Effects of oligohydramnios on the histological structure of full term placenta

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Abstract

The present study is an attempt to define the histological effects of oligohydramnios on the structure of the full term human placenta and to correlate the histological changes found with the severity of the condition.The study was conducted in Mosul city from March 2006 to August 2006, the placentae were obtained from 40 pregnant women diagnosed by ultrasound to have oligohydramnios and other 40 pregnants having an ultrasound revealing normal amount of amniotic fluid (as a control group). From the group with oligohydramnios 15 pregnants diagnosed to have a history of preeclampsia according to their clinical manifestations of elevated blood pressure (more than 140/90 mmHg), edema of the face and hands and their urine examination for proteinuria (more than 300 mg of protein in a 24 hr.specimen correlated with +1 or +2 reading on dipstick), other 15 pregnants with oligohydramnios were post mature and their gestational age prolonged beyond 41 weeks according to their last menstruation and their ultrasound.The remaining 10 pregnants with oligohydramnios were discovered to have small-for gestational age babies with birth weights less than 2500gm.All the pregnant diagnosed to have oliugohydramnios showed the same placental histological appearance whatever the underlying cause. The specimens of the placenta were prepared for histological examination then the sections were stained with various histological stains (including Haematoxylin and Eosin) and some histochemical stains (such as PAS) then statistical analysis of the clinical observations and histological findings was done to define the significance of each finding (using Chi-square test and Z-test of two proportions) and to correlate the incidence of these changes with the severity of oligohydramnios.The study showed that oligohydramnios is strongly associated with fetal growth retardation as a result of uteroplacental insufficiency when compared with the control group. On microscopic examination of the placentae pronounced histological changes were noted in the group with oligohydramnion, the incidences of these changes increased with increasing severity of the condition. The characteristic changes include: Excessive syncytial knots, cytotrophoblastic hyperplasia, thickening of the trophoblastic basement membrane, stromal fibrosis, fibrin deposition within the villi and in between the cells of decidua basalis, villous edema, arteriosclerosis of the uteroplacental arteries. There was a decrease in the vascularity of the villi of the placentae of the group with oligohydramnios (by counting the number of the capillaries in the villi) in addition to areas of focal hemorrhage in the intervillous space and decidual cell calcification.

Introduction

The placenta was chosen as a model for evaluating the vascular and tissue injuries provoked by oligohydramnios because the effects of oligohydramnios on the structure of human full term placenta were well documented. Oligohydramnios complicates 0.5-5% of all pregnancies however, it is more common in pregnancies beyond term because the amniotic fluid volume normally decreases at term [(28)].

The placenta consists of two components, a fetal part called chorionic frondosum and a maternal part known as deciduas basalis which is regarded as the functional layer of the gravid endometrium [(15)].The amniotic fluid surrounds the fetus throughout pregnancy plays a vital role in the normal fetal growth and development, helps to protect and cushion the fetus and facilitates easy movement within the amniotic sac. Amniotic fluid is produced by the fetal lungs and kidneys, then it is taken up by fetal swallowing and sent across the placenta to the mother's circulation [(6)], the normal amount of amniotic fluid may varies but generally, the normal pregnant women carries about 500 ml of amniotic fluid at term[(26)].

Oligohydramnios is a condition characterized by diminished amount of amniotic fluid less than 200 ml [29], it occurs secondary to either an excessive loss of amniotic fluid or a decrease in the fetal urine production [30]. The routine use of ultrasonography has been considered as a safe, repeatable, and reliable method for measuring the amniotic fluid volume as well as screening for congenital anomalies both associated and non associated with oligohydramnios [26].By ultrasound, the characteristic features of oligohydramnios included the absence of fluid pockets throughout the uterine cavity, crowding of the fetal limbs, absence pockets surrounding the fetal legs and overlapping of the fetal ribs in severe cases [4].The ultrasonographic diagnostic findings observed in cases of oligohydramnios include: maximum vertical pocket (MVP) of less than 2 cm and amniotic fluid index of less than 5 cm or less than the 5th percentile [23].

Generally, oligohydramnios is related to the premature rupture of amniotic membrane, congenital absence of functional renal tissues (like urinary tract malformation including renal agenesis, cystic dysplasia) or obstructive uropathy as in case of uretral atresia [30].Furthermore, a chronic reduction in the urine production may be secondary to decreased renal perfusion as a result of hypoxemia as in cases of preeclampsia and intrauterine growth retardation [5], post term gestation is also associated with oligohydramnios as a result of uteroplacental insufficiency [11].

Twin –to- twin transfusion syndrome is a syndrome occurs in monochorionic twin pregnancy which develops a vascular connection through the chorionic plate of the placenta, this results in shunting of the blood to the other twin [17] thus the recipient member of the twin becomes plethoric and hypervolemic while the doner twin becomes anemic, hypovolemic and growth retarded, these features were accompanied by polyhydramnios in the sac of the recipient and oligohydramnios in that of the doner [21]. The condition is usually confirmed after birth by the discovery of hemoglobin difference >5gm /dl and birth weight >20% between the members of the twin [8].

Patient and Methods

Histological study was carried out on the placentae obtained from pregnant women in the last four weeks of their gestation. The specimens were obtained from Al-Batool and Al-Khansaa Teaching Hospitals in Mosul, between March (2006) and August (2006) and studied in the Department of Anatomy, College of Medicine, University of Mosul.

eighty placentae were used in this study, 40 placentae were collected from pregnant women who were diagnosed by antenatal ultrasound examination to have oligohydramnios in their last trimester and other 40 placentae were collected from women who had normal amount of amniotic fluid and had no other complication throughout their pregnancy (as a control group).

Criteria of oligohydramnios suggested by ultrasound applied in this study include:

Diminished amniotic fluid volume <500 ml. Maximum vertical pocket (MVP) <2 cm. Amniotic fluid index <5 cm or <5th percentile.

Forty women were diagnosed to have oligohydramnios clinically and by ultrasound, 15 of them were discovered to have a history of preeclampsia through measuring their blood pressure and urine examination for proteinuria and history of generalized edema particularly of the hand and face. Other 15 pregnant women were postmature and their gestational age was beyond 41 weeks, the remaining 10 pregnant had ultrasound revealed oligohydramnios and manifestations of intrauterine growth retardation thus the birth weight of their newborn babies was <2500 gm.

A complete record for every pregnant woman was reported including:

Name, age, parity, gestational age (estimated by taking into account the menstrual history, early ultrasound measurements and clinical examination), blood pressure, any medication taken, history of generalized edema and edema of the hand and/or face, ultrasound findings (oligohydramnios, amniotic fluid index, congenital anomalies including renal agenesis, polycystic kidney, urethral obstruction), review of the past medical history, obstetric history (abortion, dead

babies, oligohydramnios in the previous pregnancy), investigations including ultrasound, urine examination for proteinuria, fasting blood sugar during pregnancy, in addition to any antepartum complications such as preterm labour, diabetes mellitus, placenta praevia, fetal anomalies and abruptioplacenta.

