

MICROSCOPIC ANATOMY OF HUMAN FETAL PANCREATIC ISLETS

Dr. Latha sreedhar.L.S.*¹, Dr. Suja Robert.S.², Dr. Aswathy Maria Oommen³ & Dr. Aleyamma Fenn. T.K.⁴

*^{1,2,3}Associate Professor of Anatomy GMC, Trivandrum

⁴Prof. of HOD, Anatomy Govt. Medical College Palakkad.

Abstract

Background and Aim

Keywords:

Human fetal pancreatic islets, beta cells, alpha cells and diabetic mothers.

The islets of Langerhans is an important endocrine gland which regulate the blood glucose level. The micro anatomic studies in human fetal pancreatic islets were significant since there were controversies regarding their origin and time of appearance. The role of maternal insulin in the development of fetal pancreatic islet cells and their significance in the fetal period is clinically important. Pregnant mothers with diabetes seems to protect their children from diabetes by the early stimulation of beta cells. So the incidence of diabetes in children of diabetic mothers were less when compared to diabetic fathers.

Materials and Methods

Thirty five autopsy specimens were collected from the department of pathology medical college, Trivandrum, Kerala. The gestational age of the fetuses range from 10-40 weeks. The histological examination of the specimens were done in the department of anatomy, Govt. Medical College, Trivandrum.

Result

According to the present study both acini and islets were developed from the endodermal secretory tubules. Acini and islets were appeared by 12 weeks, and beta cells appeared by 14 weeks. At 28 weeks alpha cells appeared and the cytoplasm of beta cells contained granules. At 32 weeks well defined islets and the granules appeared in the cytoplasm of alpha cells.

Conclusions

The fetal pancreatic islets and acini had a common endodermal origin- the secretory tubules. The islets and acini appeared by 12 weeks, the beta cells by 14 weeks. A diabetic mother can protect their children from diabetes to some extent than a diabetic father.

INTRODUCTION

The micro anatomic studies in human fetal pancreatic islets were less. There were controversies regarding the origin and time of appearance of fetal pancreatic islets and its cells. Human pregnancy is marked by alterations in endocrine system, the most notable one being the striking increase or decrease in insulin hormone production by maternal and fetal pancreas. The role of maternal insulin in the development of fetal pancreatic islets and its cells were significant in fetal period. The insulin level in fetuses has a role in maturation of fetal organs and induction of abortions and premature labour without any other apparent cause. The microscopic study of fetal islets could prove to be useful for radiologists, neonatologists and diabetologists alike

MATERIALS AND METHODS

Thirty five fetal autopsy specimens were collected from the department of pathology medical college, Trivandrum, Kerala. The gestational age of the fetuses ranged from 10-40 weeks. (Table -1) The histological examination of the specimens were done in the department of anatomy, Govt. Medical College, Trivandrum

Inclusion Criteria

Fetuses in which autopsies were done within six hours after death.

Exclusion Criteria***Macerated fetuses***

Fetuses with congenital anomalies of abdominal viscera.

The age of the fetuses were taken from the hospital records and crown –rump length. During autopsy after opening the abdomen, found out the pinkish grey coloured gland lying transversely across the posterior abdominal wall, retroperitoneally, extending from the concavity of duodenum to the hilum of spleen.(Fig 1)

The bits were taken from the gland and fixed in Bouins fluid for 24 hours. After fixation the specimens were subjected to routine histological processing. As per the standard procedures described by Mac Manus and Moury - 1960. The paraffin blocks were serially sectioned at a thickness of five microns using a rotary microtome. After incubating for one hour the sections were stained with standard haematoxylin and eosin (H&E). For microscopic evaluation of the islet cells, special stains like Massons- trichrome, Chromealum haematoxylin, PAS etc. were used. The mounted specimens were observed under low power, high power and oil immersion- objectives of a binocular microscope. The sections were observed under oil immersion objective to study the characteristics of different types of cells and measurements were taken with a horizontal eye piece micrometer called the graticule, which is calibrated with a stage micrometer.

RESULT

The samples collected for the present study were grouped as per their gestational age (Table-1) According to the present study both acini and islets were developed from the secretory tubules (fig-6)

In the present study the lowest aged sample was of a 10 weeks old fetus which showed connective tissue and duct system only (fig-2)

In a 12 weeks old fetus, both acini and islets started to appear. (fig-3). In a 14th week old fetus, beta cells were first appeared.(fig-4) The beta cells were more basophilic and devoid of granules. In a 28 weeks old fetus, the alpha cells showed appearance and cytoplasm of beta cells showed granules. The acini were grouped into well defined lobules by the interlobular connective tissue (fig-7). At 32 weeks the islets were well defined, with a reticular capsule. A large secretory tubule surrounded by islets, acini and capillaries were observed. The alpha cells contained granules in their cytoplasm during 32 weeks.(fig-8). In full term fetus, the staining characteristic of islets cells were similar to that of adult islets. In a full term fetus 2/3 of pancreatic tissue is filled with islets (Fig-10)

