

HISTOLOGICAL STUDY OF PLACENTA AND ITS APPENDAGES IN CASES WITH PREMATURE RUPTURE OF MEMBRANES

by

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Many complications, both maternal and foetal, have been attributed to premature rupture of membranes. These range from premature labour, prolonged labour, dry labour, maternal infections and increased chances of operative deliveries in the mother to intrapartum pneumonias, prematurity, neonatal infections, inflammations of the placenta and cord and the like, in the neonate.

It is obvious that with the antiseptic barrier removed, the chances of inflammation of the genital tract are increased. These would naturally reflect directly on the histological structure of the placenta and its appendages and indirectly on the foetal status. It is with a view to study these changes that the present study was undertaken.

Material and Methods

A total of 120 cases under observation comprised of 70 cases of premature rupture of membranes and 50 normal controls.

The placentae and its appendages were initially submitted to a gross examination to observe the general appearance, colour, size, weight, cord insertion and length and any other gross abnormality.

Subsequently, a thorough histological examination of the placenta, cord, membranes and decidua was done.

Observations

The weight of the placenta varied between 950 to 1150 grams in controls as well as in the group of premature rupture of membranes. There was no significant difference between the weight of the placentae in the two groups. In 8 study group cases, areas of haemorrhage and infarction were seen on the maternal surface of the placenta.

In majority of the control cases, the membranes were normal but in the study group cases which showed evidence of intrauterine infection, the membranes were dull, ragged, shaggy and dirty in appearance. The average length of the

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umbilical cord in both the groups was 38 cm.

In the control group, the cord was attached eccentrically in 14 cases (28 per cent) and centrally in 36 cases (72 per cent). In the study group, the umbilical cord was attached to the placenta eccentrically in 28 cases (40 per cent) whereas in 42 cases (60 per cent), it was attached to the centre of the placenta.

the Decidua in the control group cases was only mild to moderate in 15 cases (30.0 per cent), whereas in the study group, the inflammation varied from 24 cases (34.4 per cent) in the mild, 15 cases (21.4 per cent) in the moderate and 3 cases (4.2 per cent) in the severe inflammation group.

In the control group, 10 cases (20 per cent) showed moderate inflammatory changes in the foetal membranes while

TABLE I

Frequency of Inflammatory Response in Decidua, Foetal Membranes, Placenta and Umbilical Cord in 70 Cases of Premature Rupture of Membranes as Compared with 50 Cases of Control Group

	Study Group		Control Group			
	No. of cases	Percentage	No. of cases	Percentage	No. of cases	Percentage
Inflammatory Decidual Response						
Inflammation						
Mild	24	34.4%	9	18%	—	—
Moderate	15	21.4%	6	12%	15	30%
Severe	3	4.2%	—	—	—	—
No Inflammation	—	—	—	—	35	70%
Inflammatory Foetal Membrane Response						
Inflammation						
Mild	15	21.4%	—	—	—	—
Moderate	11	15.7%	10	20%	10	20%
Severe	2	2.9%	—	—	—	—
No Inflammation	—	—	—	—	40	80%
Placental Inflammatory Response						
Inflammation						
Mild	23	32.2%	7	14%	—	—
Moderate	5	7.8%	3	6%	10	20%
Severe	—	—	—	—	—	—
No Inflammation	—	—	—	—	40	80%
Umbilical Cord Inflammatory Response						
Inflammation						
Mild	15	21.0%	5	10%	—	—
Moderate	3	4.8%	—	—	5	10%
Severe	—	—	—	—	—	—
No Inflammation	—	—	—	—	45	90%

in the study group, 15 cases (21.4 per cent) showed mild, 11 cases (15.7 per cent) moderate and 2 cases (2.9 per cent) severe inflammatory changes (Table 1). In the remaining 42 cases in the study group, no inflammatory changes were found (Table 1).