80 full term newborn babies were delivered 40 of them from the control group and the other 40 babies from the group with oligohydramnios. Information was recorded for the newborn babies about their birth weights, mode of delivery, single or twin, any complications of labour, and Apgar scores.

Following delivery of the fetus and the placenta two pieces were chosen from each placenta, one from the fetal surface and the other from the maternal surface, then the specimens were put in a fixative solution (10% neutral formalin) for 24 hour then each specimen was cut into 1 cm thick slices and dehydrated in graded alcohol solutions (70% alcohol for overnight, two changes in 90% alcohol one hour for each and two changes in 100% alcohol for two hours).then the specimens were immersed in xylene using three changes with one-hour interval for each.

Complete removal of the clearing solution was made by immersing the tissue specimens into three successive paraffin bathes in oven, one hour for each. Finally paraffin blocks were prepared by embedding the tissue specimens using paraffin wax (melting point is 55-60co) and these paraffin blocks were now ready for sections using Reichert Rotary Microtome, serial paraffin sections of 4 micrometers in thickness were cut from each block, the sections were collected and mounted (using DPX) on glass slides. Then the sections were stained with various histological stains including Haematoxylin and Eosin and PAS (20).

Statistical methods were used for the analysis of the collected data (16) including the mean and standard error of mean. Chi-square test was used to compare the results of the clinical and histological parameters for the study group and Z-test of two proportions test was used to identify statistical differences in the mean values of the various parameters, the differences were considered as significant if P-value <0.05 and highly significant if P-value <0.001 and not significant if P >0.0

Results

The average maternal age in the pregnant women diagnosed to have oligohydramnios was 27.8±0.9 years, while it was 22.15±0.5 in the control group, which was not significantly different (P>0.05) (Table 1).

The average gestational age in the group diagnosed to have oligohydramnios group was 37.5±0.2 weeks and 37.9±0.2 weeks in the control group, the difference was not significant (P>0.05) (Table 1).

The mean birth weight of the babies delivered in women with oligohydramnios was 3.2±0.05 Kg, and this figure compared with the mean birth weight of the babies delivered from the control pregnant women which were 3.5±0.05 Kg. The difference was highly significant indicating a significant association of low birth weight with the severity of oligohydramnios (Table 1).

The mean Apgar scores at first minute for the babies of women with oligohydramnios was 6.15±0.1, while in the control pregnant women, the mean Apgar scores at first minute was 7.88±0.2. This difference was statistically significant (P<0.05) indicating a significant association of Apgar scores with the severity of oligohydramnios (Table 1).

Gross examination of the placentae showed normal circular shape of the placental disc in all cases, all placentae were covered with translucent smooth membrane and the site of insertion of the umbilical cord was central in all the cases.

Light microscopical examination of sections obtained from the fetal surface of each placental disc was made with H&E perpetration, special attention was first directed to the examination of the chorionic villi. Each chorionic villous appeared as vascular villous stroma covered with multinucleated syncytiotrophoblastic layer with indistinct cell boundaries and darkly stained nuclei. The villous stroma was composed of connective tissue core having few bundles of collagen fibers and flattened fibroblasts. The villous stroma contained 2-6 dilated capillaries filled with fetal blood (Fig.1). On the other hand, a section of the

maternal surface showed large polygonal, sometimes irregular shaped decidual cells with large nuclei and foamy cytoplasm. These cells are surrounded with little amount of collagen fibers, normally small amount of fibrinoid material was deposited in between the decidual cells (Fig.2)

On examination of the placentae obtained from the women with oligohydramnios using light microscope, the following histological changes with different proportions were demonstrated:

Excessive Formation of Syncytial Knots: These are focal clumps of syncytial nuclei that protrude into the intervillous spaces from the surface of the villi observed in (55%) of the placentae obtained in the oligohydramnios and in (25%) of the control group, the difference was statistically significant (P-value <0.05), (Fig.3), (Table 2).

Cytotrophoblastic Hyperplasia: An excessive number of cytotrophoblast cells were observed in 57.5% of the oligohydramnios and 10% only of the control group, the difference was highly significant (P< 0.001), (Fig. 4) (Table 2).

Thickening of Trophoblastic Basement Membrane: This change was difficult to be demonstrated using routine H&E stain, however, using PAS stain it becomes clear. Thickening of trophoblastic basement membrane is regarded as an abnormal finding if it affects more then 30% of the total villous population. It was difficult to find placentae with thickening of the trophoblastic basement membrane affecting more then 30% of the villi in the control group (0%). This change was found in 57.5% of the oligohydramnios group which was highly significant (P < 0.001), (Fig.5), (Table 2).

Hypovascular Villi: Some chorionic villi are small and hypovascular showing small non-dilated capillaries (Fig. 6).This finding was seen in 15% of the control group, 35% of those with oligohydramnios which was statistically non significant (P-value >0.05), (Table 2).

Stromal Fibrinoid Deposition: Stromal fibrinoid deposition appears as small eosinophilic hypocellular PAS positive masses attached to the villous surface often at the sites of discontinuity in the syncytiotrophoblasts. Perivillous fibrinoid deposition affecting more than 3% of the villi was found only 25% of the control group while in placentae obtained from the group with oligohydramnios, fibrinoid deposition dose not only involve the chorionic villi (Fig.7) but also occurs at an increased rate in the maternal decidua (Fig.8).Occasionally a large fibrinoid nodule might be observed indenting the villous stroma (Fig.9) thus this finding is regarded as a highly significant change (P < 0.001), (Table 2).

Excessive Stromal Fibrosis: Excessive amount of collagen fibers in the stroma of the villi was easily demonstrated. Stromal fibrosis was observed only in 10% of the control group, but this figure increased up to 47.5% of those with oligohydramnios which is also regarded as highly significant change (P < 0.001) (Fig.10 and Fig.11), (Table 2).

Villous Edema: This change was easily seen in placentae stained with routine H&E stain, it was seen in 15% of those with oligohydramnios. However, it was difficult to find placentae with villous edema in the control group which made this lesion a highly significant one (P < 0.001), (Fig. 11), (Table 1).

Arteriosclerosis of the Fetal Capillaries: Arteriosclerosis of the uteroplacental arteries was demonstrated in 25% of the control group and 45% of the group with oligohydramnios (Fig.12) and it is regarded as a significant change (P < 0.05) (Table 2).

