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Original Article

STUDY OF TROPHOBLASTS AND HISTOLOGICAL CHANGES OF FALLOPIAN TUBE IN TUBAL PREGNANCY AND ANATOMICAL CONSIDERATIONS FOR ITS EARLY RUPTURE

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ABSTRACT

Fertilisation takes place in the lumen of fallopian tube, from where, aided by the ciliated columnar epithelium of the tube, the fertilised ovum makes its way into uterine cavity and the implantation occurs. The passage of fertilised ovum into uterine cavity is delayed or obstructed by developmental, mechanical or other defects which lead to tubal gestation. The incidence varies from 1 in 300 to 1 in 150 pregnancies and it contributes significantly to the maternal mortality and morbidity. Early diagnosis and therapy has helped to reduce the maternal death due to ectopic pregnancy. However, study on histological changes of early ectopic pregnancy are rather scarce and therefore, the present study was conducted on 25 patients of ectopic pregnancy specimens observed for the mode and extent of invasion of chorionic villi, and the histological changes in wall of the fallopian tubes to evaluate the causes of early tubal rupture as well as estimation of gestational age by the study of chorionic villi.

KEYWORDS: Fallopian tube, Chorionic villi, Ectopic pregnancy, Tubal rupture, Nidation.

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INTRODUCTION

Fallopian tubes lie on each side of the uterus, each measuring about 10cm in length. The tube consists of four parts. From lateral to medial side, these are infundibulum, ampulla, isthmus and intramural parts. The tube has an internal mucosa, an intermediate muscular stratum and an external serosa.

Fallopian tube is lined by simple columnar ciliated and secretory epithelium with relatively large nucleus (Fig 1A). The muscular layer composed of external thinner longitudinal and internal thicker circular layers of smooth muscles, additional internal longitudinal fibres appear in some parts of tube like isthmus. In the ampulla this layer is absent. The serosa is peritoneum with subjacent connective tissue [1].

The chrionic villi possess double covering, an outer layer of syncytium resting on a single inner layer of cytotrophoblast, until about the fourth month.

Ectopic pregnancies still contribute significantly to the cause of maternal mortality and morbidity. While there has been about four fold increase incidence over the couple of decades, but the mortality has been slashed down to about 80%. The incidence of tubal pregnancy has increased. The reasons are increased prevalence of chronic pelvic inflammatory disease, tubal plastic operations, ovulation induction and IUD use. The incidence varies from 1 in 300 to 1 in 150 pregnancies. Because of unfavourable environment, early interruption of pregnancy is inevitable within 6-8wks. Tubal pregnancy may terminate by tubal abortion or tubal rupture [2].

The fallopian tube and uterus both are developed from paramesonephric duct, but the tube lacks the property of hyperplasia and hypertrophy as like in uterus during pregnancy, still there are many other anatomical factors which is deficient in tube responsible for its early rupture which are discussed here.

MATERIALS AND METHODS

A cross sectional study done on haematoxylin and eosin stained 25 slides of tubal pregnancy around 6wks gestational age in the Department of Anatomy, obtained from the department of pathology, Bangalore medical college and research institute, by using compound microscope under 100X magnification.

RESULTS

In this study among 25 tubal pregnancy slides, 14 were ruptured tubes and remaining 11 were unruptured. The histological features are describing according to the layers of the fallopian tube.

Lumen: Consists of product of conceptus in all unruptured tubes, in 9 ruptured tubes whereas in remaining 5 tubes the product of conceptus was extraluminal. The lumen also consists of blood clots and chorionic villi in all ruptured tubes.

Epithelium: The epithelial cells are low columnar with large, darkly stained nucleus, and cytoplasm was eosinophilic, the cilia was not appreciable. The subepithelial connective tissue (Fig. 1B & 1C) cells showed minimal decidual reaction(Fig 1D), these cells were swollen, lightly basophilic with darkly stained large nucleus, which shows their high secretory activity due to influence of progesterone. The lamina propria consists of few lymphocytes and few engorged blood vessels; also it was invaded by the chorionic villi in all ruptured tubes.

