International Journal of ChemTech Research
CODEN (USA): IJCRGG, ISSN: 0974-4290, ISSN(Online):2455-9555
Vol.10 No.6, pp 873-881, 2017

The effects of Anemia on Morphohistological of placenta in AL-Najaf city

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Abstract : Anemia in pregnancy is related with viable histomorphological changes in placenta . The present study aims to clear the extent of structural changes on the efficiency of the placenta to support the growth of fetus . The study included 100 placenta taken from 100 delivery which have been collected at birth from women attended Obestatric Department in AL-Zahra Education Hospital for Maternity and children and the middle Euphrate Hospital in AL-Najaf city –Iraq at the period from Octobar 2015 to April 2016 . The result of this study explor that the maternal age distribution predomination at the age group 36 year . The anemia in multigravida was found to be more than number in primigravida . The anemic pregnant mother give birth with fluctuation from normal babies to low birth weight . The gross observation of the placenta revealed a slight decrease in placental weight in anemic pregnant mothers .with irregular in shape and decrease mean diameter of placenta , while the green color observed only in anemic group and absent in non-anemic . In the present study the placental < 330 gm observed only in anemic group .The gross observation of umbilical cord showed umbilical cord thrombosis in 20 % of anemic group and absent in non-anemic group. Anemic in the pregnancy alters the Histological structural of placenta . The present study showed significant microscopic change in placenta of anemic group as compared to the non-anemic . Placenta in anemia showed excessive syncytial knots fibrinoid necrosis of the villi , increase villous stromal fibrosis.
Keyword : Placenta , Pregnancy anemia , Brith weight , Preterm , Neonatal outcome , Apcar score.

Introduction

The placenta is a mirror which reflects the intrauterine status of the fetus. It is a vital organ for maintaining pregnancy and promoting normal fetal development. It is an organ that connects the developing fetus to the uterine wall, thereby allowing nutrient uptake, waste elimination, and gas exchange via the mother's blood supply. Proper vascular development in the placenta is fundamental to ensuring a healthy fetus and successful pregnancy (Wang and Shuang,2010). The placenta plays an active role of fetal programming during intrauterine life (Waterland and Michels,2007). Placental maturity, with fully mature villous tree and increased capillarization, is required for spontaneous induction of labor (Benirschke*et al*.,2012). Amount of available oxygen is a critical factor in placental development and development of placental blood vessels as well (Huang et al.,2001). The placenta is known to have two important functions, (1) as an endocrine organ and (2) in maternal-fetal exchange (Jameson and Hollenberg,1993 ; Srisuparp *et al*.,2001 ; Maston and Ruvolo,2002). The expelled placenta is a flattened discoid with an approximately circular or oval outline, with an average volume of some 500ml (range 200-950ml), average weight about 500g (range 200-800g), average diameter

185mm (range 150-200mm), average thickness 23mm (range 10-40mm). Thickest at its center (the original embryonic pole) it rapidly diminishes in thickness towards its periphery where it continues as the chorion leave (Williams,1995). According to WHO standards, maternal anemia is present when the haemoglobin level in blood is less than 11gm/dl. (Buseri *et al*.,2008).There are three main types of anemia: (1)that due to blood loss, (2) due to decreased red blood cell production, (3)that due to increased red blood cell breakdown. Causes of blood loss include trauma and gastrointestinal bleeding, among others. Causes of decreased production include iron deficiency, a lack of vitamin B12, thalassemia, and a number of neoplasm of the bone marrow. Causes of increased breakdown include a number of genetic conditions such as sickle cell anemia, infections like malaria, and certain autoimmune diseases. It can also be classified based on the size of red blood cells and amount of hemoglobin in each cell. If the cells are small, it is microcytic anemia. If they are large, it ismacrocytic anemia. And, if they are normal sized, it is normocytic anemia. (Janz*et al*.,2003 ; Smith,2010). Anemia in pregnancy is related with variable histomorphological changes in placenta, including widening of intervillous space and decrease in thickness of villous membrane, which show a clear reflection for the poor fetal outcome (Mongia*et al*.,2011).There is a need to explore the extent of above mentioned structural changes, because severity of these histomorphological parameters is correlated with the efficiency of placenta to support the growth of a fetus, and this condition is likely to be related to insufficient function of the placenta (Baptiste *et al*.,2008).