Discussion

Oligohydramnios is regarded as one of the pregnancy complication that affects both the mother and the fetus due to uteroplacental insufficiency which results in suppressed fetal growth, it is secondary to either an excessive loss or reduced production of amniotic fluid. However, it is generally related to the rupture of amniotic membrane (ROM), congenital absence of functioning renal tissues or obstructive uropathy which either prevent the formation of urine or the entry of urine into the amniotic sac like renal agenesis, cystic dysplasia and uretral atresia.

On the other hand, any condition causing chronic reduction in the fetal urine

production might be secondary to the reduced renal perfusion causing the problem of oligohydramnios as a consequence of hypoxemia-induced redistribution of fetal cardiac output as in case of preeclampsia and growth retarded fetuses also post-term gestation is associated with oligohydramnios as well as twin-to-twin transfusion syndrome.

In this study 15 cases of the group diagnosed to have oligohydramnios using ultrasound were discovered to have a history of preeclampsia. Preeclampsia is a multisystem disorder characterized by widespread dysfunction of maternal vascular endothelium due to placental ischemia [(24)].Endothelial dysfunction in turn, leads to reduced formation of vasodilators and enhanced formation of vasoconstrictors such an imbalance causes hypertension by impairing renal pressure natriuresis and increasing peripheral vascular resistance [(14)].

A characteristic renal lesion in preeclampsia is termed as "glomerendotheliosis" due to enlargement of the glomeruli and widening of the capillary lumina, thus both glomerular filtration rate and renal blood flow decrease in preeclampsia leading to oliguria which is subsequently manifested as oligohydramnios in later stage of preeclampsia.

Other 15 cases of those women diagnosed to have oligohydramnios were post term and their pregnancy prolonged beyond 42 weeks, the cause of oligohydramnios in this group is unknown. The most possible explanation is the reduced efficiency of placental function thus reduced fetal renal blood flow and decreased fetal urine output and subsequent oligohydramnios have been demonstrated in the pregnancies beyond 42 weeks, this finding agrees with [(1)].

The remaining pregnant women delivered newborn babies who showed manifestations of intrauterine growth retardation by their ultrasound prenatally and low birth weight <2500 gram postnatally.The explanation of this finding is that in the growth restricted fetuses, chronic hypoxia results in shunting of fetal blood away from the kidneys to the more vital organs leading to reduced blood flow to the kidneys and then reduced filtration rate and urine output leading to oligohydramnios.This observation agrees with that noticed by [(4)].

In the current study we demonstrate a tendency for excessive syncytial knotting in placentae obtained from the group with oligohydramnios, this highly significant finding was attributed to the trophoblastic hypoxia and placental ischemia. This observation was also reported by [(27)] who had related this change to occlusion or narrowing of the uteroplacental vasculature that lead to placental ischemia. Fox, (1997) observed that with hypoxia the syncytial nuclei of most of the villi will be clustered together at one pole carrying with them small amount of cytoplasm leading to knot formation. However Kaufmann et.al, (1987) explained this tendency for excessive syncytial knotting on the basis of villous mal-development.

On the other hand, hypoxia appears to stimulate trophoblast apoptosis (10) and this could increase not only the formation of syncytial knots but also the extrusion of syncytial fragments into the intervillous spaces (22).

The current study also showed an increase number of villous cytotrophoblastic cells (cytotrophoblastic hyperplasia), this is the result of more proliferation of these cells in response to syncytial injury and placental hypoxia and this observation is in agreement with what has been reported by [(13)] who showed that in vitro hypoxia will shift the balance between the proliferative and invasive phenotypes of cytotrophoblast cells toward the proliferative one. Fox, (1978) observed that few cytotrophoblastic cells remain in the mature placenta and after trophoblast injury this layer will be activated, and serves as a regenerative layer for the damaged or necrotic syncytium, this excessive proliferation of cytotrophoblasts increases progressively with the severity and duration of the disease.

Other microscopical finding in the present study was undue thickening of the trophoblastic basement membrane, this is explained to be a response to the uteroplacental ischemia, similar change in the trophoblastic basement membrane previously observed in the placenta obtained from the smoker mothers, a condition, which is also regarded as a cause of uteroplacental hypoxia [(2)], this finding strongly reflects an impaired fetal placental blood flow and low

oxygen tension particularly in the heavy smokers at the third trimester [9].This change was further explained in relation to the cytotrophoblastic hyperplasia because the basement membrane is secreted by the cytotrophoblast cells thus the proliferative activity of these cells under ischemic conditions is accompanied by an excessive secretion of the basement membrane substances [1].

Stromal fibrosis was frequently seen in placentae obtained from those with oligohydramnios, such lesion is mainly attributed to the reduced villous perfusion.The villous stroma of the placenta serves to support the overlying trophoblasts, and showed marked fibrosis in response to any interruption in the fetal circulation due to activation of fibroblasts and consequently excessive collagen fibers deposition.This finding was in agreement with that observed by [7].Other hypoxic conditions that might affect the placenta also appear to be associated with excessive collagen fibers deposition in the villous stroma and this is evident in cases of maternal malnutrition and pregnancy in high altitude [3].

Occasional villi showing stromal fibrinoid deposition seen in the placentae from women with oligohydramnios.The most acceptable explanation attributes fibrinoid deposition to a mechanical defect in the blood flow to the intervillous spaces, stasis of maternal blood then subsequent clotting, this usually occurs following trophoblastic injury which will stimulate the cleavage of fibrinogen into fibrin that would then be deposited into the trophoblastic basement membrane [24].

Villous edema is seen in an increasing rate in cases of oligohydramnios associated with preeclampsia, it is attributed to the vascular endothelial injury which is associated with disturbances in the vascular endothelial tight junctional complexes with a concomitant loss of regulated protein and fluid transport which leads to extravasation of fluid and protein from the intravascular compartment (14).

Another highly significant lesion was the arteriosclerosis of the uteroplacental arteries with the accumulation of collagen and elastic fibers in the wall of the spiral arteries.This finding agrees with that observed by [19] who noticed that arterial wall sclerosis was regarded as a marker of increased activity of fibroblasts in response to hypoxia which occur in certain conditions associated with pregnancy such as preeclampsia, intrauterine growth retardation and postmaturity. Villous hypovascularity was common in the placentae obtained form women with oligohydramnios, terminal villi of the mature placenta usually contained between 2-6 capillaries, less than this number with the presence of small and non dilated capillaries were the characteristic features of these placentae.

The extent of this vascular lesions correlates well with the severity of oligohydramnios and the likelihood of the clinical diagnosis by ultrasound and all these histological findings in oligohydramnios whether it was associated with preeclampsia, intrauterine growth retardation or postmaturity were accelerated with the progressive impairment of the uteroplacental circulation to the fetus.