In the placentae of the control group, 7 cases (11 per cent) showed mild changes and 3 (6 per cent) moderate inflammatory changes. In the study group on the other hand, 23 cases (32.2 per cent) showed mild, 5 (9.7 per cent) moderate and 42 (60 per cent) no inflammatory changes.

Inflammation in the umbilical cord was observed in 18 cases (25.8 per cent) in the study group as compared with 5 cases (10 per cent) in the control group. Inflammation in the umbilical cord was about 2.5 times that of the control group (Table 1).

The overall incidence of inflammation in the placenta, membranes and umbilical cord is presented in Table II. Deciduitis, chorioamnionitis, placentitis and inflammation of the cord were present, in 60 per cent, 40 per cent, 40 per cent and 25.8

per cent of the cases in the study group and 30 per cent, 20 per cent, 20 per cent and 10 per cent of the cases of the control group, respectively.

Table III depicts the incidence of perinatal mortality in relation to the histopathological changes. There is a direct relationship between the extent of degenerative changes and perinatal mortality. It is observed that in the cases where deposition of fibrin and intervillous fibrin deposition was present, there was no perinatal mortality but there was a high perinatal mortality with severe changes like ischaemic necrosis, diffuse calcification and endarteritis.

Discussion

Placental abnormalities are associated with a number of obstetrical complications. However, the cause and effect relationship between premature rupture of membranes and alterations in the placental structure and function remains a mystery.

The present study comprises of 70 cases of premature rupture of membranes in whom the histological and morphological

TABLE II
Histopathological Changes in the Placenta in Premature Rupture of Membranes and in the Control Group

	Study Group		Control Group	
	No. of cases	Percentage	No. of cases	Percentage
Deposition of fibrin	2	2.84%	2	4%
Intervillous thrombosis	2	2.84%	—	—
Intervillous fibrin deposition	4	5.71%	2	4%
Ischaemic necrosis	3	4.28%	—	—
Calcification	6	8.57%	—	—
Endarteritis	4	5.71%	—	—
No changes	49	70.00%	48	96%
Total	70		50	

TABLE III
Incidence of Foetal and Perinatal Mortality in Relation to Histopathological Changes

Histopathological Changes	Study Group			Control Group		
	No. of cases	Perinatal mortality	Percentage	No. of cases	Perinatal mortality	Percentage
Deposition of fibrin	2	—	—	2	—	—
Intervillous thrombosis	2	1	50%	—	—	—
Intervillous fibrin deposition	4	—	—	—	—	—
Ischaemic necrosis	3	3	100%	—	—	—
Calcification	6	6	100%	—	—	—
Endarteritis	4	4	100%	—	—	—
No change	49			48		
Total	70			50		

changes in placenta were observed in comparison to 50 cases where the membranes ruptured late in labour.

No gross abnormality in the placenta and adenexa could be observed in the cases of premature rupture of membranes in comparison to the control cases. However, in cases with evidence of intrauterine infection, the membranes were dull, ragged, shaggy and dirty in appearance.

In 40 per cent of the cases, the cord was attached eccentrically in comparison to 28 per cent control cases. This finding, therefore, does not appear to have much importance.

Decidual inflammation was present in 60 per cent of the study cases as compared to 30 per cent control cases, inflammation of the foetal membranes in 40 per cent as compared to 20 per cent in controls, umbilical cord inflammation in 25.8 per cent as compared to 10 per cent in controls, and placental inflammatory response in 40 per cent as compared to 20 per cent in the controls.

It is obvious that an inflammatory response is observed in placentae and adenexae even in those cases where the membranes do not rupture prematurely

and there is no evidence suggestive of genital infections. These findings are in concurrence with Maudsley *et al* (1966) and Malkani and Bhasin (1971).

There was a close relationship between the placental changes and the perinatal morbidity and mortality. With ischaemic necrosis, calcification and endarteritis, the perinatal mortality was 100 per cent while with intervillous thrombosis, the perinatal mortality was 50 per cent.

