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LECTIN HISTOCHEMICAL STUDIES ON RENAL TUMORS

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INDEXING WORDS

nephroblastoma; renal cell carcinoma; lectin; renal development

SYNOPSIS

Histochemical characteristics on lectin binding of 16 cases of nephroblastoma and of 57 cases of renal cell carcinoma were studied on those of six embryonal and fetal kidneys in 4 to 24 gestational weeks and in six normal adult kidneys using four biotinylated lectins (PNA, LTA, SBA and UEA-1), which show segment specific binding for normal nephron. In normal nephrogenesis, Gal β 1-3 GalNAc (PNA receptor) appeared at first in developing tubuli of the metanephros at 10 gestational weeks, and remained positive in proximal tubuli of 13 gestational weeks, where the Lewis X antigen (LTA receptor) emerged. In adult kidneys, Gal β 1-3 GalNAc distributed along distal tubuli and collecting ducts and not in proximal tubuli. Type2 H antigen (UEA-1 receptor) was confined to collecting ducts. In nephroblastomas, positive reaction to these lectins were almost exclusively confined to tubular structures. Gal β 1-3 GalNAc was constantly positive

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there. Intensely positive stain of Lewis X antigen was observed in three cases of histologically well differentiated type. Lewis X antigen positive tubuli co-expressed Gal β 1-3 GalNAc. There were six type2 H antigen positive cases showing differentiation toward ureteric bud or collecting ducts. In view of the cell surface glycosylation, regular differentiation of fetal renal tubuli was also retained in nephroblastomas according to their degree of histological differentiation. In renal cell carcinoma, Gal β 1-3 GalNAc was positive in 49% (28/57) and Lewis X antigen was positive in 54% (31/57). Type2 H antigen was completely negative. In contrast to the nephroblastoma, there was no correlation between histological features and lectin binding patterns in renal cell carcinoma.

INTRODUCTION

Nephroblastoma has been known to originate from the metanephric blastema histologically.[20] It has been suggested that the nephroblastoma represents an oncogenic course of normal histogenetic process of the kidney, from histological similarities between some types of nephroblastoma and different developmental stages of embryonal kidneys.[2,3] Recent studies using immunohistochemical techniques and on embryonic kidneys as an age control may support this concept.[7,8,15]

Renal cell carcinoma has been thought to originate from proximal tubuli morphologically.[24] However, it is now disputed from immunohistochemical studies showing simultaneous demonstration of antigens specific to the lower nephron.[1,7,8]

The cell surface of human and other mammalian cells is abundunt in glycoproteins and glycolipids. Its character is known to be changeable visibly during embryogenesis and even after birth. In this decade, various lectins which can recognize specifically a certain oligosaccharide had been used in order to clarify the cell surface structure of renal cell carcinoma or nephroblastoma histochemically. The presence of lower nephron specific sugar residue has been demonstrated in addition to the proximal tubule specific LTA reacting fucosyl residue in renal cell carcinoma[1,4,21,22,25] or nephroblastoma.[10,11,26] However, little is known about their relationship between the sequential change during nephrogenesis and its diagnostic significance of various renal

tumors.

We examined characteristics of nephroblastoma and renal cell carcinoma using lectins specific to a certain segment of nephron and compared it to those of histogenetic alteration in order to clarify the relationship between their histological differentiation and their morphological backgrounds.

MATERIALS AND METHODS

Materials

Sixteen cases of nephroblastoma and 57 cases of renal cell carcinoma obtained from surgical operation were used in this study. Embryonal and fetal kidneys of 4, 9, 10, 13, 19, and 24 gestational weeks from artificial abortion or autopsy were examined as developing control. Six normal renal tissues from autopsied cases were used as adult normal control. Examined tissues were routinely fixed by 10% formalin solution and embedded in paraffin. Histological sections were cut at four microns from these blocks and stained routinely and by lectin histochemical procedures.

Reagents

All biotin-labelled lectins were purchased from E-Y laboratory, but Avidin-biotin complex (ABC) was from Vector laboratory. The precise property of each lectin is listed in Table 1.

Table 1 Lectins used in this study.

Lectins	Abbreviation	Specificity	Inhibitory sugar
Arachis hypogaea(peanut)	PNA	D-Gal β1→3 GalNAc	Lactose
Lotus tetragonolobus	LTA	Galβ1→4 (Fucα1-3) GlcNac (LewisX)	L-Fucose
Ulex europaeus-I	UEA-I	Fucα1→2 Galβ1→4 GlcNac (Type2H)	L-Fucose
Glycine max(soy bean)	SBA	α-D-GalNAc) β-D-GalNac》 α-D-Gal	D-Gal