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Table (1): Clinical Criteria in the Study Groups

Parameters	**Control (n=40) Mean ±SE**	**Oligohydramnios (n=40) Mean ±SE**	**P-values**
Maternal Age	22.15±0.54	27.82±0.96	0.22
Gestational age	37.97±0.20	37.55±0.22	0.30
Birth weight	3.50±0.05	3.28±0.05	0.001
Apgar scores	7.88±0.20	6.15±0.14	0.03

Table (2): Frequency of Various Histological Findings in the Study Groups:

Histolo. finding	***Control n= 40(%)***	***Oligohydramnios n= 40(%)***	***P-value***
Excessive syncytial knotting	5 (25 %)	22 (55 %)	0.004
Cytotrophoblast hyperplasia	2 (10 %)	23 (57.5 %)	0.001
Thickening of villous BM	0 (0 %)	23 (57.5 %)	0.001
Hypovascular villi	3 (15 %)	14 (35 %)	0.180
Excessive fibrinoid deposition	5 (25 %)	22 (55 %)	0.014
Stromal fibrosis	2 (10 %)	19 (47.5 %)	0.001
Villous edema	0 (0 %)	6 (15 %)	0.011
Arterial sclerosis	5 (25 %)	18 (45 %)	0.039

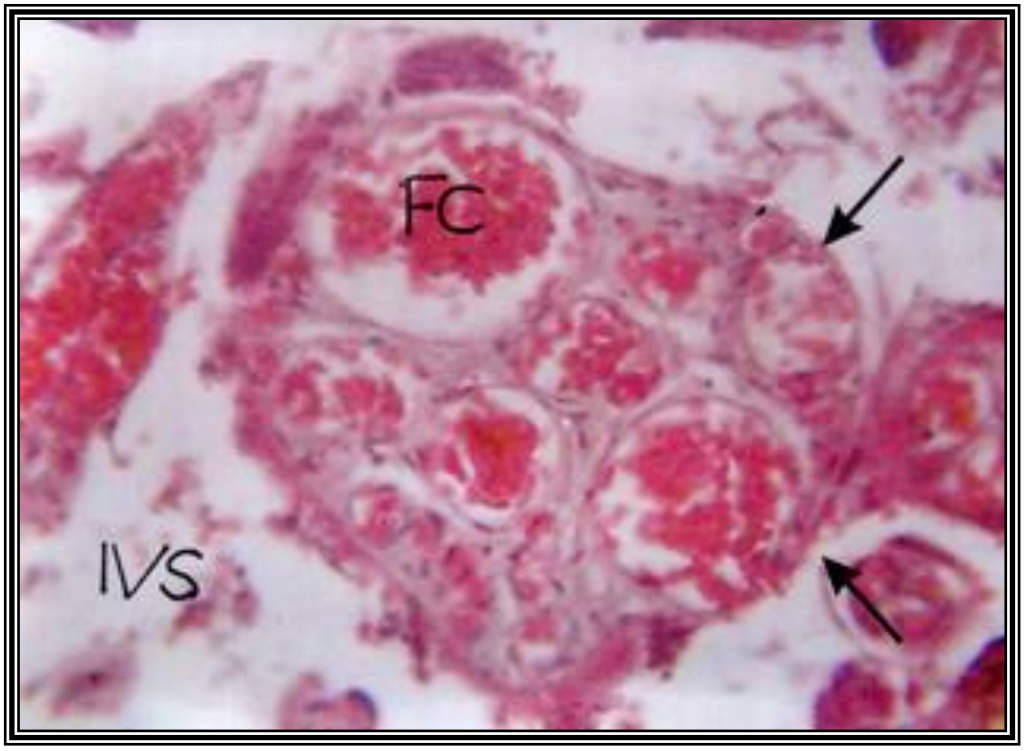


Figure (1): Photomicrograph of normal term human placenta, shows the vasclo syncytial membranes (arrows). Numerous fetal capillaries (FC) are seen within the villous stroma. Villi are surrounded by the intervillous space (IVS), (H&E X 400).

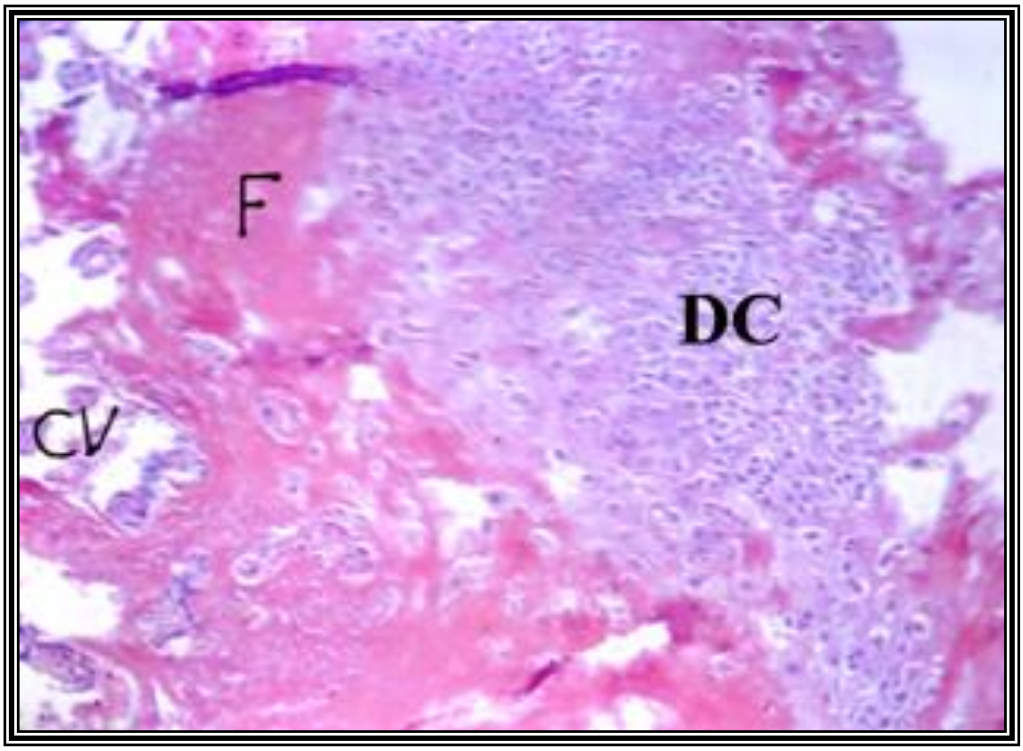
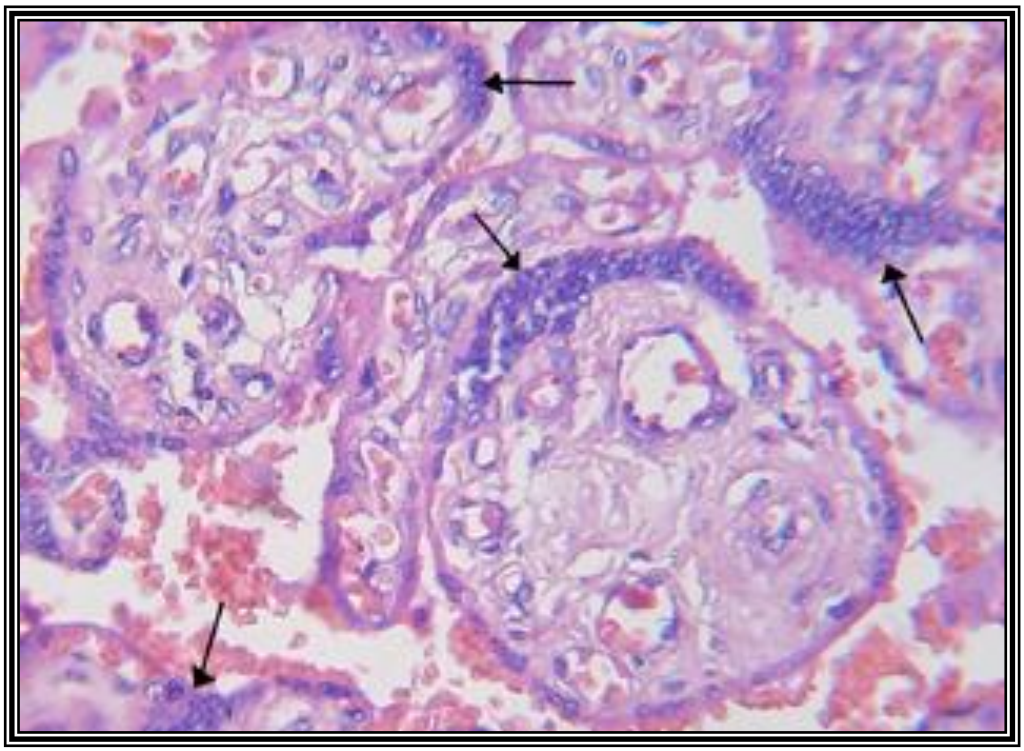