Myosalphinx: Consists of prominent elongated smooth muscle cells with elongated darkly stained nucleus. The thinning of myosalphinx (Fig. 1F) was seen in 12 cases of ruptured tubes. Intramural haemorrhagic spots were present in all of ruptured tubes. The myosalphinx was invaded by chorionic villi in 13 cases of ruptured tubes and in 5 cases of unruptured tubes. In very few cases the normal architecture of myosalphinx was seen. The thinning of serosal layer with flattened mesothelium and engorged blood vessels (Fig. 1E) were present. In ruptured

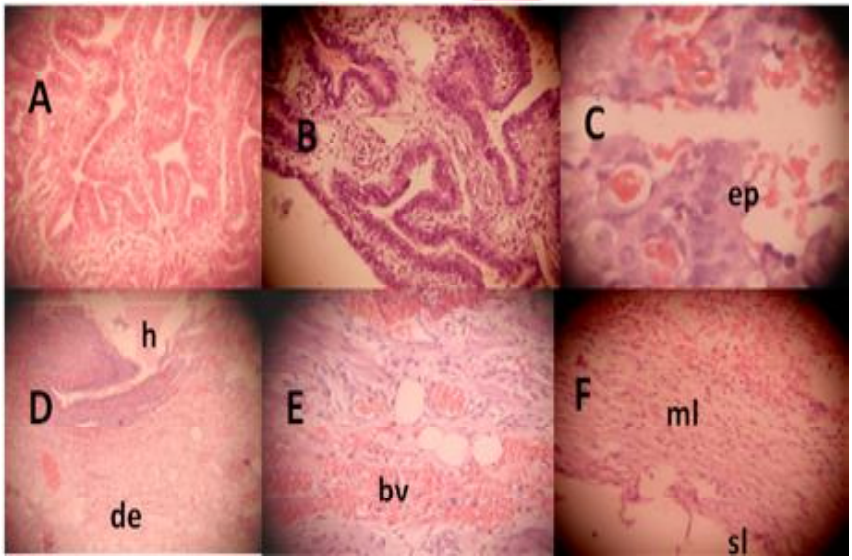

Fig. 1: Photomicrographs of
A. Human fallopian tube of normal architecture with mucosal folds
B. Epithelial and subepithelial cells of tubal pregnancy
C. Epithelial changes (ep) under higher magnification (100X)
D. Haemorrhagic spots(h) and decidual changes(de)
E. Engorged blood vessels(bv) in muscular layer
F. Thinned muscular(ml) and serosal layers(sl).

tubes very much thinning of serosal and subserosal layers (Fig. 1F) were seen.

In all ruptured tubes extraluminal haemorrhagic spots and chorionic villi were present. The chorionic villi (Fig. 1G) were found more in number; these were extensive in invasion, invaded till the serosa. The chorionic villi consist of inner layer of cytotrophoblast which were regularly arranged cuboidal in appearance, and outer irregular flattened syncytiotrophoblast (Fig 1H). The aggregations of syncytiotrophoblasts were observed in the lumen of tube. Haemorrhagic spots were present in all the ruptured tubes where as only in luminal aspect of myosalphix in unruptured tubes.

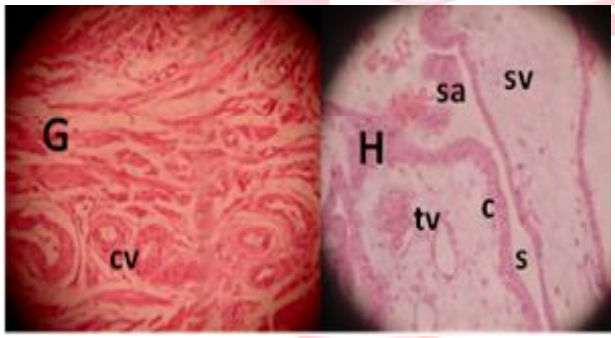

Fig. 2: Photomicrograph showing crossection chorionic villi (cv) in muscular layer (G), secondary (sv) and tertiary villi (tv) with inner cytotrophoblast (c) and outer syncytiotrophoblast(s) with syncytiotrophoblastic aggregation (sa) in (H).

DISCUSSION

The present study provides the valuable information about the mode and extent of invasion of chorionic villi in the tubal wall, the microscopic cause for tubal rupture and estimation of gestationl age by the appearance of tertiary chorionic villi. Tubal pregnancy induced changes in the histology of fallopian tube have not been extensively studied. This is an effort to study the histological changes of fallopian tube in tubal pregnancy and evaluation of causes for its early rupture.

Luminal dilatation was caused by the presence of the gestational sac with or without embryo, by varying amount of trophblastic tissue but primarily by maternal blood causing destruction and disruption of fallopian tube [3].

Implantation is intercolumnar fashion that is in between two mucosal folds and also intramural. Decidual change at the site of implantation is minimal. The muscles undergo limited hyperplasia and hypertrophy but more stretching. A pseudo capsularis is formed consisting of fibrin, lining epithelium and few muscle fibres. Blood vessels are eroded by the chorionic villi. The walls thinned out due to distension by the growing ovum, accumulation of blood and erosion by the chorionic villi. The tertiary chorionic villi appear at 21st post ovulatory days [2].

