenesis. The data indicating maternal age, gestational age at diagnosis, ultrasonographic and MRI findings, prenatal death and postnatal neurodevelopmental results are shown in table.

Table 1. Summary of USG and MRI findings and postnatal neurodevelopmental results

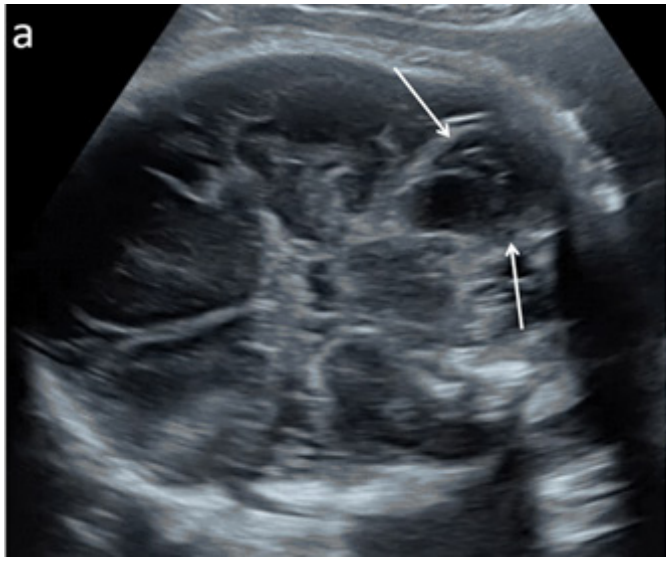
Case	Maternal age	Risk factors	Diagnosis of gestational week	USG findings	MRI findings	Grade / side	Pregnancy termination (week/sex)	Postnatal imaging	Postnatal neurodevelopment + operation
1	23	Fetal hydrops	21	ventriculomegaly	IVH	III / right	Termination (25w/female)	No autopsy	
2	23	Congenital toxoplasmosis	20	IVH+ ICC	IVH+ CCA	IV / right	Termination (23w/female)	No autopsy	
3	27	None	33	IVH	IVH	Left grade IV, right grade II / bilateral	Live born (36w/male)	MRI: Periventricular hemorrhagic calcifications in the left frontoparietal lobe (chronic parenchymal damage)	Right moderate hemiparesis + VPS
4	40	Chronic hypertansion + aspirin therapy	20	periventricular hemorrhage + DWM	Periventricular encephalomalacia + DWM	IV /bilateral	Live born (35w/female)	MRI: Bilateral lateral ventricular bleeding within the occipital horn	Bilateral axial hypotonia, convulsion
5	31	None	29	Cerebellar hemorrhage	Cerebellar hemorrhage		Live born (38w/male)	Transfontanel USG: Cerebellar hematoma	Normal
6	36	Chronic hypertansion + aspirin therapy	21	IVH	periventricular hemorrhage + porencephalic cyst	IV / bilateral	Live born (37w/male)	Transfontanel USG : normal	Normal
7	34	Congenital toxoplasmosis	27	Bilateral ventriculomegaly	IVH + cystic encephalomalacia	IV / bilateral	Stillbirth (33w/female)	No autopsy	
8	35	None	31	Periventricular hemorrhage + Interhemispheric and cerebellar cysts	IVH + Porencephalic cyst + CCA + VH + cerebellar encephalomalacia	IV / bilateral	Live born (39w/female)	MRI: Bilateral caudothalamic grooves and intraventricular bleeding	Convulsion, bilateral moderated hemiparesis
9	23	None	21	Ventriculomegaly + SPA + RES	IVH + SPA + RES	III / bilateral	Live born (35w/male)	MRI: left IVH + SPA + RES	Motor development regression
10	20	Congenital syphilis + fetal hydrops	27	IVH	IVH + SDH	Grade IV / bilateral	Live born (34w/male)	Transfontanel USG: normal	Normal

IVH: Intraventricular hemorrhage, ICC: Intracranial calcification, DWM: Dandy-Walker Malformation, CCA: Corpus callosum agenesis, VH: Vermian hypoplasia, SPA: Septum pellicidum agenesis, RES: Rhombencephalosynapsis, SDH: Subdural hematoma, USG: Ultrasonography, MR: Magnetic resonance imaging, VPS: Ventriculoperitoneal shunt