Abbreviation: GalNAc = N-acetyl galactosamine; Gal = Galactose

Staining procedure

The paraffin sections were deparaffinized with xylene and rehydrated through graded alcohols in routine processing. Prior to lectin staining histological sections were digested with 0.05%

trypsin added 0.01% calcium dichloride in pH 7.2 buffered Tris solution for 20-30 minutes at 37C. Then they were treated with 0.3% methanolic hydrogen peroxide for 30 minutes in order to block the endogenous peroxidase activity. After washing in buffered phosphate saline (PBS), they were incubated with biotin labelled lectins for 30 minutes at room temperature in a moist chamber. Lectins were diluted with PBS at the concentration of 0.01 mg/ml for peanut agglutinin (PNA) and soy bean agglutinin (SBA), 0.05 mg/ml for lotus tatragonolubus agglutinin (LTA) and ulex europaeus-I agglutinin (UEA-I), respectively. Histological sections were incubated with ABC for 60 minutes in a same way, after rinsing in PBS. They were washed with PBS again and again. The final reaction was performed by adding 2% diaminobenzidine 0.1% H_2O_2 solution during 5 minutes. These sections were washed with tap-water, and then counter staining was done by hematoxyline or methyl green stain.

Control staining

Negative control staining was carried out by two different procedures. Histological sections were incubated at first with PBS devoid of lectin. Another method was used by preincubated lectins with 0.1M of haptenic carbohydrates shown in Table 1. Histological sections were stained in a same way as the former procedure.

RESULTS

1. Lectin binding sites of embryonal and fetal kidneys

Staining reaction in embryonal and fetal kidneys coorresponding to the gestational weeks is summarized in Table 2. Blastemal cells and mesenchymal stroma were negative for all lectins in both mesonephros and metanephros. Lectin staining of tubuli in the mesonephros was quite uniform in an embryo and fetuses examined. PNA stained the luminal surface of mesonephric tubuli but not in others. The fetal metanephros of the 9 gestational weeks showed no positive reactions, neither in uretric buds nor in blastemal aggregations forming frequently spheroid or vesicular structures (Fig. 1a and 1b). In 10 gestational weeks, fetal metanephros positive reaction to the PNA appeared at first along the luminal surface of developing tubuli, which was identified by its close

Table 2 Lectin binding pattern of adult and intra-uterine developing kidneys.

	PNA	LTA	UEA-1	SBA
Adult kidney				
Glomerular capillary tuft	-	-	+ *	-
Proximal tubule	-	+	-	+
Henle's loop	+	-	-	+
Distal tubule	+	-	-	+
Collecting duct	+	-	+	+
Embryonal and fetal kidneys				
Mesonephros of 4 and 9 gestational weeks				
Glomerular capillary tuft	-	-	-	-
Mesonephric tubule	+	-	-	+
Metanephros of 9 gestational weeks				
Metanephric blastema	-	-	-	-
Ureteric bud	+	-	+	-
Developing tubuli from the S body	+	-	+	-
Fetal kidney of 13 gestational weeks				
Glomerular capillary tuft	-	-	+*	-
Proximal tubule	+	+	-	+
Distal tubule	+	-	+	+
Collecting duct	+	-	+	+
Fetal kidney of 19 gestational weeks				
Glomerular capillary tuft	-	-	+	-
Proximal tubule	+	+	-	+
Distal tubule	+	-	-	+
Collecting duct	+	-	+	+

Staining degree shown as : -; negative, +; positive
* UEA-1 positive in the glomerular endothelium

proximity to the S-body and tall columnar appearance of the tubular epithelium (Fig. 1c and 1d). The surface of podocytes and Bowman's capsules developing from upper pole of S-shaped bodies was positively stained by PNA. The inner surface of ureteric buds, defined by its branching configuration, was also positive for PNA. Although UEA-1 was positive in the luminal surface of the ureteric buds, it was very weak and scarcely recognizable. LTA and SBA were negative in this stage of the metanephros. The luminal surface of S-shaped bodies was totally negative for lectins except developing glomeruli above mentioned. S-bodies in the subcapsular area of the later stage showed no affinity to these lectins. In the kidney of 13 gestational weeks, proximal and distal tubuli became distin-