Degenerative changes in the placenta are of particular interest since the source of nutrition to the placenta—maternal or foetal—is doubtful. Young (1928) is of the opinion that the placental nutrition is through the maternal circulation. In this case a generalised malnutrition in the mother could explain both the degenerative changes in the placenta as well as rupture of an inherently weak membrane.

Summary and Conclusions

1. Definite histopathological changes were observed in the placentae and its appendages in cases of premature rupture of membranes in the form of fibrin deposition, intervillous thrombosis,

ischaemic necrosis, diffuse calcification and endarteritis.

2. Definite inflammatory changes could be observed in the cord, membranes, decidua and placenta in both the groups.

3. In cases showing marked histological changes in the form of ischaemic necrosis, calcification and endarteritis, the perinatal mortality was 100 per cent; with intervillous thrombosis it was 50 per cent while in those cases exhibiting fibrin deposition and intervillous fibrin deposition, the perinatal mortality was nil.

4. It appears that a generalised maternal malnutrition syndrome is operative in causing premature rupture of membranes, placental dégeneration and perinatal morbidity and mortality.

References

1. Malkani, P. K. and Bhasin, K.: *J. Obst. & Gynec. India.* 20: 340, 1971.
2. Maudsley, R. J., Brix, G. A., Hinton, M. A., Robertson, E. G., Bryans, A. M. and Haust, M. D.: *Amer. J. Obst. & Gynec.* 95: 648, 1966.
3. Young, J.: *J. Obst. & Gynec. Brit. Emp.* 26: 1, 1928.

See Figs. on Art Paper III-IV



Fig. 1
Equipment used for Cryosurgery.

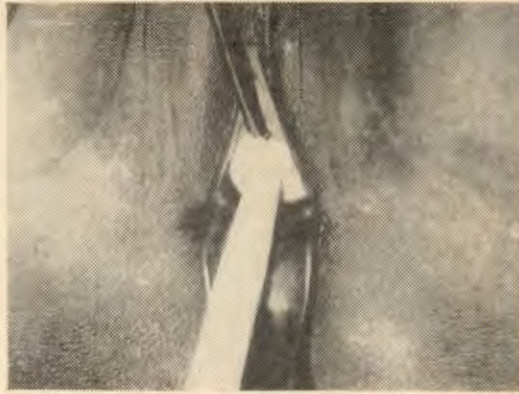


Fig. 2
Ice-ball formed during freezing.

*Vesicular Mole Associated with Viable Foetus—
Kachroo and Khanum pp. 605-606*

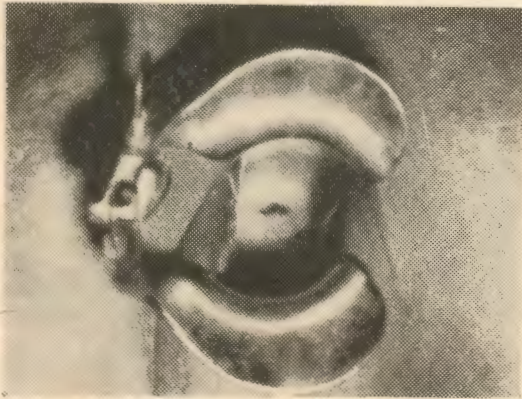


Fig. 3
Healed Cervix after treatment.

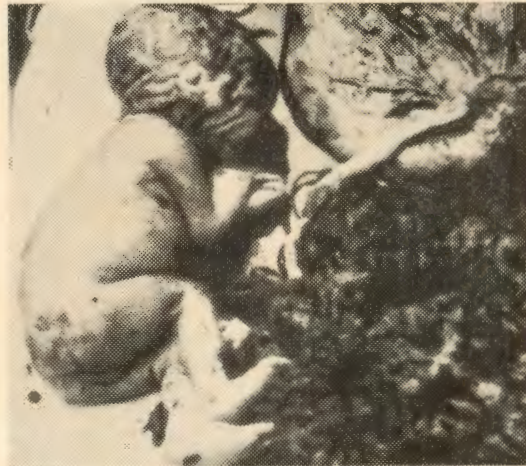


Fig. 1
Viable foetus placenta and vesicular mole.