Figure (2): Photomicrograph of normal term human placenta shows decidual cells(DC) surrounded by connective tissue and separated from the chorunic villi(CV) by area of fibrinoid (F),



(H&E X100).

Figure (3): Light microscopic appearance of the placenta from the group with oligohydramnios shows excessive syncytial knotting on the surface of the chorionic villi (arrows) (H&E X 400).

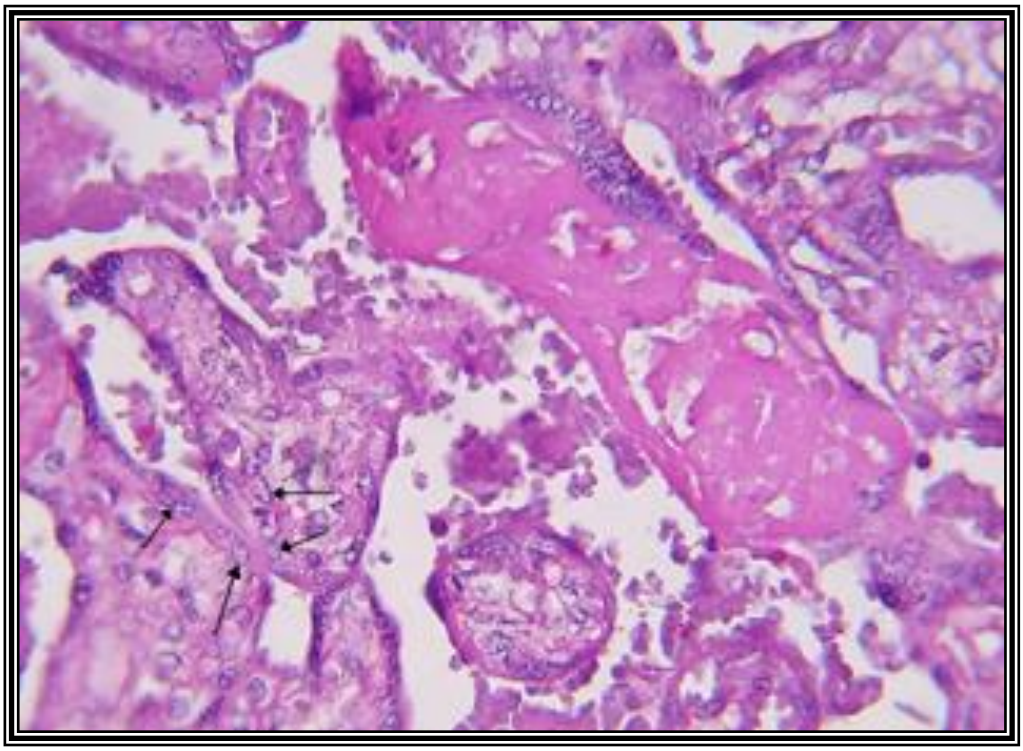


Figure (4): Light microscopic appearance of the placenta from the group with oligohydramnios shows cytotrophoblastic hyperplasia (arrows) (PAS X 400).