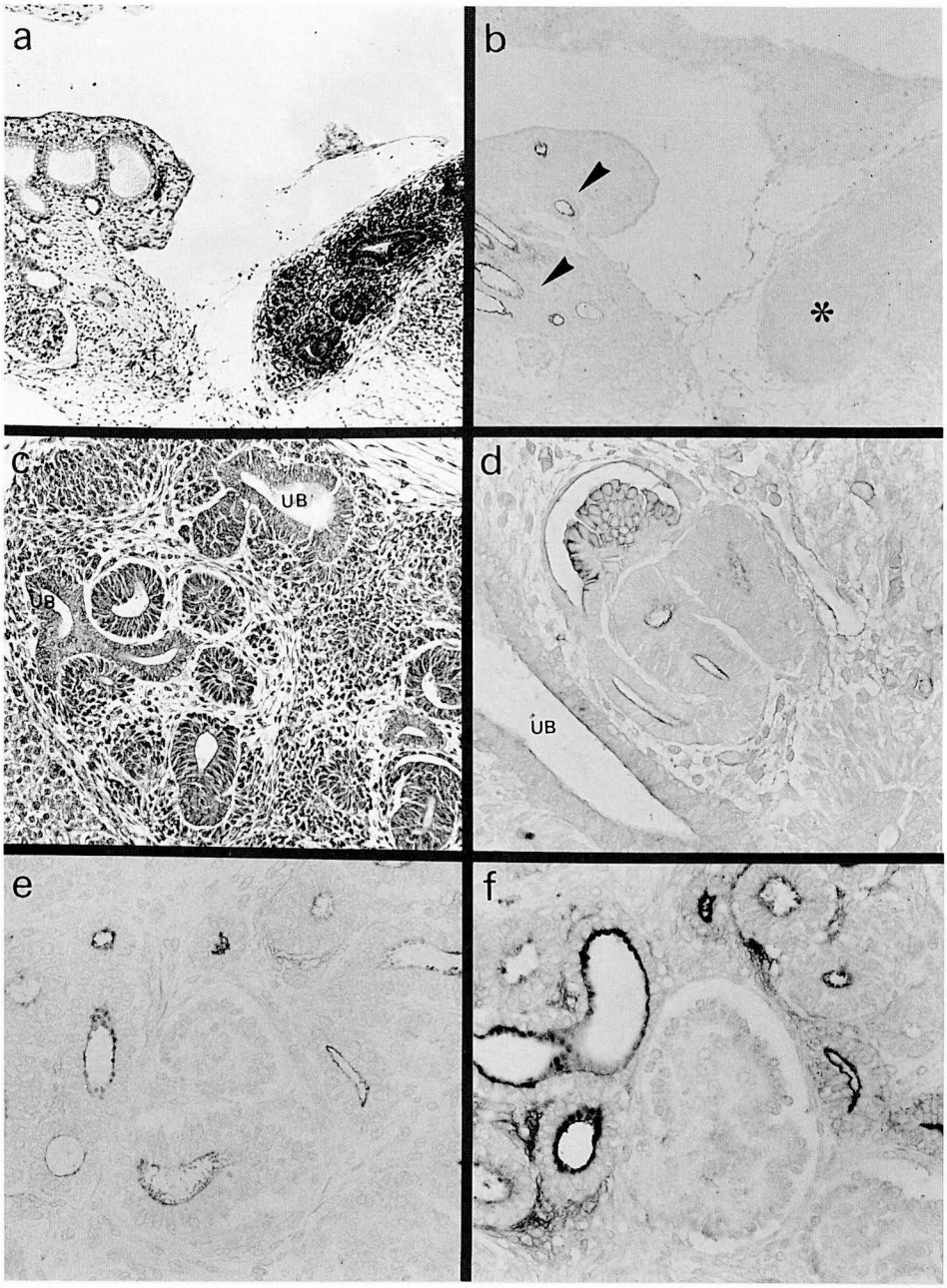

Fig. 1

guishable morphologically. LTA and SBA became positive at the brush border of the proximal tubuli, and PNA remained also positive in the same portion (Fig. 1e and 1f). The luminal surface of distal tubuli was stained intensely by PNA and SBA. PNA and UEA-1 stained positively collecting ducts as well as ureteric buds in the subcapsular area. Fine and intense positively of the vascular endothelium appeared by UEA-1 in this stage of the kidney. In 19 and 24 gestational weeks, a similar pattern of the lectin binding to a normal adult kidney was observed except in the subcapsular nephrogenic zone.

2. Lectin binding sites in the normal kidney

Staining reaction of normal kidney is summarized in Table 2. LTA stained almost exclusively the brush border of proximal tubuli (Fig. 2a). UEA-1 bound specifically collecting ducts and the vascular endothelium including the glomerular capillary loop (Fig. 2d). PNA stained the luminal surface of collecting ducts and distal tubuli including Henle's loop (Fig. 2b). SBA was positive in the luminal surface of whole tubuli except glomeruli (Fig. 2c). The transitional epithelium of renal calyces was negative for these lectins. Negative control stain was carried out by two different procedures, but both negative.

Figure 1 Binding sites of lectins in the fetal kidney.

a. The mesonephros with tubuli and glomeruli on the left side, metanephros showing early differentiation is seen in the right side. (H.E. x 66)

b. PNA stain in the fetus of 9 gestational weeks. Some renal tubuli are positive in the mesonephros (arrow head), but the metanephros is completely negative(*) for PNA. (x 66)

c. The metanephros in the fetus of 10 gestational weeks. Immature tubuli and glomeruli begin to differentiate from the S-body. UB: ureteric bud. (x 100)

d. PNA stain of the metanephros in the fetus of 10 gestational weeks. The luminal surface of developing tubuli, immature glomeruli and podocytes are positive. The luminal surface of ureteric bud (UB) is also postive for PNA. (x 200)

e. LTA stain of the kidney in the fetus of 13 gestational weeks. (x 200)

f. PNA stain of the kidney in 13 gestational weeks. Note many tubuli to be positive both for LTA and PNA. (x 200)