Eight cases had intraventicular hemorrhage (IVH), one case had cerebellar hemorrhage (Figure 1), and one case had both intraventricular and subdural hemorrhage. Porencephalic cysts were observed in Case 6 and Case 8 with Grade IV hemorrhage (Figure 2). In nine cases with intraventricular hemorrhage, one had right-sided grade III, one had right-sided grade IV, one had grade IV on the left and grade II on the right, one case had bilateral grade III, and the other five had bilateral grade IV hemorrhage. While ICH was detected in seven (70%) cases by USG and confirmed by MRI, in 3 cases it was missed by USG and detected by MRI. The indications for referral of these three cases to MRI were isolated ventriculomegaly in two of them, and absence of cavum septum pellicidum and rhombencephalosinapsis in addition to ventriculomegaly in one which were all detected through sonographic evaluation.

The indications for referral of these three cases to MRI were sonographic findings as isolated ventriculomegaly in two of them and the absence of cavum septum pellicidum, and rhombencephalosinapsis in addition to ventriculomegaly in one of them. In three cases MRI gave additional information to sonographic findings as corpus callosum agenesis (CCA) in Case 2, CCA + vermian hypoplasia in Case 8, and subdural hemorrhage in Case 10.

Figure 1 (case 5) USG image at the oblique axial plane (a) and coronal T2-weighted MR (b) image show left cerebellar hemisphere hematoma in the subacute-chronic period (arrow) as a cystic anechoic mass, and hyperintense mass with hemosiderin ring respectively.



