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Tissue isoantigens A, B and H in primary carcinoma of the pancreas

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Abstract

AIM: To investigate the quantity of demonstrable tissue isoantigens A, B and H in primary carcinoma of the pancreas and their relationship with the degree of anaplasia.

METHODS: The pathological classification of 26 primary carcinomas of the pancreas and their anaplasia were studied by light microscopy with HE staining. The quantity of isoantigens A, B and H in carcinomas of the pancreas and their adjacent normal pancreas tissues were studied by immunohistochemical method.

RESULTS: Twenty-six primary carcinomas of the pancreas were classified as follows: Two acinar cell carcinomas, 22 adenocarcinoma, 1 adenocanthoma and 1 undifferentiated carcinoma. One of 2 acinar cell carcinomas, 6 of 22 adenocarinomas and 1 adenocanthoma were well differentiated; 1 of 2 acinar carcinomas and 12 of 22 adenocarcinoma were moderately differentiated; 4 of 22 adenocarcinoma and 1 undifferentiated carcinoma were poorly differentiated. The results of the immunohistochemical method showed that the ABH positive cells were more frequent in acinar cells and ductal epithelial cells in the adjacent normal pancreas than those in carcinoma of the pancreas. In 22 adenocarcinoma, the ABH positive cells were more frequent in well differentiated and moderately differentiated cancer cells than in poorly differentiated cells. In 11 invasive adenocarcinoma positive cells (++ or +++) were more frequent in primary foci than in metastatic foci. Twenty of 26 carcinomas of the pancreas belonged to A, B and AB blood groups. Most H isoantigens were converted to A or B or AB substances in 8 well differentiated, 7 moderately differentiated and 2 poorly differentiated carcinomas, while less transformation of H to A or B antigen was observed in 3 poorly differentiated carcinomas of the pancreas. Furthermore, it was observed that the secretion of isoantigens A, B, and H was less in carcinomas of the pancreas than in normal pancreas.

CONCLUSION: The loss of demonstrable isoantigens ABH parallels morphological anaplasia. Furthermore, we found that the functions of transformation of H substance into A and B substances as well as the secretion of ABH substances into the pancreatic duct were hampered.

Key words: Pancreatic neoplasms; ABH antigens; Immunohistochemistry

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INTRODUCTION

The isoantigens A, B and H are not only present on red blood cells of groups A, B, O and AB individuals, but also present in normal human tissues and body fluids[1-3]. The pancreas is one of the organs in which large quantities of these antigens are present in the epithelial cells of the exocrine glands and the pancreatic ducts. Szulman[1] was the first to study the histological distribution of ABH isoantigens in human tissues by immunofluresence technique. Some authors have investigated the effect of cancerous transformation on tissue isoantigens A, B and H[4-8]. This article is an extension of our previous studies on the distribution and cellular and subcellular localization of ABH isoantigens in normal human tissue cells by immunohistochemical and immunoelectro-microscopy[3,9]. The aim of this study was to investigate the relationship between the quantities of ABH antigens present in normal pancreas and anaplasia in cancerous transformation.

MATERIALS AND METHODS

Formalin-fixed, paraffin-embedded tissue sections (5 μm) were made from 26 cases of carcinoma of the pancreas. Five sections of each case were used for study. One section was stained with HE for determining the type of cancer and the degree of anaplasia. Three sections were used for immunohistochemical study using avidin-biotin complex/HRP (ABC) technique. After deparaffinization, rehydration, and treatment with H_2O_2 to block the intrinsic peroxidase activity and with bovine serum albumin, the sections were incubated with McAb > A, McAb > B and McAb > H (1:50, Dako), respectively, for 60 min at room temperature. After washing with PBS-T, sections were incubated with rabbit anti-mouse Igs/biotin (1:300, Dako) for 50 min, conjugated with ABC/HRP for 30 min, treated with H_2O_2-DAB (3,3'-dianimo-benzidine) for 3 min, counter-stained with hematoxylin, and mounted. The interal positive controls were: (1) erythrocytes and endothelial cells in vessels; and (2) the epithelial cells of the exocrine gland and the