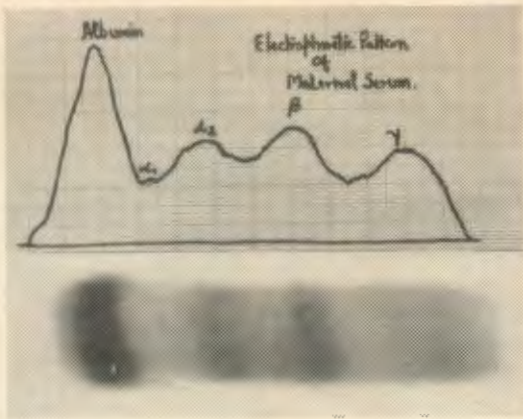


Fig. 1
Immunoelectrophoresis pattern of maternal serum proteins.

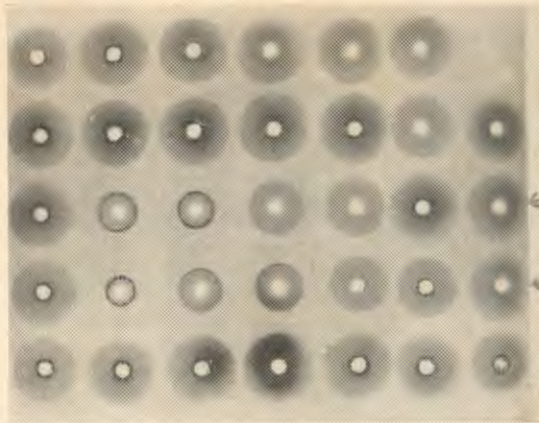


Fig. 2
Single radial immuno-diffusion in agar gel stained with amido black. Arrows indicate the results obtained with graded dilution of the W.H.O. standard.

A Study on the Antigenicity of the Amniotic Fluid— Mukerjee pp. 532-533

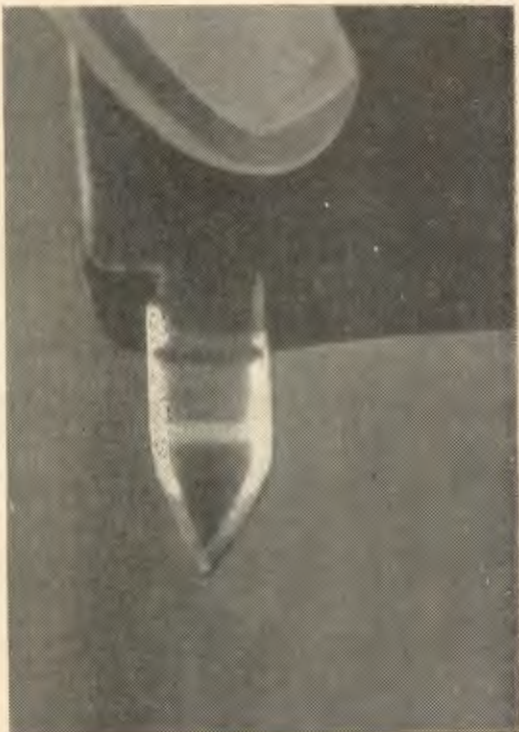


Fig. 1
Interfacial precipitation ring between the liquor amnii and anti-amniotic serum.