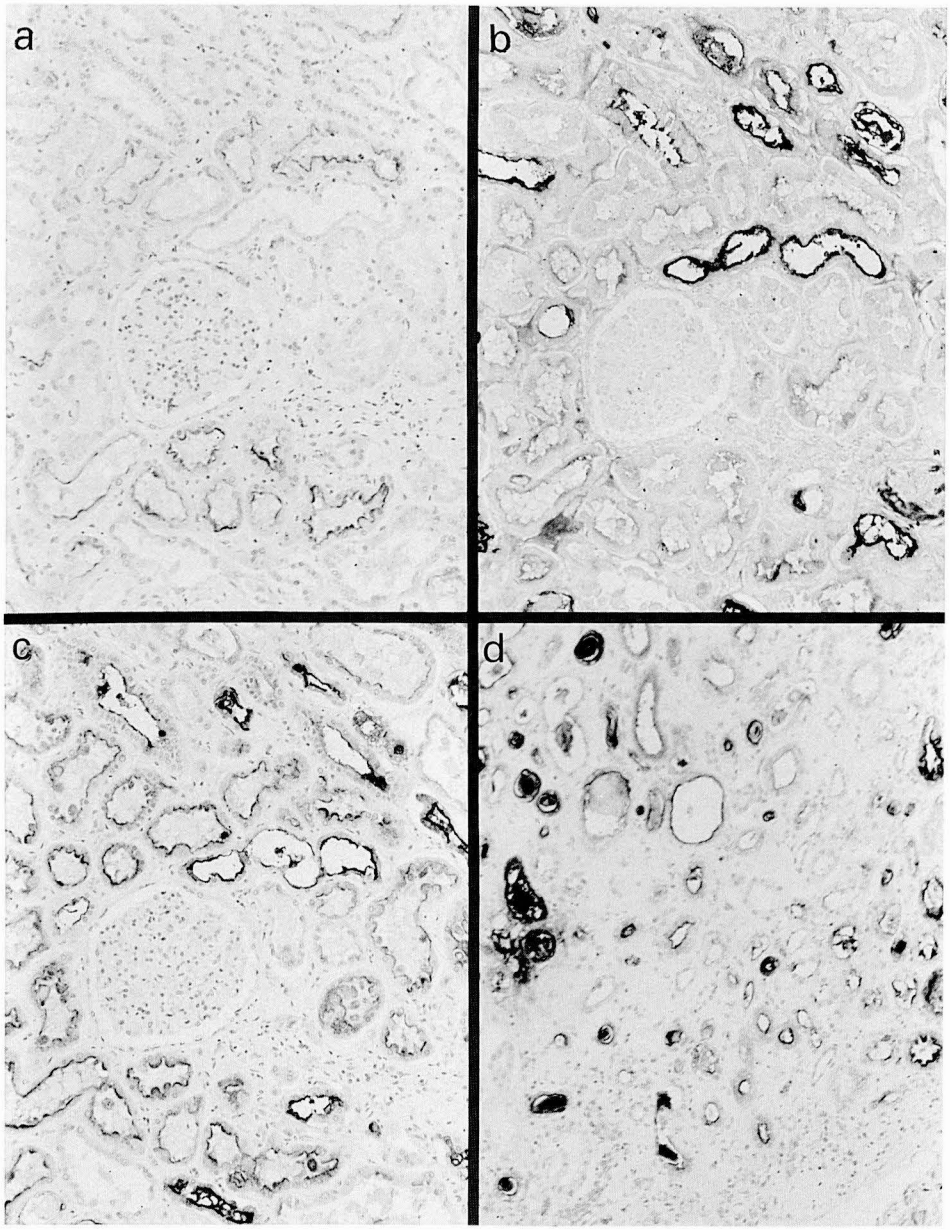

Fig. 2

3. Nephroblastoma

Histopathology

Pathological classification of nephroblastoma cases was done in this study according to the classification of Japanese Pathological Society.[23] Numbers and diagnosed subtypes of examined cases are itemized in Table 3, with possible corresponding histological types according to Kidd.[17] As case 1 was predominantly comprised of epithelial tubuli, and as case 2 was admixture of tubuli and glomeruloid structures, both cases were subclassified as epithelial type. Ten cases were nephroblastic type, in which five cases showed tubular differentiation. Complex subtype was seen in two cases, showing rather prominent epithelial components at case 3, and containing rhabdoid structures in the stromal components at case 4. Case 5 was designated as focal nephroblastic subtype with a feature of clear boundary between mesenchymal stroma and blastemal islands, in which obvious tubular structure and cystically dilated tubuli are seen with papillary projection. Cases 6 to 12 were subclassified as diffuse nephroblastic subtype with blastemal predominance lacking clear boundary from stromal elements. Cases 6 and 7 were diffuse nephroblastic type with tubular differentiation. Rosette structures were found in cases 1 to 8. Cases 9 to 12 were comprised of diffuse proliferation of closely packed blastema with or without mesenchymal stroma, devoid of structures of tubuli or rosettes. Case 13 was mesenchymal type, almost lacking blastemal or epithelial components. Cases of 14, 15 and 16 were sarcomatous variants of nephroblastoma, i.e. abortive subtype. Cases 14 and 15 were malignant rhabdoid tumor of the kidney, and the case 16 was clear cell sarcoma of the kidney.