Materials and method :-

The present study included 100 placenta and umbilical cord were collected immediately after delivery, both from normal deliveries and Caesarean sections. Which have been collected at birth from the women attended obstetrics department in al-Zahra educational Hospital for Maternity and Children and the Middle Euphrates hospital in AL-Najaf city from period from October 2015 to April 2016 . This study sample was divided into two groups :- First Group: pregnant women suffering from anemia and they're 50 sample (placenta and umbilical cord). Second Group: pregnant women who have good health (naturalist) and they're 50 sample(placenta and umbilical cord) .A sample of the placenta and umbilical cord collected from women after delivery and removed the tissues of the placenta and put in a plastic container and washed well with water for 10-15 minutes with a little compress on the placenta to removed excess blood and clots of blood were then drying the sample by pieces of gauze and cotton and then placed on a piece plastic colorful and filmed by a camera (Digital) for the maternal and fetal surface with umbilical cord. The cord and membranes were examined for any abnormalities. and data collected like period of gestation, delivery approach , number of gestation , number of living , number of death , number of abortion , living address , age of the mother and health status. Histological sections prepared to sample of placenta according to Bancroft and Stevens (1982). Then Light microscopic studies of the histological sections of placentas under different magnifications revealed changes. These changes were observed in both anemic and control group of placentas and photo of that by camera of light microscopy.

Results and Discussion

In table (1) shows that the mean of placental weight (gm) was (504.77)in the anemic group , v.s. (578.913) in the non- anemic group . A past study mentioned that the placental weight effected by anemia pregnant women (Beischer *et al*.,1970 ; Barker *et al*.,1990 ; Godfrey *et al*.,1991). Also, The intrauterine existence of the fetus depends on the placenta elaborated by both maternal and fetal tissues to sever as an instrument of transfer of essential elements. Many researchers concluded that the maternal anemia is associated with large placental weight (Lao and Tam,2000).While reported that in anemia the weight and volume of placenta decrease with a statistically significant results (Dhall,1994). In previous study, Bis was observed that the weights of placentas in anemic group ranged from 220-500gm (mean 383gm). and in the control group the weight of placenta ranged from 250-500 (mean 370.3 gm) which mean placenta with increase weight and volume (Biswas*et al*., 2014). In percent study the mean of cotyledons per placenta have a slight decrease in number in the anemic group which was(22.42) while in the non- anemic group was (22.86). Similar results found in the study by Singla et.al (2008) that the birth weight , placental weight and number of placental cotyledons were reduced in anemic mothers related with the maternal haemoglobin level . The previous study reported that less septation of placenta in hypoxic conditions is caused byanemic pregnant, which leading to decreased number of cotyledons (Singla *et al*.,1978). The mean central thickness of placenta area (mm) in

anemic group was (24.27) mm, while in non-anemic group was (28.20)mm. Past study observed mean thickness of the placenta decrease in maternal anemia (Grewal,2010) .In the present study it was found that the mean of diameter of placenta (cm) was (16.26) cm in comparison with that non-anemic group which was (16.79) cm. As reported by Kulaudaiveluet. al.(2014).they observed that the mean ±SD diameter of placenta was 14.65 cm in the anemic group (Kulaudaivelu *et al*.,2014). The shape of placenta in the anemic group, the regular (cycle) was 27 , while the numbers of them in the non-anemic group was 36 and regular (oval) shape of anemic group was 13 , while the number of them in non-anemic group was 14 . The irregular shape is predominated only in the anemic group in comparison to non- anemic . This table also shows that the gray color of placenta in anemic group was 30 , while absence in the non-anemic group and observation red and green color was 14 and 6 respectively in the anemic group while red color was 50 and green color of placenta absence in the non- anemic group.