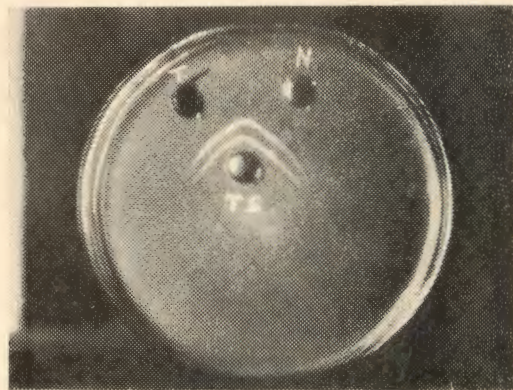


Fig. 2
Gel diffusion between anti-amniotic rabbits serum from a case of eclampsia (TS) and the amniotic fluid from normal pregnancy (N) and antepartum eclampsia (T). Exactly similar result was obtained when anti-amniotic rabbit's serum from cases of normal pregnancy was used (not shown).

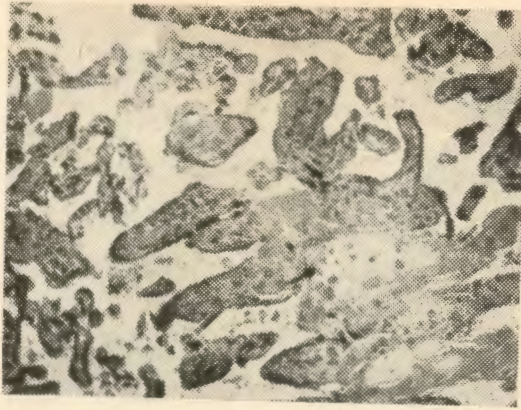


Fig. 1: H. & E. x 80.
Section of the placenta from a case of premature rupture of membranes, showing intravillous deposition of fibrin (↑) and focal areas of calcification (↑↑).

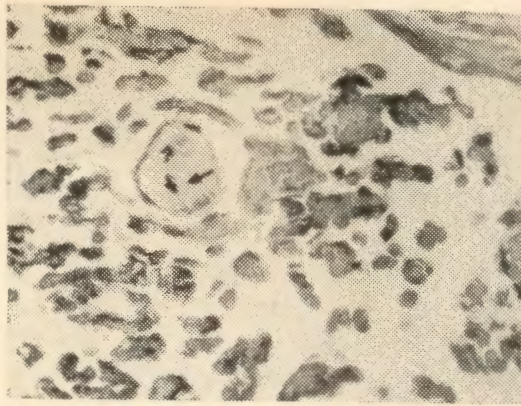


Fig. 2: H. & E. x 80.
Section of the placenta showing intervillous fibrin deposition along with thickening of the vessel wall (↑).

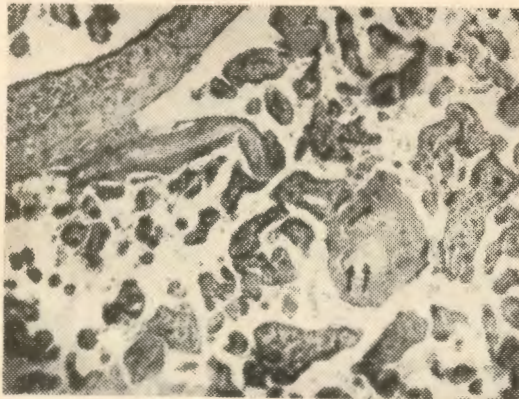


Fig. 3: H. & E. x 80.
Villi showing ischaemic necrosis (↑) and intervillous fibrin deposition (↑↑).

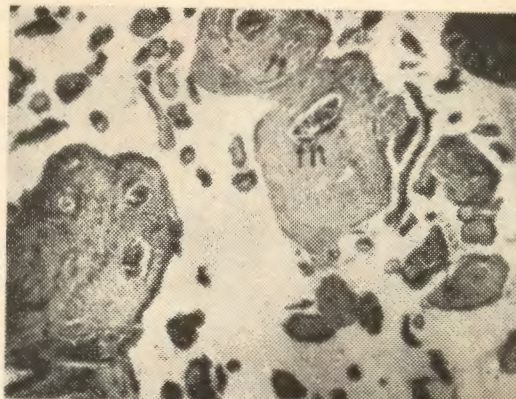


Fig. 4: H. & E. x 80.
Section of the placenta from a case of premature rupture of membranes, showing, extravillous fibrin deposition (↑) endarteritis (↑↑↑) of lumen of the lumen (↑↑).