Figure 2 Binding pattern of lectins to normal adult kidney.

a. LTA reactivity is seen in the brush border of the proximal tubuli. (x 100)

b. PNA stains the luminal surface of the distal tubuli including Henle's loops. (x 100)

c. SBA is positive in both proximal and distal tubuli. (x 100)

d. UEA-1 reactivity is seen in the luminal surface of the collecting duct. (x 100)

Table 3 Histopathology of Wilms' tumors examined.

Histological Classification According to Urano et al.[23]	Case Number	Corresponding Histological Classification According to Kidd[17]
Epithelial type	cases 1 and 2	Classic triphasic Wilms' tumor
Nephroblastic type		
Complex subtype	cases 3 and 4	Classic triphasic Wilms' tumor
Focal nephroblastic subtype	case 5	Classic triphasic Wilms' tumor
Diffuse nephroblastic subtype	cases 6-12	Classic triphasic Wilms' tumor (case 6 and 7) Poorly differentiated Wilms' tumor (blastemal predominant) (case 8-12)
Mesenchymal type	case 13	Fetal rhabdomyomatous nephroblastoma
Abortive type		
Malignant rhabdoid tumor of the kidney (MRTK)	cases 14 and 15	Malignant rhabdoid tumor of the kidney
Clear cell sarcoma of the kidney (CCSK)	case 16	Clear cell sarcoma of the kidney

Lectin binding pattern of nephroblastoma

The results by lectins in nephroblastomas are summarized in Table 4. Positive reaction to lectins was almost exclusively confined to the epithelial cell of the tubular structure. Blastemal cells and mesenchymal stromas were negative for all lectins except PNA, but positive for them in rhabdoid cells of case 4. Rosette and glomeruloid structures were also negative for any lectin. There were no positive stains in three cases of abortive subtype. All seven cases showing tubular differentiation were positive to PNA at their luminal surface of tubuli in and around blastemal islands (Fig. 3a). About for epithelial type, PNA was positive even in immature tubuli which have not yet formed their lumina (Fig. 3b). SBA was positive in six cases, and UEA-1 was so in five cases (Fig. 3d). SBA was clearly positive in the case 1 showing epithelial type, in the case 5 focal nephroblastic type, and cases 6 and 7 of diffuse nephroblastic type (Fig. 3c). In the case 2 of epithelial type and the case 3 of complex type, SBA

Table 4 Lectin binding pattern of nephroblastoma cases.

Case No.	Histological subtype	PNA	LTA	UEA-1	SBA
1	Epithelial	+	+	+	+
2	Epithelial#	+	+	+	+/-
3	Complex	+	+/-	+/-	+/-
4	Complex ##	+	+/-	-	-
5	Focal nephroblastic	+	+	+/-	+
6	Diffuse nephroblastic (tubuli+)	+	-	-	+
7	Diffuse nephroblastic (tubuli+)	+	-	+/-	+
8	Diffuse nephroblastic (rosset+)	-	-	-	-
9	Diffuse nephroblastic (tubuli-,rosette-)	-	-	-	-
10	Diffuse nephroblastic (tubuli-,rosette-)	-	-	-	-
11	Diffuse nephroblastic (tubuli-,rosette-)	-	-	-	-
12	Diffuse nephroblastic (tubuli-,rosette-)	-	-	-	-
13	Mesenchymal	-	-	-	-
14	Malignant rhabdoid tumor of the kidney (MRTK)	-	-	-	-
15	Malignant rhabdoid tumor of the kidney (MRTK)	-	-	-	-
16	Clear cell sarcoma of the kidney (CCSK)	-	-	-	-

Staining degree shown as -: negative +/- : positive in only a few tubuli +: positive
: Lectins negative to glomeruloid bodies
##: PNA focally positive to stromal cells

bound only to a few tybuli. The case 4 of complex type showing poor epithelial components failed to react against SBA. UEA-1 was clearly positive in case 1 and case 2 of epithelial type. In cases of 3, 5 and 7, UEA-1 stain was very weak. LTA was clearly positive in epithelial and focal nephroblastic types. In the complex type, LTA bound only to a few tubuli. Cases 6 and 7 of diffuse nephroblastic type with tubular differentiation failed to react against LTA. By serial sections, we confirmed some tubular structures shared receptor for LTA and PNA in cases 1, 2 and 5 (Fig. 3e and 3f). However, LTA receptor didn't coexist with UEA-1 receptors at any tubuli.