Table 1:The observation characteristics of placenta in anemically state of pregnant women :-

Gross Observation			**Anemic**	**non-anemic**	***p*-value**
Mean of placenta weight(gm)			504.772	578.913	0.928
Mean of cotyledons per placenta			22.428	22.86	0.401
Mean central thickness of placenta area (mm)			24.272	28.204	0.410
Mean of diameter of placenta (cm)			16.266	16.798	0.379
Shape of placenta (no)	Regular	Cycle	27	36	3.333
		Oval	13	14	0.281
	Irregular		10	0	3.888
color of placenta (no)	Gray		30	0	0.0002
	Red		14	50	3.888
	Green		6	0	0.0002

In table (2) shows the mean of birth weight of babies (Kgm)which was (3.016Kgm) in anemic group , when compared with non- anemic group which was (3.264Kgm). The mean of placental weight in the anemic group was(504.772 gm) while in non- anemic groupe was (578.913 gm). The ratio of the fetal mass to the placental mass is recognized as feto-placental proportion. Which is usually 6:1 . In the present study , this ratio was (5.975) in anemic group was in comparison with non- anemic groupe which was (5.638).These results coincide with the results obtained by (Kulaudaivelu *et al*.,2014) who stated that the feto-placental ratio is 5:1 with increase in the number of defined cotyledons. Previous studies said that mother anemia has advanced occurrence of prematurity and LBW babies (Brabin *et al*.,2001 ; Marchant*et al*.,2004; Shobeiri *et al*.,2006). Also, previous study mentioned that anemia in pregnancy may have adverse effects on pregnancy maternal anemia is an self-governing danger influence for little birth weight (Levy *et al*.,2005). Also, previous studies mentioned that the placental weight are effected by anemia pregnant women and the relationship with birth weight and displayed to mean of feto-placental ratio (Godfrey *et al*.,1991 ; Loa and Tam,2000).

Table2: Relation of placental weight gain and corresponding birth weight in anemic and non-anemic gravid females:-

Observation	Anemic	Non- anemic	*p*.value
Mean birth weight of babies (K.gm)	3.0163	3.264	0.928
Mean of placental weight in (gm)	504.772	578.913	0.928
Mean of feto-placental weight ratio	5.975	5.638	0.928

In table (3) the insertion site of umbilical cord is divided into centric, excentric, marginal and Velamentousin the anemic group. They were (13 , 20 , 12 and 5) respectively, while they were (22 , 28 , 0 and 0) respectively in comparison with the non-anemic group. The mean of umbilical cord length in the anemic group was(42.02)cm, while it was (58.36)cm in the non-anemic group . In the present study umbilical cord thrombosis has been 20 % in anemic group and absence of thrombosis in non-anemic group. Maternal anemia has no effect on haematological profile of cord blood (Ekta and Snehal,2014). In the previous study complete by Sweet et. al.(2008).originate at motherly iron reduction is related with abridged fetal hepatic and intelligence fillings but no alteration in iron obtain ability to fetus.

Table 3:-Gross observation of umbilical cord in anemic and non-anemic gropes :

		Anemic	**non-anemic**	***p*.value**
Insertion site of umbilical cord (No)	**Centric Excentric marginal Velamentous**	**13 20 12 5**	**22 28 0 0**	**0.00000**
Mean umbilical cordlength (cm)		**42.02**	**58.36**	**0.00000**
Umbilical Cord thrombosis (%)		**20%**	**0%**	**0.00000**