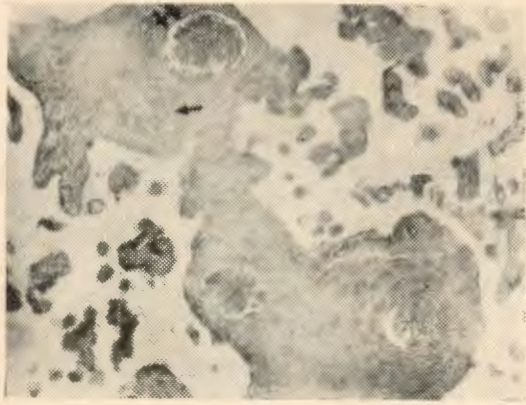


Fig. 5: H. & E. x 80.
Villi showing extensive thickening of the vessel wall (↑↑) with completely obliterated lumen (↑).



Fig. 6
Section of the placenta from a case of premature rupture of membranes, showing extensive areas of calcifications. (↑). H. & E. x 80.

Primary Malignant Melanoma—Kishore et al pp. 613-615

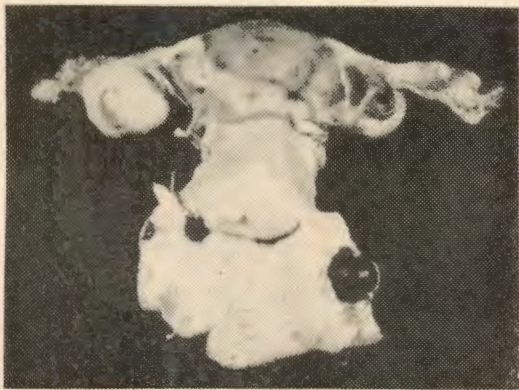


Fig. 1
Photograph of specimen removed after penhysterectomy with total colpectomy. Three bluish nodules are seen in the vaginal flap.

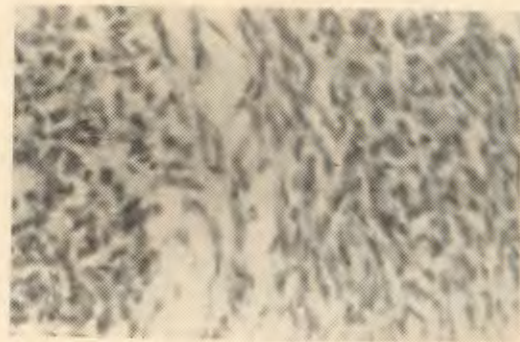


Fig. 2: H & E x 480.
Section of one nodule showing groups of round oval and spindle cells with large vesicular nuclei, normal and abnormal mitotic figures and fine pigment granules in cytoplasm.



Fig. 1
Grape like sarcoma from vagina and cervix with bicornuate uterus (TAH with Bilsalpingo-oophrectomy and in situ specimen).