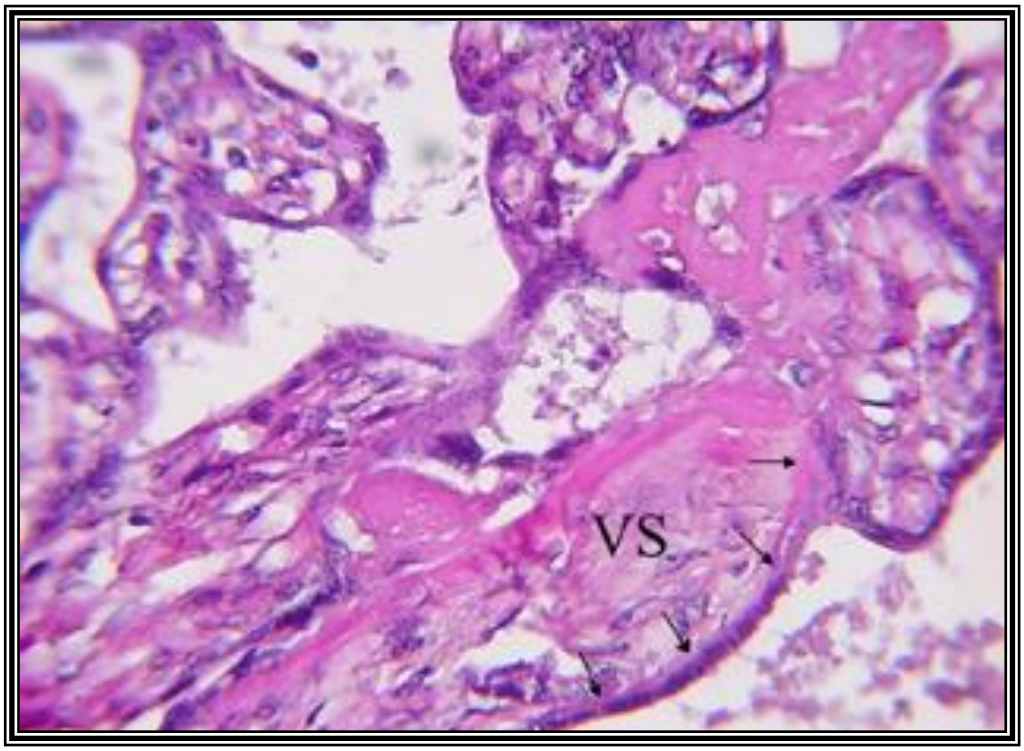


Figure (5): Light microscopic appearance of the placenta from the group with oligohydramnios shows increased thickening of trophoblastic basement membrane (arrows) that separates the villous stroma (VS) from the overlying trophoblastic cells (PAS X 400).

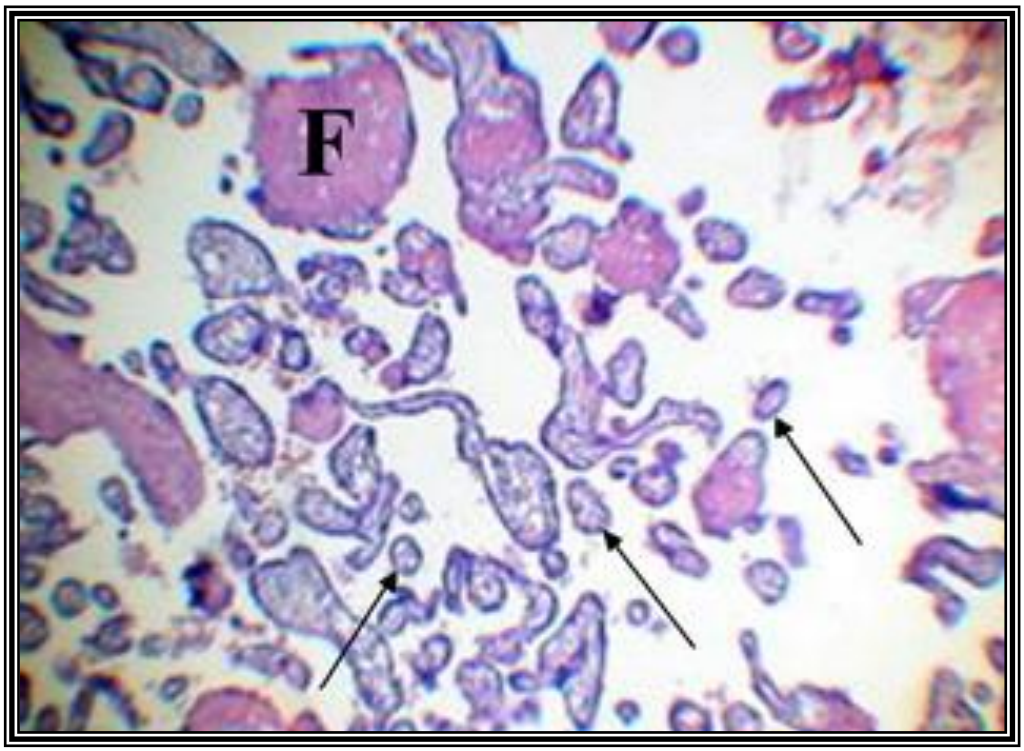


Figure (6): Light microscopic appearance of the placenta from the group with oligohydramnios shows small size hypovascular chorionic villi (arrows), (H&E X 35).

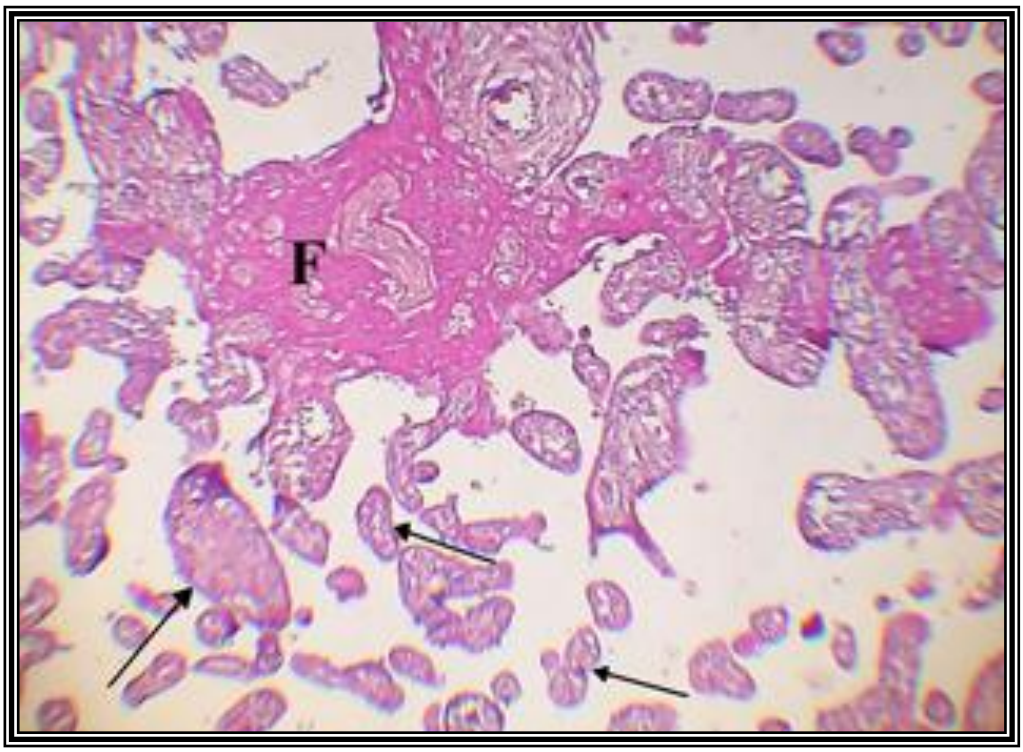


Figure (7): Light microscopic appearance of the placenta from the group with oligohydramnios shows multiple hypovascular chorionic villi (arrows) with an increased villous fibrinoid deposition (F) (PAS X 100).