Beneath the mucosa is a zone of rich blood vessels especially venous sinuses and lymphatics, intermingled with smooth muscle fibres, this appears to have mechanical functions similar to cavernous erectile tissue which stiffen the tube in midcycle. There is also evidence that the isthmus, especially in its lateral region, acts as sphincter by the contraction of internal longitudinal fibres, which can able to delay the progress of the fertilized, segmenting ovum so that it reaches to uterus for implantation (1).

The fertilized ovum burrows itself into the wall of the tube on account of the eroding and penetrating properties of the chorionic epithelium. There is no real decidual reaction in the stroma of tubal mucosa. There is however, increased congestion and softening of the tissues. The ovum, after burrowing rapidly into the softened and highly vascularised tissues, is encapsulated within the muscular tissue of the tubal wall. As the ovum grows, the muscular tissue, attempts to hypertrophy. But there is only hyperplasia of the muscle cells and no hypertrophy and so the tube is not able to accommodate the growing ovum. The thinning of the fallopian tube on account of the trophoblastic infiltration, uncontrolled by decidual cells, and the mechanical distension of the lumen of the tube by the growth of the ovum, results in early rupture of the tube [4].

At higher magnification of standard slide, the muscles of the fallopian tube are vascular and edematous. The tube is lined with deciduas and decidual tissue lies free within the lumen. Adherent to the latter area sheet of cytotrophoblast and a chorionic villus with its pale staining core of myxoid tissue covered with a thin layer of syncytiotrophoblast and cytotrophoblast. No fetal elements were found among the contents of tube [5].

Trophoblastic spread was predominantly intraluminal in 67% of cases. Intratubal haemorrhage, generally in parallel to trophoblastic spread, often led to marked tubal destruction [6]. Ectopic tubal pregnancies may grow either intratubally or extratubally by villous invasion into the wall and blood vessels [7].

Correlation between the gestational age and the maximal diameter of the respected fallopian tube, the average diameter of chorionic villi, the number of chorionic villi per square millimetre of the microscopic section, the number of degenerated villi per square millimetre, the number of syncytial knots per square millimetre, and the average number of layers of cytotrophoblastic cells covering the villi [8].The growing tubal gestation does not have any unique characteristics as far as the maternal-foetal tissue interface is concerned. The placentation is relatively superficial, and the growth is intraluminal. Tubal rupture occurs as a result of progressive tubal distention with focal hemorrhagic necrosis [9].

When luminal distension was marked the myosalphinx became stressed and compressed. Trophblastic invasion into the myosalphinx ranged from localized penetration by anchoring villi to complete destruction of muscularis. In majority of cases trophoblastic invasion was limited to luminal aspect of the myosalphinx along with inflammatory cell infiltration. The serosa and subserosa overlying the site of ectopic pregnancy was usually thinned out and became only single layer of mesothelial. In cases of rupture the serosa was breached by the invading trophoblast which eroded through the tubal tissue. In many cases, the invading trophoblast invaded subserosal blood vessel [3].

In 52.2% of ampullary pregnancies, the gestations were found intraluminally, the muscularis was preserved. This study suggests that in isthmic pregnancy, the trophoblast penetrates the tubal wall relatively early [10]. The tube was histologically normal in 17.7% of cases (61 patients). The rate of histologically normal tubes was significantly greater in patients with no past history of tubal problems (28.9%, 22 patients vs. 14.5%, 39 patients [11].

CONCLUSION

Early resumption of the trophoblastic activity is probably due to premature degeneration of the zona pellucida which facilitates nidation in the tube. The extensive trophoblastic invasion may be due to some anatomical consideration of the tube where the intramuscular implantation is usual. And other anatomical factors like minimal decidual changes and trophoblastic invasion towards muscular and serosal layers due to rich blood supply where more number of large engorged blood vessels were present. Because of the trophoblastic invasion the walls of the tube undergo haemorrhagic necrosis. There is only hyperplasia of the muscle cells and no hypertrophy and so the tube is not able to accommodate the growing ovum. Due to luminal distension there was thinning of myosalphinx and serosal layers. The thinning of the fallopian tube on account of the trophoblastic infiltration, uncontrolled decidual cells, and the mechanical distension of the lumen of the tube by the growth of the ovum, results in early rupture of the tube. The appearance of tertiary chorionic villi and the number of layers of chorionic villi give the gestational age. In cases where the penetration of villi occur outside the tube into the walls of tube or into the vessels, for these cases only surgical evacuation of products of conception is not sufficient, these may need salphingectomy. It is concluded that because of anatomical limitations of tube, it is more prone to rupture early in tubal pregnancies, so early diagnosis and resection of affected tube is essential before rupture.

Conflicts of Interests: **None**

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