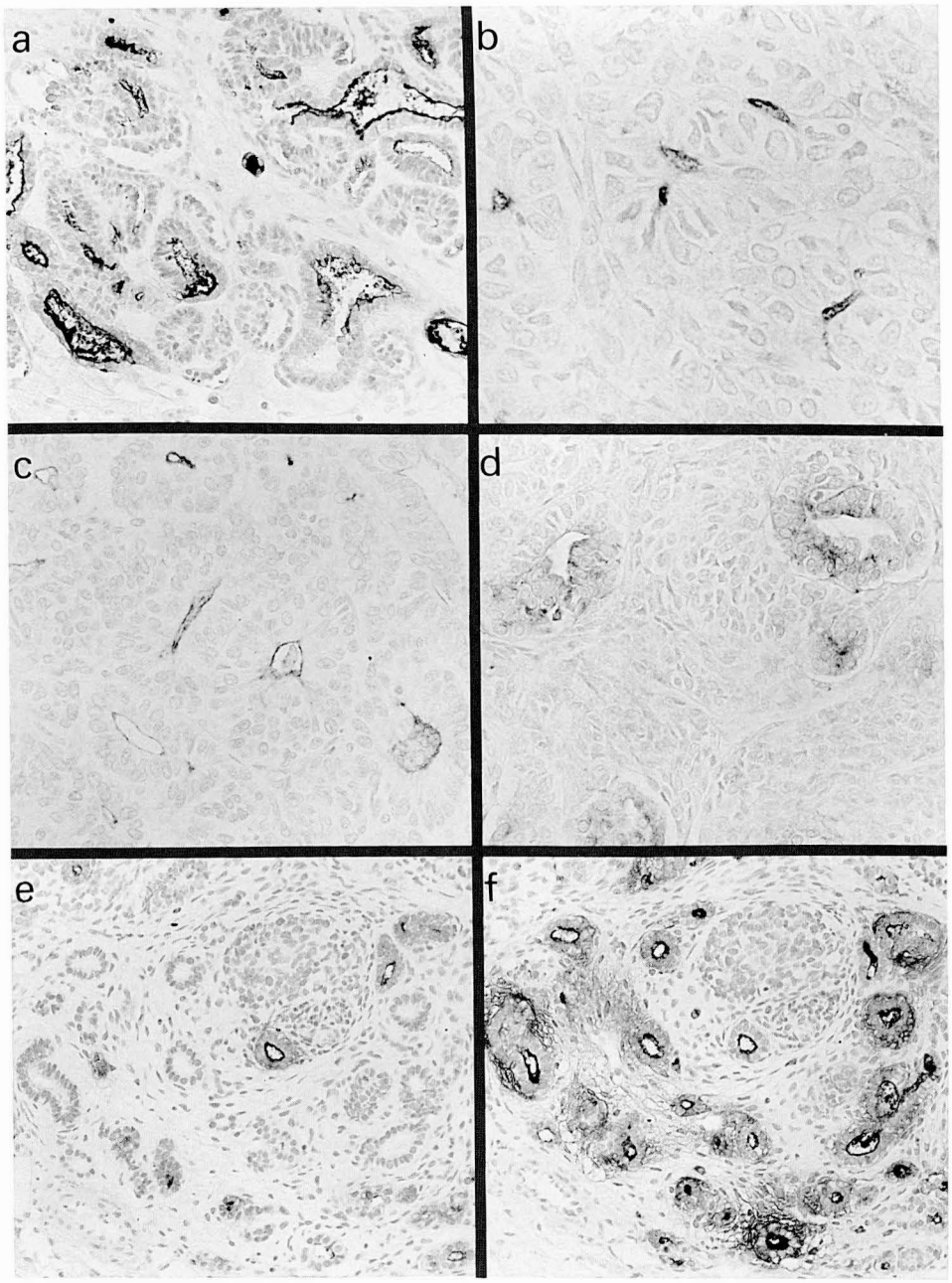

Fig. 3

4. Renal cell carcinoma

Histopathology

According to the classification of Japanese Pathological Society,[19] renal cell carcinoma was subdivided morphologically by their predominant cell type and their main histological architecture. When two or more architectures co-exist in examined cases, they were counted redundantly. There were 41 cases of clear cell predominant subtype and 9 cases of granular cell one. Thirty three cases of renal carcinoma were mainly comprised of alveolar structure and 23 cases were admixture of alveolar and tubular structure. Papillary and cystic structures were partial findings of predominantly alveolar or combined alveolar and tubular carcinoma. No cases showed pure papillary or papillotubular appearance suggesting Bellini's duct carcinoma. In 6 cases, solid or sarcomatous architecture was found including a case of pseudo-sarcomatous renal cell carcinoma.