Table 1 Type and differentiation degree of 26 cases of primary carcinoma of the pancreas

Type	Acinar cell carcinoma	Adeno-carcinoma	Undifferentiated carcinoma	Adenoa-canthoma	Total
Well differentiated	1	6		1	8
Moderately differentiated	1	12			13
Poorly differentiated		4	1		
Total	2	22	1	1	26

Table 2 The frequency of ABH positive cells in 26 normal pancreatic ducts and in ducts of 22 adenocarcinoma

Frequency of ABH positive cells	4/4	3/4	2/4	1/4	< 1/4	0	Total
Normal pancreatic ducts	10	5	4	3	2	2	26
Adenocarcinoma (cases)							
Well differentiated	4	1	1				6
Moderately differentiated	5	3	2	2			12
Poorly differentiated	1	1	2				4

Note: 4/4 indicates that the epithelial cells of the whole circumference of duct were ABH positive, and others were analyzed in this way.

pancreatic duct adjacent to the carcinoma. The internal negative controls were (1) the connective tissue; and (2) the islands of Langerhans.

RESULTS

ABO blood groups of 26 cases of carcinoma of the pancreas

The ABO blood groups of 26 cases of carcinoma of the pancreas were as follows: A, 8; B, 9; AB, 3; and O, 6.

Type and degree of anaplasia of carcinoma of the pancreas

The type of carcinoma and the degree of anaplasia are shown in Table 1.

Relation of ABH positive cells to the anaplasia of carcinoma of pancreas

Eight of 26 cases of carcinoma of the pancreas had adjacent normal pancreas in which 26 normal interlobular pancreatic ducts were found. ABH positive epithelial cells (+-+++) were demonstrated in 24 ducts. The positive epithelial cells were more frequent in them than in adenocarcinoma, and were more frequent in well and moderately differentiated adenocarcinoma than in poorly differentiated adenocarcinoma (Table 2).

Relation of ABH antigens present in primary foci to those in metastatic tumors

Twelve of 26 cases of carcinoma of the pancreas had the adjacent normal pancreas in which carcinoma invasion was found in 11 cases. It was found that the ABH positive staining was stronger in cells of primary foci (+-+++) than in the invasive tumor (Table 3).

Comparison of secretion of ABH isoantigens between normal pancreatic ducts and in ducts of adenocarcinoma

In general, the ABH antigens were located in infra-nuclear regions, supranuclear regions, brush borders, and cytoplasm as well as in cellular secretions. Secretions of ABH antigens in 26 normal pancreatic ducts and in 26 ducts randomly selected from 5 cases of well differentiated and moderately differentiated adenocarcinoma each were compared. The adenocarcinoma cells secreted less ABH antigens in their secretions than those in the normal pancreatic ducts (Table 4).

Table 3 Comparison of the staining intensity of ABH positive cells in primary foci and in the invasive carcinoma

Intensity of ABH positive staining of cells in		Cases (n = 11)
Primary foci	Invasive carcinoma	
+-+++	0	7
+-+++	0-++	3
++-+++	0-+	1

Table 4 The localization of ABH antigens in 26 normal pancreatic cells and in 26 ducts of adenocarcinoma

Localization of ABH antigens on cell	Whole cytoplasm	Infranuclear region	Supranuclear region	brush border	Secretion
Normal ducts	6	11	11	24	24
Well differentiated	10	12	15	18	8
Moderately differentiated	5	8	14	14	8

DISCUSSION

Our results revealed that the quantity of ABH antigens in the pancreas was related with the anaplasia in cancerous transformation. It was less in carcinomas of the pancreas and their ductal secretions than in normal pancreatic tissue and their secretions, less in undifferentiated adenocarcinoma than in moderately and well differentiated adenocarcinoma, less in invaded tumors than in primary foci. It is suggested that ABH antigens in the pancreas are related to the cancerous transformation of the pancreas. That is to say, in carcinoma of the pancreas partial loss of ABH antigens occurred. These results agree with Davidsohn's research[6,7]. Furthermore, we found a phenomenon of hindering A or B antigens from H antigens in carcinoma of the pancreas.

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