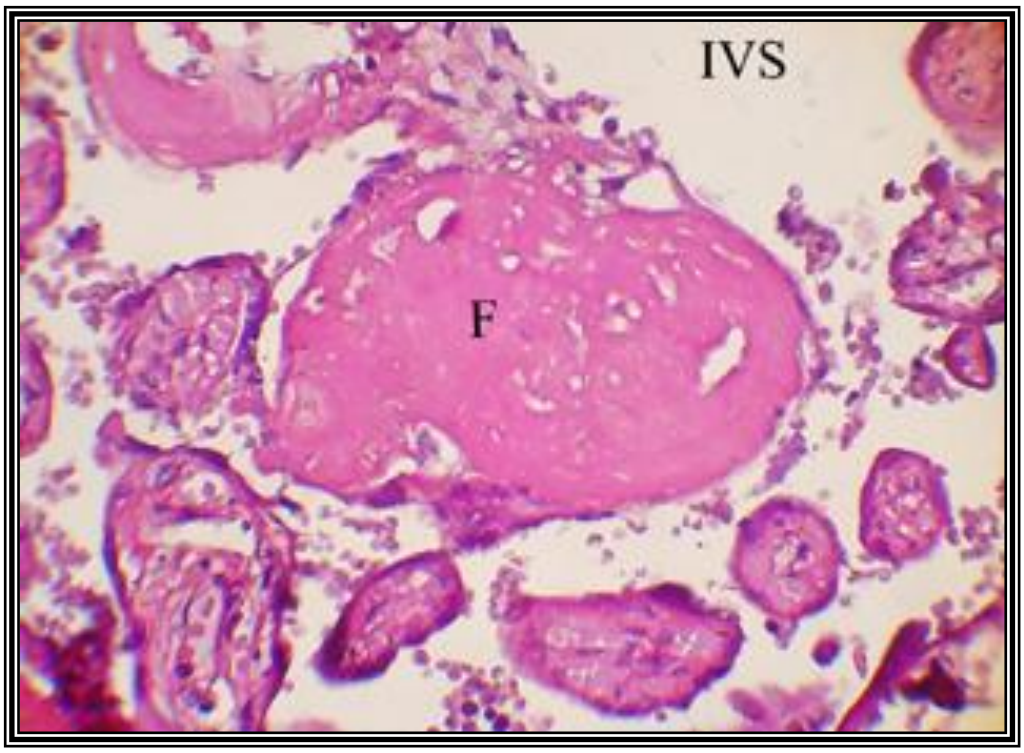


Figure (8): Light microscopic appearance of the placenta from the group with oligohydramnios shows a large fibrinoid nodule (F) indenting the villous stroma, the villi are surrounded by the intervillous space (IVS) (PAS X 400).

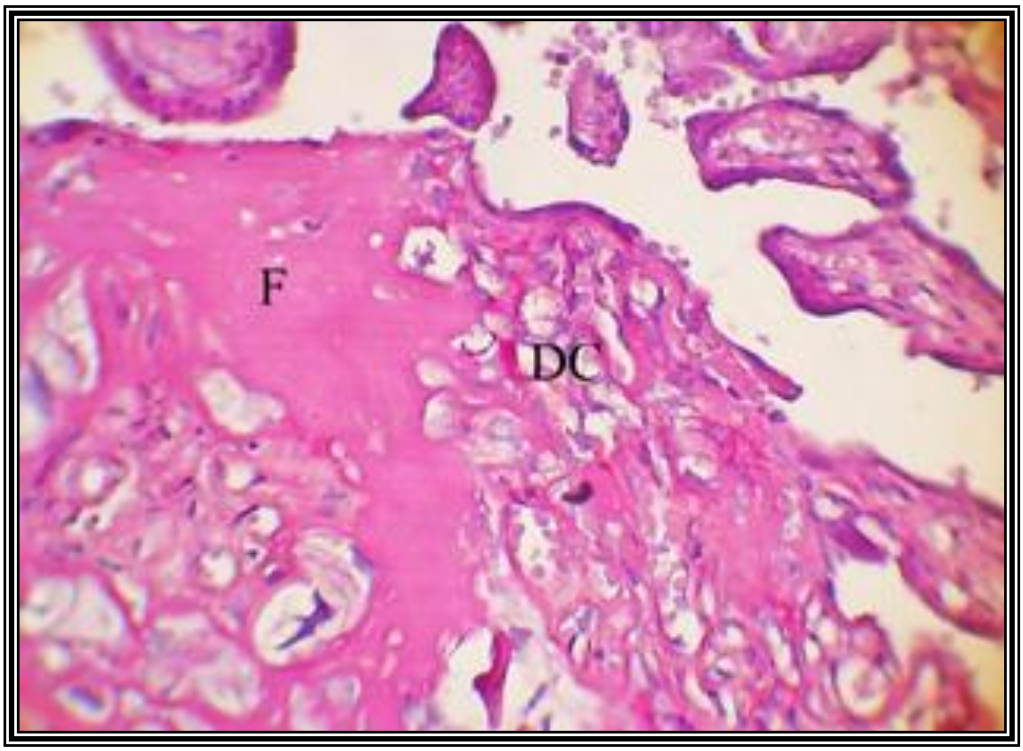


Figure (9): Light microscopic appearance of the placenta from the group with oligohydramnios shows fibrinoid deposition (F) in between the decidual cells (DC) which appear as (PAS-positive) homogenous material (PAS X 400).