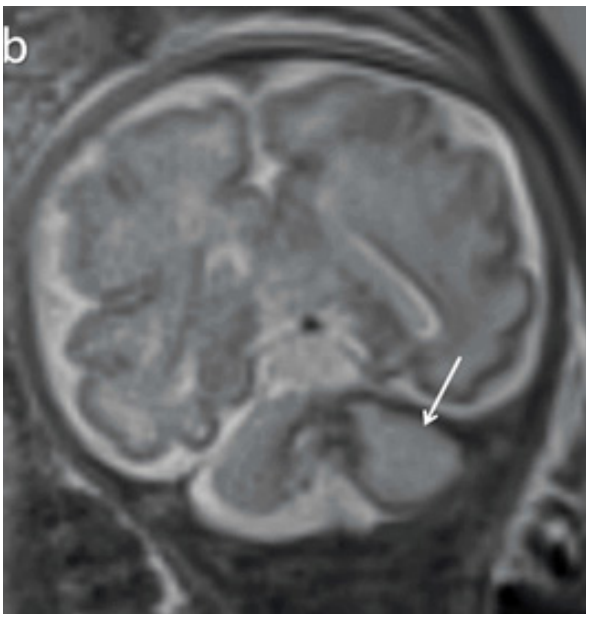
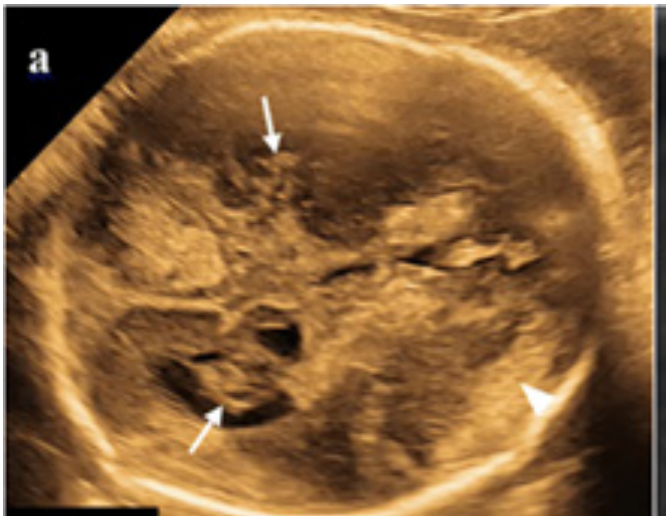
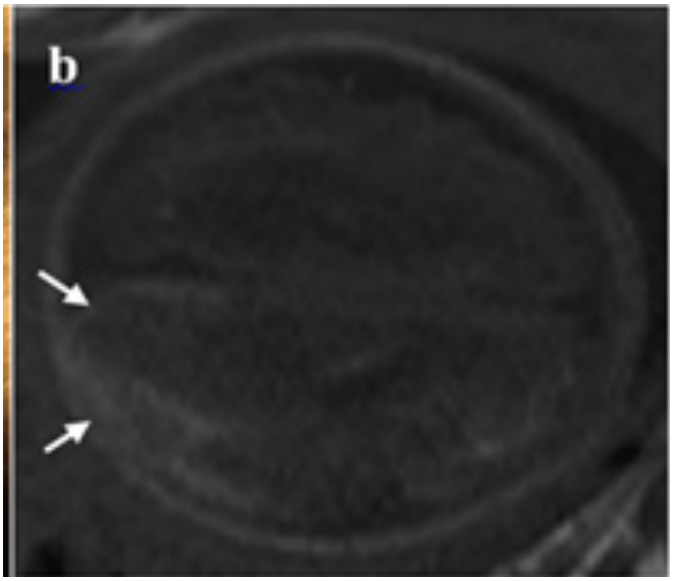
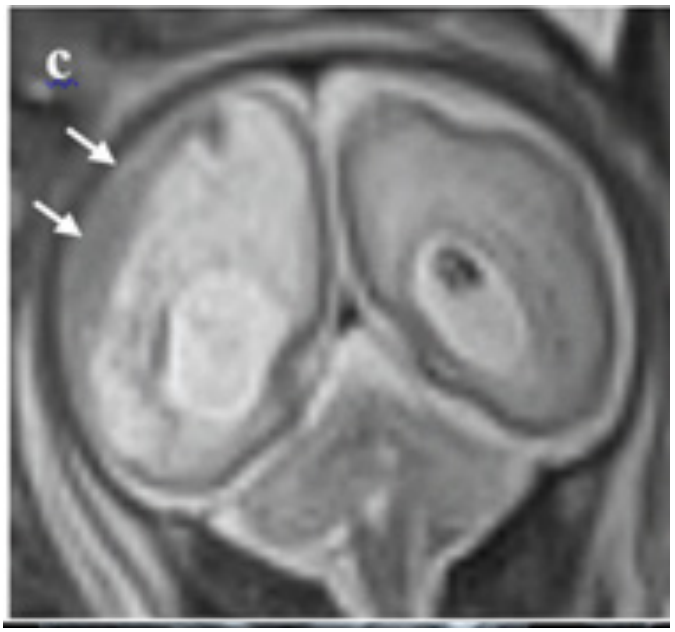
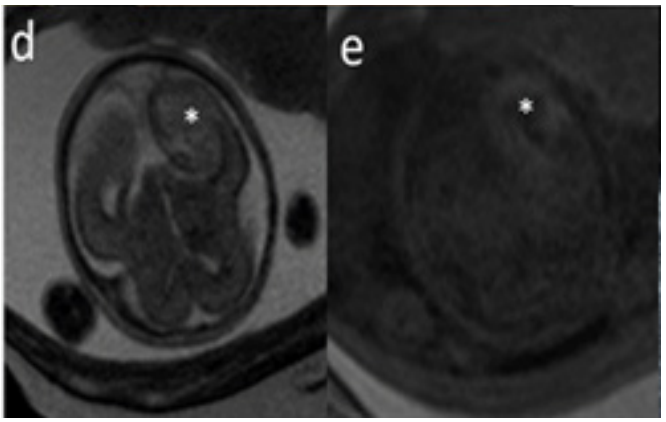