In a fetus of diabetic mother at 32 weeks, more number of islets and beta cells were observed (fig-9). In a fullterm fetus, the alpha cells were more in number and larger in size compared to a normal fetus.(fig-11)

DISCUSSION

The present work provides an opportunity to understand the normal histological appearance of fetal pancreatic islets at various gestational ages. But in the present study both islets and acini had a common origin, the secretory tubules. Studies have shown that islets and acini take origin from duct system and centroacinar cells². M.E Lagusse was the first scientist who studied the developing pancreases and he described two generations of islets. At first the acini developed from ducts, which disappeared later and new generations developed from secretory tubules. Acinar cells were thought to be capable of transforming into islet cells and islet cells in to acini. The relative proportion of each being governed by functional requirements. He described it as the ‘Theory of balance’⁹. In a 10 weeks old fetus, the

pancreas microscopically contains connective tissue and duct system¹⁰. In a 12 weeks old fetus, round or oval light stained areas of islets without any cells could be observed^{1,5}. The acini were first originated and beta cells appeared shortly after the development of capillary net work^{6,8}.

At 28 weeks, the primitive islet cells were composed of cell group of various sizes surrounded by capillaries¹¹. This study also high lights that the beta cells become functionally active at 28 weeks from which period the fetus is said to be viable. The alpha cells were appeared at 28 weeks and their granules appeared by 32 weeks³. The glucose required for the growth of the fetus is obtained from the maternal blood. The level of blood glucose in fetus has to be maintained by both beta and alpha cells for the proper development of fetuses. Compare to beta cells, the alpha cells were larger with prominent mitochondria and golgi apparatus, which show their activity during the later stages of fetal period⁷.

According to Ferner-1952 not less than one third of total human pancreatic volume in a full term fetus consist of islet tissue, compared to less percentage in adults⁴. It was also observed that diabetes can be prevented and the incidence of diabetes, can be substantially reduced by neonatal stimulation of pancreatic beta cells. They stated that children of diabetic fathers, developed diabetes at an earlier age and incidence was 6.1% where as children of diabetic mothers, the incidence was only 2.1% and at a later age^{12,13}.

CONCLUSIONS

1. The fetal pancreatic islets and acini had a common endodermal origin the secretory tubules.
2. The islets and acini appeared by 12 weeks, the beta cells by 14 weeks, and their granules appeared by 28 weeks. The alpha cells appeared by 28 weeks and their granules by 32 weeks.
3. A diabetic mother can protect their children from diabetes, than a diabetic father to some extend by the neonatal stimulation of pancreatic beta cells.

In recent days, pancreatic imaging has improved enormously with the introduction of USG, CT, endoscopic retrograde cholangio pancreatography and selective angiography. MRI and positron emission tomography (PET) provide a further refinement in the morphological study of pancreas. Therefore it has become imperative to carry out morphometric and microscopic study of pancreas on living subjects. This is only a preliminary study as this is done on autopsy specimens. This study can serve as a base for future research pertaining to the role of diabetic mothers in fetal pancreatic islets.

ACKNOWLEDGEMENTS

I thankfully acknowledge the guidance and valuable suggestions of Dr.V.M.Kushid former professor and HOD, Department of Anatomy, GMC, Trivandrum and Dr.K.R. Chandramohan Nair Senior Scientific Officer Dept. of Anatomy GMC, Trivandrum.

REFERENCES

1. Bayley.J.M -1937 staining methods for the islets of Langerhans. *J-patho and bact.* 44 (272-276)
2. Bencosome, S.A-1995. *The histogenesis and cytology of the pancreatic islets in the rabbit. American Journal of Anatomy-96, (103-151)*
3. Bloom W-1931- *New type of Granular Cells in islets of Langerhans of man Anat. Record-49 (363-371)*
4. Ferner-H-1952 *Cytogenesis desinsel systems beins menschen Zell forsch 35, 147-48*
5. Gomori G-1941, *Observations with differential stains on human islets of Langerhans, American Journal Patho. Vol -17 (395-406)*
6. Hard. W- *The origin and differentiation of the alpha and beta cells in the pancreatic islets of the rat. 1944. American journal of Anatomy 75, 369.*
7. Hellman B-1959, *Actual distribution of the number and volume of the islets of Langerhans in non diabetic humans of varying ages. Nature, 184, 1498-99*
8. Lacy P.E -1961, *Electron micro scope of beta cells of pancreas, American Journal , Medicine 3, 851-859.*
9. Laguesse, ME 1955 *Ilots endocrine et formers de transition dansle louble, Social. Biol, 58, 542-544*
10. Pearce RM, 1902-03, *The development of the islets of Langerhans in the human embryo. American journal of Anatomy, 2, 445-55.*

11. *Saguchi.S-1950, Cytological studies of Langerhans with special reference to the problem of their relation to the pancreatic acinous tissue, American journal of Anatomy, 28, 186-90*
12. *Vanralte DA, Bunck M- Ultra Structural study on human pancreatic islets, Euro.Journal Endocrinology - 2016,1, 77-113*
13. *Woerner C.A-1938, Studies of the islets of Langerhans after continuous intravenous injection of dextrose Anatomy Records, 71, 33-57*