Lectin binding pattern of renal cell carcinoma

The staining reaction to each lectin according to cell type or histological architecture is summarized in Table 5. UEA-1 was completely negative in whole cases of renal cell carcinoma. The difference of binding pattern between PNA, LTA and SBA was indistinct. They were stained lineally along the luminal surface of tubular or cystic structures, or along the immature lumina of alveolar structure (Fig. 4a). Papillary structure was stained lineally along the surface (Fig. 4b). The cytoplasm of the granular cells was rarely stained. The solid tumor structure comprised

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Figure 3 Binding pattern of lectins to the nephroblastoma.

a. PNA stain in case 3; Tubular structure is positive for PNA in its luminal surface. Glomeruloid body is negative for PNA. (x 66)

b. PNA stain in case 1: Positive reaction is observed in immature tubuli which have not yet formed their lumina. (x 200)

c. SBA stain in case 5: The luminal surface of the tubular structure is positive for SBA. (x 126)

d. UEA-i Stain in case 1: UEA-1 is positive in the luminal surface and parts of tubular cytoplasms. (x 126)

e. LTA stain in case 2: (x 66)

f. PNA stain in case 2: Note the co-expression of both PNA and LTA stains in some tubuli. (x 66)

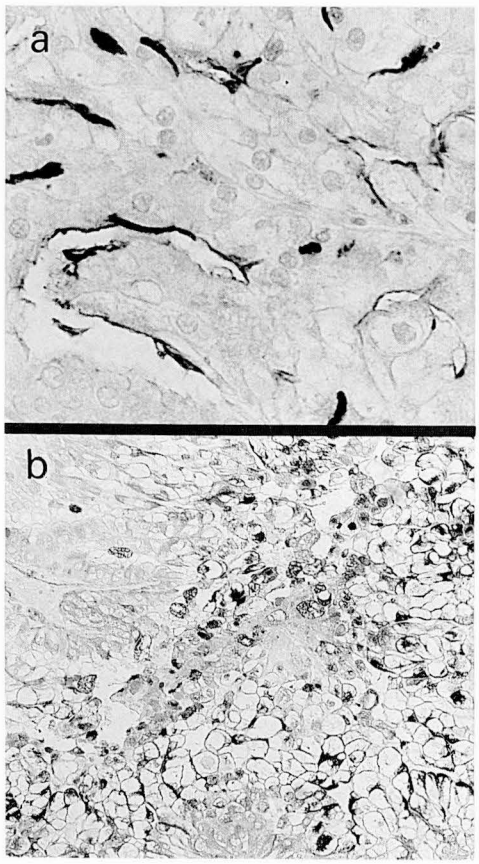

Figure 4 Binding pattern of lectins to the renal cell carcinoma.
a. LTA is positive in the midst of alveolar structure and along the luminal surface of tubular structure. (x 200)
b. Papillary structure shows positive reaction for PNA along its surface. (x 100)

of pleomorphic or spindle cells was completely negative to any lectins. Except the solid structure, other histological architecture didn't effect significantly the frequency of positive reaction to each lectin. For example, PNA was positive in 43% (24/56) of alveolar structure, 52% (12/23) of tubular structure, 50% (4/8) of papillary structure and 59% (13/22) of cystic structure. As for the cell type, SBA was positive in 75% (12/16) of granular cell carcinoma and 95% (39/41) of clear carcinoma showing a little difference in positive rate. However, there was no significant difference of positive rate against PNA and LTA between granular cell carcinomas and clear cell one. In total cases of renal cell carcinoma, PNA was positive in 49% (28/57), LTA was positive in 54% (31/57) and SBA was positive in 91% (51/57).

Table 5 Lectin positive cases of renal cell carcinoma according to cell type.

Cell type	Number	PNA	LTA	UEA-1	SBA
Granular	16 cases	9/16	7/16	0/16	12/16
Clear	41 cases	19/41	24/41	0/41	39/41
Total(%)	57 cases	28/57 (49)	31/57 (54)	0/57(0)	51/57(91)

DISCUSSION

In this study, we choose lectins specific to rather restricted segments of renal tubule. Solid type renal cell carcinoma and abortive or poorly differentiated nephroblastoma, both lacking tubular structure, revealed no affinities to all lectins. So, it is clear that the expression of these lectin receptors represents the tubular differentiation, even in tumor cells. Human kidney gultamyltranspeptidase which localizes in brush border of proximal tubuli contains X antigenic determinant.[26] Staining reaction of normal human kidney by LTA well reflects this fact. The emergence of Lewis X antigenic determinant in renal tumors may also represents such functional differentiation of tumor cells.