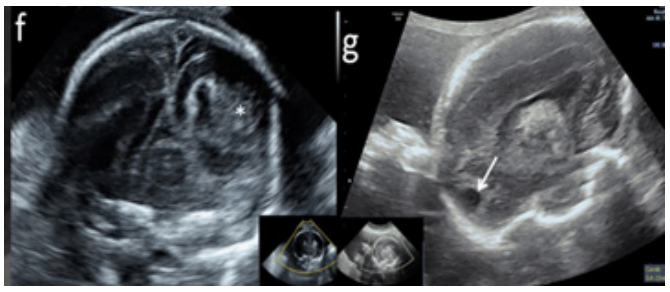
Figure 2 (case 10) axial USG image shows (a), parenchymal hemorrhage in the right frontal lobe(arrowhead) and bilateral intraventricular hyperechogenic clots (arrow) (arrowhead), axial T1 weighted (b) and coronal HASTE (c) images show subdural hemorrhage extending into the posterior of the interhemispheric fissure. (case 6), T2-weighted (d), T1-weight (e) MR and USG (f) images show extensive bleeding (*) in the cortex and white matter in the left occipital lobe, (g) sagittal USG image shows porencephalic cyst (arrow) in the left occipital lobe.











An identifiable risk factor was detected in 60% (6/10) of the cases. In two cases there was maternal antihypertensive drug and aspirin use due to chronic hypertension. Maternal Toxoplasma IgM and IgG were positive in two cases (Case 2, 7) and avidity tests also showed acute Toxoplasma infection . In addition, Case 2 had intracranial calcifications detected by USG. Toxoplasma PCR from amniotic fluid could not be studied in these cases due to non approval of amniosynthesis by the parents. In Case 2 pregnancy was terminated and stillbirth occurred in Case 7, hence toxoplasma PCR could not be studied in the postnatal period. Fetal hydrops was present in two cases and middle cerebral artery peak systolic velocity indicated severe fetal anemia in both of them.

Blood analysis was performed in the neonatal period in all live-births (7/10) and hemoglobin, platelet values, and coagulation tests were found to be normal. Autopsy could not be performed for the cases that resulted in pregnancy termination and stillbirth due to the refusal of the families. Postnatal imaging findings were obtained in all live births. Fetal ICH was detected in the postnatal imaging of five cases (71.5%), while transfontanelle USG findings were normal in two cases (28.5%). Case 5 with antenatal cerebellar hemorrhage and transfontanelle USG findings of cerebellar hemorrhage in the postnatal period showed normal neurodevelopment. Cases 6 and 10 which had IVH had normal transfontanelle USG and showed normal neurodevelopment (33.3%). Case 3, 4, 8 with antenatal Grade IV IVH and case 9 with Grade III IVH had adverse neurodevelopmental outcomes (66.6%). Postnatal ventriculoperitoneal shunt was applied in one patient (Case 3).

DISCUSSION

Neonatal ICH is an important cause of morbidity and mortality, and the situation is similar when it occurs in fetal life. Some genetic factors, maternal or fetal causes play role in the etiology of ICH. The genetic factors include congenital thrombophilia and COL4A1, and COL4A2 gene mutations. Mutations in the COL4A1 gene may cause fetal ICH hemorrhage and the development of porencephalic cysts (10). The maternal risk factors include hypertensive diseases, drugs, platelet / coagulation disorders, convulsion, trauma, viral or bacterial infections, febrile illness, and amniocentesis (11-18). Intracranial tumor, twin-twin transfusion syndrome, single twin demise, and fetal anemia (19-21). Alloimmune thrombocytopenia, which is one of the causes of ICH, is rarely seen (22). Fetal and neonatal alloimmune thrombocytopenia is the result of maternal alloantibodies attac-

king fetal platelets caused by incompatibility of human platelet antigens and causes development of fetal thrombocytopenia. The most commonly involved antigen is HPA-1a, which tends to be associated with more severe cases (23). However, most studies did not find an identifiable etiological factor, and risk factors for fetal ICH were reported to range from 20% to 45% (2,9,18,24,25). In the present study, the risk factors that may play a role in the etiology were congenital toxoplasmosis, fetal hydrops/anemia, high maternal blood pressure, aspirin use, which were shown to be found in 60% of the cases.