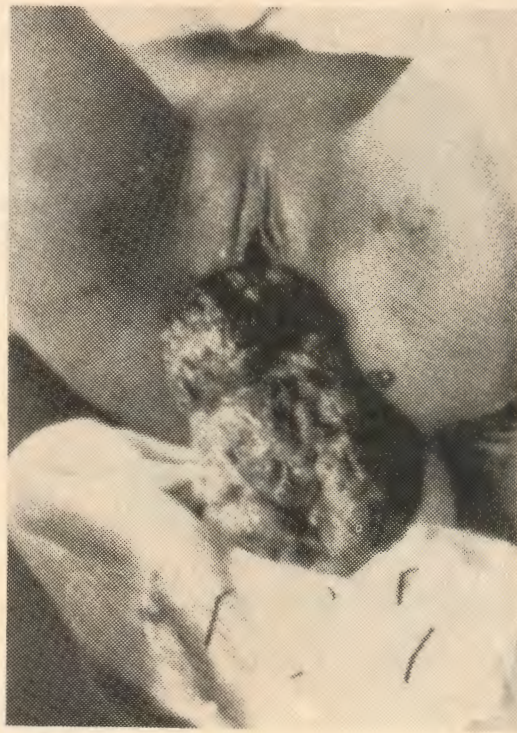


Fig. 2
Grape like sarcoma with the tumor arising from vagina (specimen in situ with the patient aged 20 years).



Fig. 3
Pure stromal sarcoma (Homologous type)
H & E Stain, X 100.

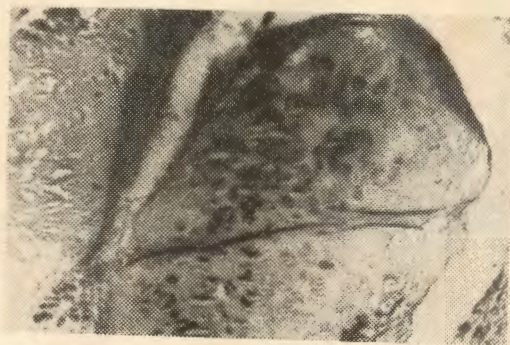


Fig. 4
Heterologous elements showing cartilage.



Fig. 5
Heterologous element rhabdomyo-blasts.

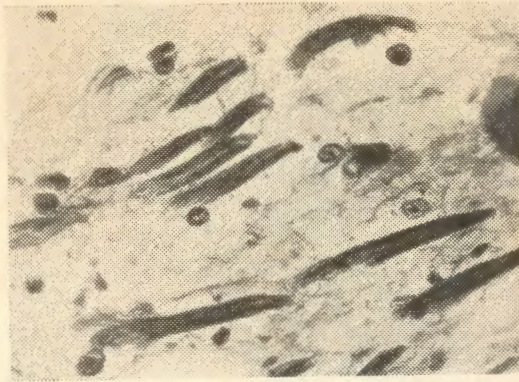


Fig. 6
Heterologous elements showing mature striated muscle elements. PTAH Stain x 400.

Pyoderma Gangrenosum Following Hysterectomy—Khurana and Lewis pp. 613-617



Fig. 1
Ulceration and gangrenous necrosis of the whole anterior abdominal wall, as far deep as the rectus sheath. (14th post operative day).



Fig. 2
Response to corticosteroid therapy. Healthy granulation tissue replaced the slough. (20th post operative day).



Fig. 1 (Case 2)
Showing the angular pregnancy in the right
cornu of the uterus.



Fig. 1
Photograph showing tuberculous granular
lesions of upper 2/3rd of the anterior and pos-
terior vaginal walls.

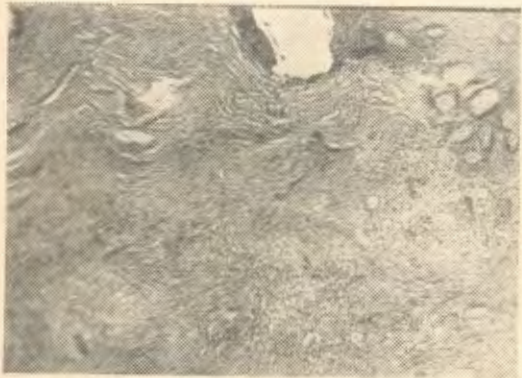


Fig. 2
Microphotograph showing vaginal tuberculosis.



Fig. 3
Photograph of vagina taken after 6 weeks of
anti-tuberculous treatment. Tuberculous lesion
is well healed. Upper part of the vagina is ste-
nosed and cervix is flushed with the vaginal
vault.

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