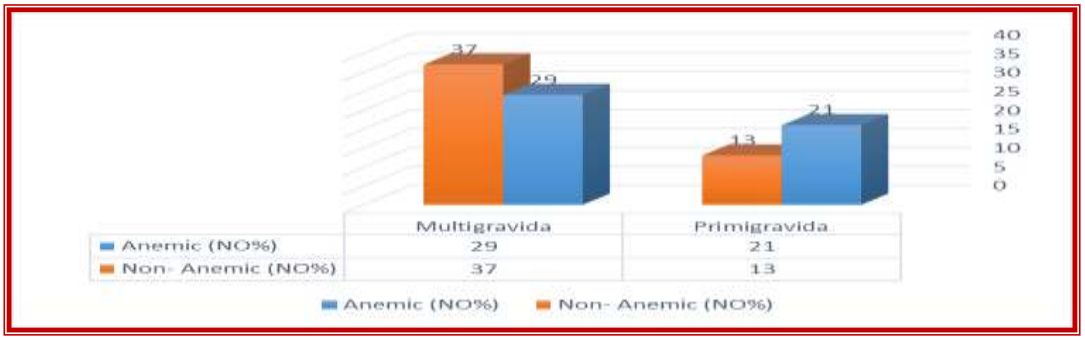

Fig 1:-Association of iron deficiency to mother parity:

In figure (1) show the distribution of iron deficiency anemia according to mother parity which shows that the multigravida include 29 (58%) in the anemia group as compared with 37(74%) in the non-anemic group. The primigravida include 21(42%) in anemic group as compared with 13(26%) in non- anemic group . That mother parity result of this study coincides with the occurrence of anemia in the previous studies which established the prevalence of anemia found significant relation of anemia with mother parity (Mart´ı *et al*.,2002 ;Dim and Onah,2007). Also, another's studies which mentioned to slight, anemia may don't require any outcome on gestation and labor excluding that the mother will require little iron supplies and may become moderately to sever anemic in following gestations (Sharma,2003). Another past study in Jordan reported by AL- Mehaisen et.al.(2011). that the maternal age, gravidity and parity were not significantly associated with anemia . Also, this consistent with other findings which stated that parity were not a significant predicator of anemia in pregnancy (Desalegn,1993).

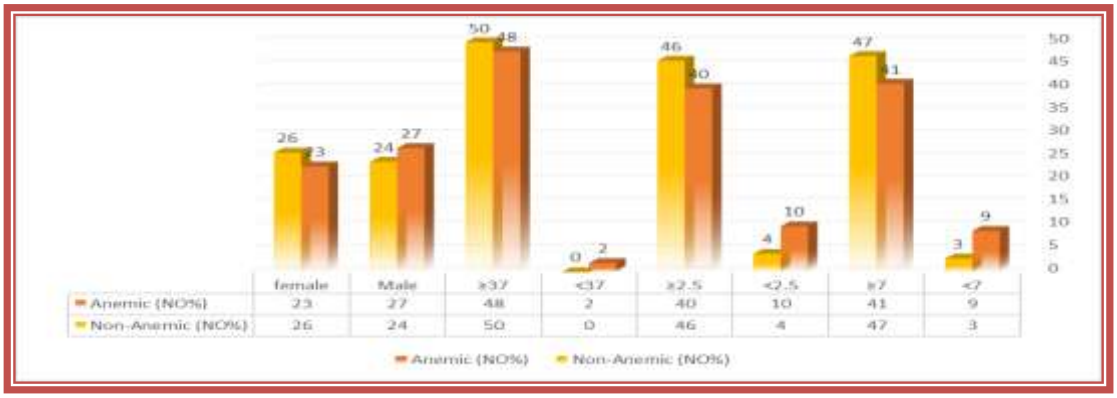

Fig.2 :- The neonatal outcome differences between anemic and non - anemic study group:

In the figure 2 revealed the relation between anemia and neonatal outcome displayed that the apgar score <7 was 9(18%) in anemic group while in non – anemic group was 3 (6%) .The anemia in pregnancy caused pre-placental hypoxia, which might provide increase to fetal hypoxia that affects both mother and neonate (Zhang *et al*.,2009). Apgar score ≥ 7 was 41 (82%) while it was 47 (94%) in non – anemic group . The birth weight in (Kgm) <2.5 was 10 (20%) in anemic while it was 4 (8%) in non- anemic .Also , the weight ≥ 2.5 was 40 (8%) in anemic v.s. 46 (92%) in non- anemic . Past studies mentioned to effect on both the mother and fetus, such as premature delivery, intrauterine growth retardation, and neonatal and prenatal death (Lone et al.,2004 ; Scholl,2005 ; Adam *et al*.,2008). The previous study mentioned to the anemia in pregnancy may have adverse effects on pregnancy, delivery and neonatal infants . Maternal anemia is an autonomous danger influence for pre-term delivery (Levy *et al*.,2005). Also, others past studies mentioned to that little Hb attention through gravidity is associated with enlarged risk for preterm distribution of low birth weight of infant (Kumar *et al*.,2013) . The previous studies mentioned to poor Apcarscore (taken as score less than 7 at 5 minutes), early neonatal death and NICU admission were higher in anaemic group than non-anaemic group and it was statistically significant. (Lone *et al*.,2004 ; Jalil,2007 ; Mahamuda *et al*.,2011 ; Laflamme,2012 ; Ram and Sita,2013) . Another study indicated that the baby born before 28 wks of gestation that death was termed since intrauterine death, while baby born before the end of 37weeks of gestation age was as termed premature and

after 37 weeks of pregnancy was termed normal delivered baby while that the mean weight, Apcar score and hemoglobin equivalent 3 month after delivery were mean complete grander in babies (Sharma and Shanker,2010). In this study gestational age affected by anemia which <37 was 2(4%) in anemic group while no one in non-anemic .The previous studies in which gestational age reported to be affected by anemia (Albsoul *et al*.,2004 ; Haniff *et al*.,2007). Another past study, regardless of motherly iron supplies, the fetus stationary gets iron after motherly transferrin, that was stuck in the placental and that eliminate, at last vigorously conveyances firm to the baby. Progressively such babies incline to have reduced firm stores because reduction of motherly stores. Opposing perinatal consequence in the procedure of pre-term and small-for-gestational-age babies and increased perinatal humanity taxes must remained experimental in the neonates of anemic moms. Iron enhancement to the mom throughout gravidity recovers perinatal consequence. Mean weight , Apgar score and haemoglobin level 3 month after delivery are meaning fully better in offspring of the added collection than the reason group (Nassar *et al*.,2006).. The sex of neonatal was 27 (54%) male sex in anemic v.s. 24 (48%) in non – anemic group while the female sex was found to be 23(46%) in anemic group v.s. 26 (52%) in non- anemic group . The past study which mentioned that the majority of the newborns were male in anemic group (Nassar *et al*.,2006). Statistically there was important changes at ($p.\leq 0.05$).

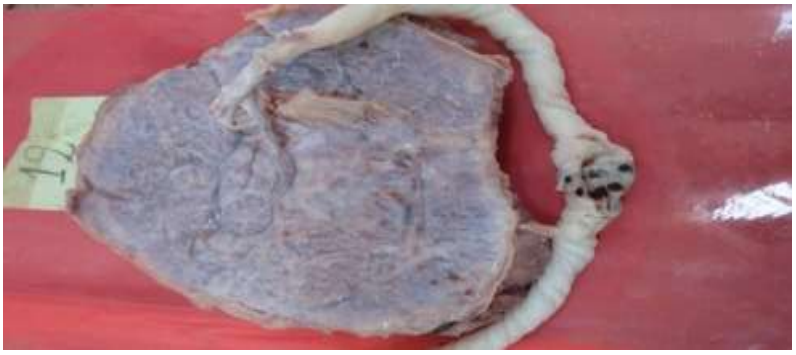

Fig.3:Fetalsurface of placenta with marginal insertion

Fig 4 : Fetal surface of placenta with eccentric insertion of umbilical cord with thrombosis and irregular shape .