UEA-1 is a lectin which specifically binds to type2 H antigen. However, ABO blood type were not examined in this study,

since no cases of ABO blood type influenced the staining property of renal tissues. Earlier studies concerning to the expression of blood type antigen in nephroblastoma suggested that the expression of H antigen for nephroblastoma was independent from the ABO blood type.[10)]

Morphological change in earlier renal development is now precisely clarifying by means of ultrastructural and immunohistochemical techniques.[6)] Proximal tubuli develop from the middle portion of S-body subsequently to the early glomerular differentiation, but distal tubuli from the upper portion of the S-body later. Cell surface glycosylation during nephrogenesis has been studied on murine kidneys to date.[18)] However, surface sugar residue of the kidney is quite different among animal species,[12,14)] and little is known about the human embryo and fetus.[5,26)] We found that Gal β 1-3 GalNAc appeared at first in the luminal surface of ureteric buds and in developing tubuli at 10 gestational weeks. Simultaneously, type2 H antigen appeared in ureteric buds. However, Lewis X antigen and SBA binding sites were not observed until 13 gestational weeks after the morphological differentiation of proximal and distal tubuli. Proximal tubuli expressed both Gal β 1-3 GalNAc and Lewis X antigen in this stage. Gal β 1-3 GalNAc on proximal tubuli were negative in 19 gestational weeks when the lectin binding pattern of the nephron became identical with that of adult kidneys. Thus, there are some discrepancies between morphological differentiation and cell surface glycosylation of renal tubuli.

It is widely believed that the nephroblastoma is derived from the metanephros, and that it shows an array of histological differentiation representing various stages of normal nephrogenesis.[2,3)] Recent immunohistochemical studies using fetal kidneys as an age control, support this concept.[7,8,15)] In our study, tubular structures were constantly positive for PNA which is identical to an earlier report by Yegar et al.[27)] Intense staining attitude by LTA was observed in three well differentiated cases histologically with abundunt tubular structures. Tubular structures in less differentiated diffuse nephroblastic types failed to react with LTA. In spite of its broad affinity in adult nephrons, the staining to SBA was weak or negative even in well differentiated cases. This was probably influenced by late emergence SBA binding sites during embryogenesis. Type2 H antigen was positive in six

Table 6 Lectin binding pattern of renal cell carcinoma according to histological classification*.

Histological Classification	Number**	PNA	LTA	UEA-1	SBA
Alveolar	56 cases	24/56	21/56	0/56	52/56
Tubular	23 cases	12/23	12/23	0/23	20/23
Papillary	8 cases	4/ 8	3/ 8	0/ 8	0/ 8
Cystic	22 cases	13/22	6/22	0/22	18/22
Solid	6 cases	0/ 6	0/ 6	0/ 6	0/ 6

* The criteria of histological classification according to Machida et al.[19]
** Cases showing two or more histological structures were redunduntly counted.

cases which specifically bound the ureteric bud in the embryonal and fetal kidneys. Previous studies have suggested that the ureteric bud components may participate in the histogenesis of nephroblastoma.[8,10,11,15,20,25] Our results support this concept, since there were no tubuli which co-expresses the type2 H antigen and Lewis X antigen. Hence, type2 H antigen positive tubuli can be considered to express the differentiation toward the ureteric bud or the collecting duct. The degree of positive UEA-1 staining was so weak as scarcely recognizable in less well differentiated type of nephroblastoma cases. In the ureteric buds of the early renal development the UEA-1 stain was very faint and became gradually intense as renal development. These findings suggest clearly that the emergence of these lectin receptors may correlate closely to regular alteration of lectin receptors in embryonal and fetal kidneys. Furthermore, there was co-expression of both Gal β 1-3 GalNAc and Lewis X antigen at same tubuli in three well differentiated cases cited above. This finding may point out the common histochemical characteristics between these cases and the fetal kidney of 13 gestational weeks, in which Gal β 1-3 GalNAc remain in differentiated proximal tubuli. It is possible from these facts that Lewis X antigen negative nephroblastoma cases represent an earlier stage of cell surface glycosylation than that of a fetus in 13 gestational weeks.

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From ultrastructural and histochemical studies, renal cell carcinoma has been believed to originate from proximal tubuli with rare exceptions of tumors derived from collecting ducts known as Bellini's duct carcinoma.[1,24] Recently, an argument is arising against this concept. Immunohistochemical studies using antibodies specific to lower nephron showed an expression of distal tubuli and/or collecting ducts specific antigens in renal cell carcinomas, with simultaneous expression for specific antigens against proximal tubuli.[1,7,8] Similar findings were observed also in our

result with regard to cell surface sugar residues. In earlier reports, there are considerable discrepancies in staining pattern or positive rate of lecins in renal cell carcinoma.[1,4,13,21,22,25] However, many authors pointed out frequently positive reaction for PNA. Our results revealed that Gal β 1-3 GalNAc and Lewis X antigen were both positive in about a half of examined cases, but several cases showed coexpression of these two receptors, which were in line with a previous report of Chen.[4] The positive reaction of SBA was double rate of PNA or LTA. It is clear that the sugar composition of cell surface in renal cell carcinoma represents a more primitive state of tubular differentiation than that of normal proximal tubuli. The cell type and histological growth pattern didn't influence significantly such expression of lectin receptors, which may imply for renal cell carcinoma not to represent histogenetic phase as that seen in nephroblastoma.

In conclusion, we would like to emphasize a regular sequence of cell surface glycosylation is retained in nephroblastoma. These histochemical results will be useful for diagnosis in nephroblastoma.

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