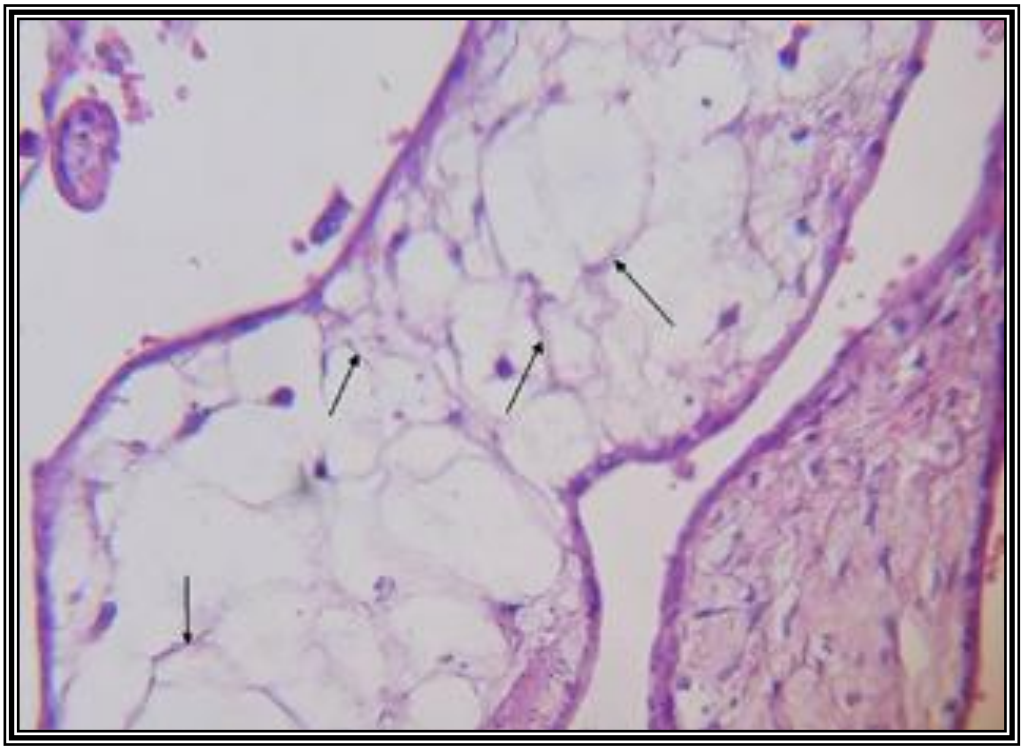


Figure (10): Light microscopic appearance of the placenta from the group with oligohydramnios shows avascular chorionic villi with stromal fibrosis (arrows) ,(H&E X 400).

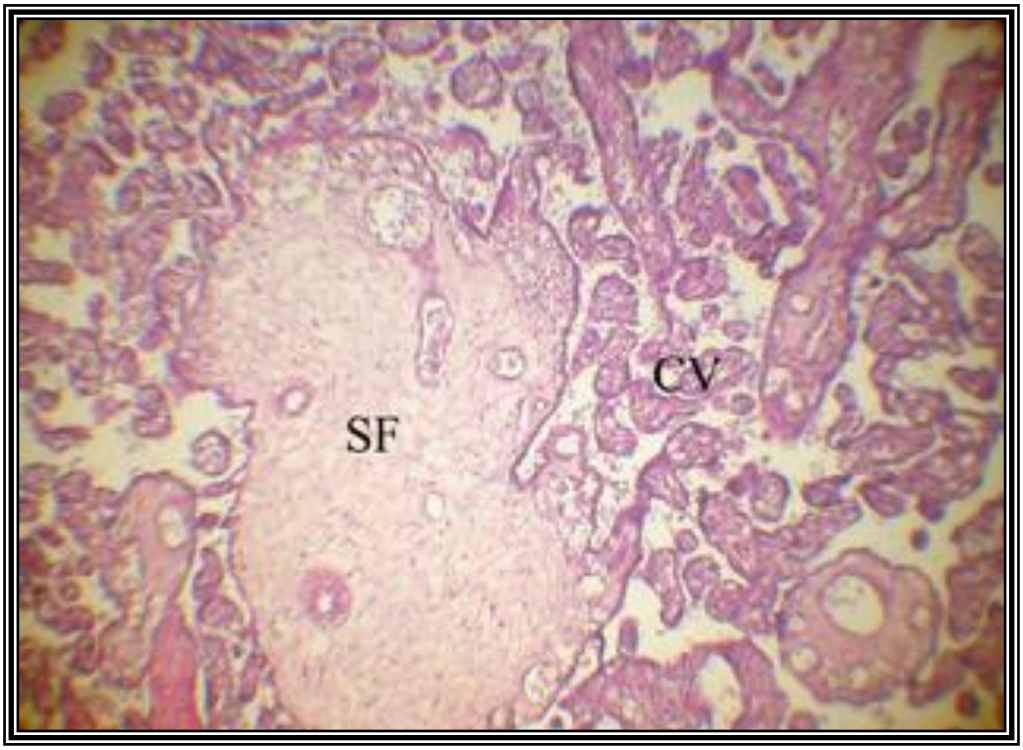


Figure (11): Light microscopic appearance of the placenta from the group with oligohydramnios shows severe villous edema with stromal fibrosis (SF) affecting the stroma of the chorionic villi (CV) (H&E X 100).

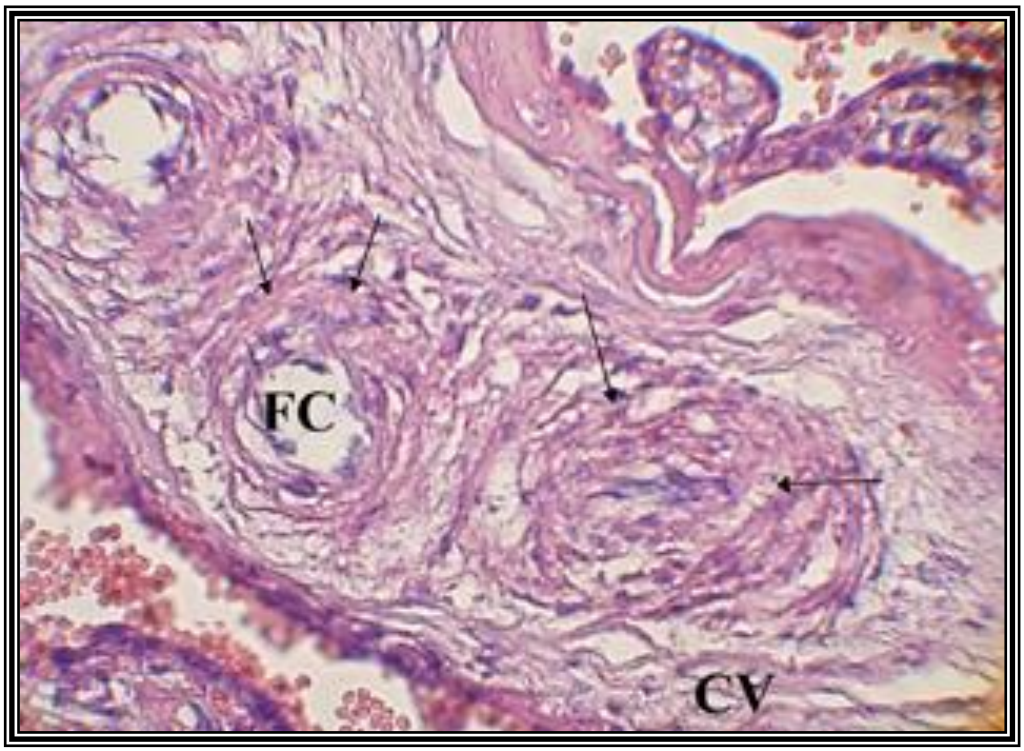


Figure (12): Light microscopic appearance of the placenta from the group with oligohydramnios shows arteriosclerosis of the chorionic fetal capillaries (FC) inside the chorionic villi (CV) with increased thickness of their wall (arrows) (H&E X 400).