Fig 5: Maternal surface with decrease number of cotyledons

Fig.6 , Fig.7 , Fig. 4.8 , Fig. 4.9 :-

Are showing histological findings it was found that the sycytial knots localized aggregation of syncytiotrophoblastic nuclei in a villi only occasionally seen in the control group . The present study deals with the effect of maternal anemia on the histomorphology of placenta and correlating it with fetal outcome for medico legal purposes and the fibrosis may be leading to decrease villous due to decreased number of villi and size . The capillaries per villous increases in number and are dilated with increasing grade of anemia is the principal adaptation to hypoxia . (simpson *et al*.,2003) reported that there is hypoxic hyper capillarization in ischemic placenta . Soni and Nair (2013) said cytotrophoblastic proliferation was for replacement of damaged cytotrophoblast caused by anemia. Cytotrophoblastic hyperplasia is a repair phenomenon and it's an adaptation to physiological stress , and said the fibrosis in non-anemic group scattered and minimal amount of fibrosis were seen where as in anemic group amount of fibrosis tend to increase in villi . Kanfman *et al* (2004). reportedin some area of villi the syncytium becomes a nuclear and attenuated overlying a dilated fetal capillary to form vasculo syncytial membrane for facilitation of gas transfer across the placenta as they represent the site of closest approximation of maternal and fetal blood circulation . Vasculosynsytial membrane is a accommodative effect to face problem of hypoxia (Shema,2013). Another study by Mongia et.al (2012). Showed an increase in the number of syncytial knots, cytotrophoblastic proliferation, basement membrane thickening , forming of vasculosyncytial membrane, capillaries per villi and stromal fibrosis with increase the severity of anemia . Some was observed by other authors that the placenta shows histomorphological changes in anemia which are an adaptation to maternal hypoxia (Burton *et al*.,1996 ; Sharma,2003). Vineeta and Sunita (2000).Observed that there is also cytotrophoblast proliferation with thickening of villious trophoblastic basement membrane as a villous response to decreased utero placental blood flow showing that the placenta is rarely in sufficient.Biswaset. al.(2014) . observed that increased intravillous and perivillous fibrin deposition , increased syncytial knotting and more a vascular villi. These features suggest hypoxia and reduced per fusion .Perivillous fibrin depositions might be acting as a barrier between fetal and membrane circulation , thereby reducing the transfer of the essential nutrients to the fetus , These probably resulted into chronic placental insufficiency , thus causing smaller fetus. When pregnancy is associated with maternal anemia , Villous mass is decreased due to decreased number of villi and their size, leading to increase in intervillous space (Ramic et al.,2006; Robert,2008). Rumana,(2012). Said syncytial knots are composed of aggregates of small closely packed densely staining nuclei protruding from the villous surface into the intervillous space. It's hypothesized that villous hypovascularity leads to formation of aged nuclei is being accelerated so as to use optimally the mount of trophoblast available for transfer purpose. Burton et.al(1981).indicated that the factors responsible for the formation of stromal fibrosis are normal ageing process and reduced uteroplacental blood flow. It's fibrin which is formed by thrombosis of maternal blood in intervillous space.