Fetal ICH may not be clinically detectable in the prenatal period (26), besides there are some ultrasonographic findings which were determined as diagnostic criteria (9,24,25). On the other hand, the sonographic appearance of ICH is highly variable and depends on the region, severity of the disease, and time of occurrence (9,27,28). There are four phases defined for fetal ICH. The first phase is fresh hemorrhagic phase at which echogenic blood filling the lateral ventricles is present (3-8 days). The liquefaction stage is the second one at which a mass with outer echogenic lining and internal anechoic focus are seen (3-8 days). The third stage is complete liquefaction stage and cystic hypoechoic mass is present (7-28 days). Ventriculomegaly and disappearance of blood clots (7-105 days) are seen at fourth stage; the solubility phase (9). Grading system of IVH in the e intrauterine period is similar to the one in the neonatal period. Since intraventricular Grade I-II hemorrhages in fetal life are frequently overlooked by USG, 70% to 100% of the detected cases are Grade III-IV hemorrhages (2,3,9,18,24,25). In line with this, in our study, six cases detected by USG all had grade III-IV hemorrhage.

Various studies have been conducted to investigate the role of MRI in fetuses with central nervous system (CNS) pathologies (29-34). Di Mascio et al. (31) investigated fetal MRI findings in cases with prenatal mild and moderate ventriculomegaly and reported that additional brain abnormalities were found with MRI in 10% of the cases and of these 0.6-2.5% had ICH. In a study by Sanapo et al. (32) among the 36 fetuses which were referred to MRI due to ventriculomegaly, ICH was detected in 56% (n=20 cases), however, ICH was suspected by USG in only 22% (n=8 cases). In a study conducted by Adiego et al. (27), MRI provided additional information in 54.5% of fetal ICH cases. Kutuk M et al. (3) stated that MRI is useful in detecting the region and extent of bleeding, and the size of hematomas and excluding other intracranial pathologies especially in Grade 3-4 hemorrhages. In another study, it has been reported that MRI has the potential to determine the etiology of ventriculomegaly with a much more sensitivity and specificity than USG and to further illuminate the mechanisms of brain injury and the impact of chronic hypoxia (33). In addition, MRI can help to predict the time of bleeding and hematoma development (1,35). In the present study USG detected 70% (7/10) of cases with ICH while 30% (3/10) of the cases missed by USG were detected by MRI which was performed with the indication of ventriculomegaly. These findings show that fetal ICH cases can be overlooked by USG and MRI provides additional information in fetal CNS pathologies, as stated in the literature (2,3,9,18,34). In the present study, MRI was found to be useful especially in for accurately grading IVH and therefore informing parents about the prognosis of the fetus. However, there are controversial opinions about whether the fetal MRI has extra benefits over USG in the diagnosis of fetal ICH or not (2,9). Abdelkader et al. (24) reported that they could not find any additional benefit of fetal MRI over USG in the diagnosis of fetal ICH.

Similar to the neonatal period, in fetal IVH, there is a relationship between the degree of the lesion and clinical outcome. Neurodevelopmental outcome of isolated grade I and II cases are good (35). It has been reported that the risk of neurodevelopmental failure increases in fetal Grade III-IV IVH cases (2). Ghi et al. (9) reported perinatal mortality rate of 7.1% in fetal Grade I - II hemorrhages and 44% in Grade III-IV hemorrhages. In our study, among the six live births,four had adverse postnatal neurodevelopmental outcome (66.7%). However, three of these had additional CNS abnormalities that would affect the prognosis. In addition, our perinatal mortality rate was 12.5%. The high rate of poor prognosis in our study group may be related to the presence of accompanying anomalies. Fetal IVH can recover spontaneously over the time or result in ventriculomegaly by causing obstruction. In two of our live birth cases, ventriculomegaly regressed over time and these cases showed normal postnatal neurodevelopment (33.3%).

Our study has some limitations. Our study population was small and had heterogeneous in terms of the distribution of cases. We also did not have grade I-II IVH cases which enables us to generalize our results regarding the outcome and prognosis.

CONCLUSION

Sonographic diagnosis of fetal ICH is possible but requires careful investigation. Fetal MRI plays an important role in characterizing ICH and is recommended as complementary to USG. Demonstration of bleeding or ischemic brain damage before

the onset of labor is also important to document the neurological problems which are not due to birth trauma.

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