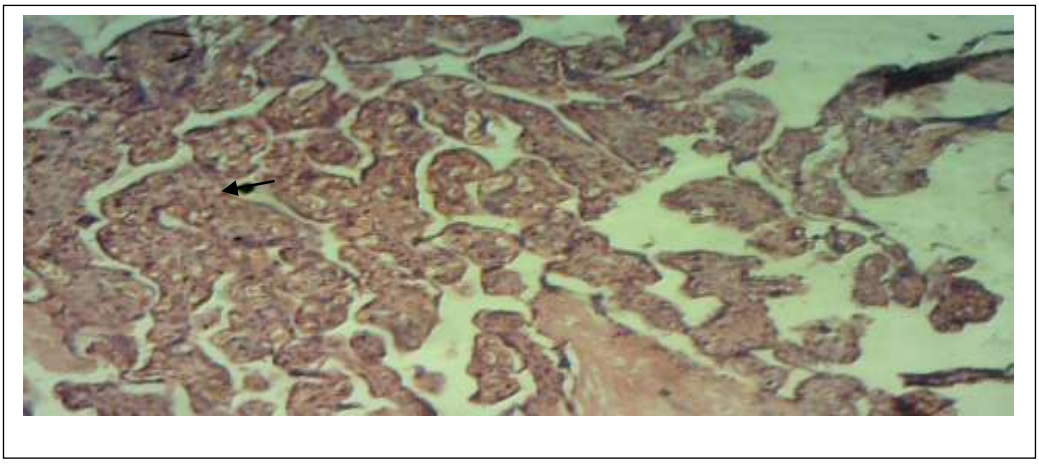

Fig 6: Increased in number of capillaries in each villi of anemic group. Stain used: H & E, x4 .

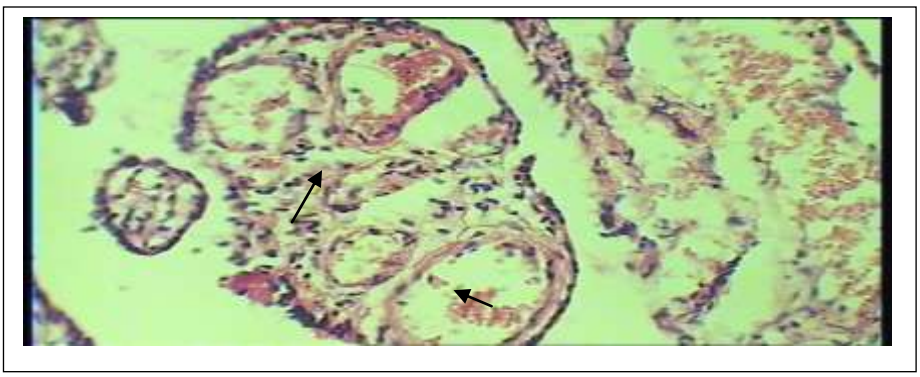

Fig 7: (Arrow) increase cytotrophoblasticproliferation , dilated and increase in number of capillary per terminal villous in anemic group . Stain used: H & E, x10 .

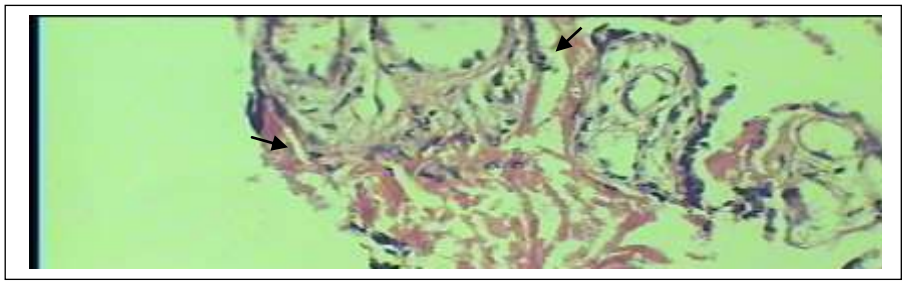

Fig8 : (Arrow) Increase in syncytial knots, thickening of basement membrane of trophoblast, intravillous and perivillous fibrin deposition . Stain used: H & E, x10 .

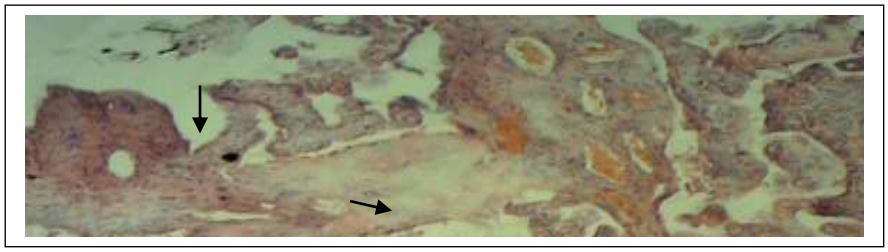

Fig 9 :(Arrow) Amount of fibrosis and increase in intervillous space . Stain used: H & E, x4 .

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