THE PROGRESSION OF MEDULLARY RAY INJURY IN KIDNEY AGING

Shinichi Nishi,[1] Shunsuke Goto,[1] Kentaro Nakai,[1] Jun Ito,[1] Hideki Fujii,[1] Shigeo Hara[2]
[1] Division of Nephrology and Kidney Center, Kobe University Graduate School of Medicine, Kobe, Japan
[2] Department of Diagnostic Pathology, Kobe University Graduate School of Medicine, Kobe, Japan

OBJECTIVES

Aging changes in kidney progress with life time and affect the renal function. In general, GFR decreases with aging without proteinuria and hematuria. It is believed the number of global glomerular sclerosis increases with aging.

Pathological aging changes in kidney have not entirely cleared yet. Especially the reason why GFR decreases without abnormal urinalysis is an unresolved and important problem. We evaluated the histological changes related to aging in normal renal tissues.

Materials and Method

Method1:

We evaluated the histological findings of donor kidneys of 19 cases from 27 to 65 years-old who received kidney transplantation from 2010 to 2011. Among them 4 patients were hypertensive patients. We counted the rate of total glomerular sclerosis (GS index), the scores of arteriosclerosis; the degree of fibroelastosis change (AS index) and arteriolosclerosis; degree of hyalinosis (Ao index). (Fig.1)

Method2:

Additionally we observed the large renal sections obtained from nephrectomized kidneys due to renal cancer. Eight cases from 29 to 79 years-old were subjective. Among them 3 patients were hypertensive patients. Nephrectomized cases had no proteinuria. The large renal sections contained wide regions from superficial cortex to deep medulla.

RESULTS

Donor cases

case	age	f/m	S-Cr	eGFR	CCr	SBP	DBP	UP	HT
	y.o.		mg/dL	ml/min/1.73m^2	ml/min/1.73 m^2	mmHg	mmHg		
case 1	27	m	[2.5]			116	62	(-)	
case2	33	f	0.48	117	166	96	54	(-)	
case 3	35	f	0.58	78	149	105	54	(-)	
case 4	43	m	0.93	75	120	106	66	(-)	
case 5	49	m	0.57	87	134	100	58	(-)	
case 6	50	m	0.78	83	112	100	66	(-)	
case 7	56	f	0.61	78	94	110	67	(-)	
case 8	56	f	0.72	65	87	127	90	(-)	
case 9	58	m	0.80	81	154	126	80	(-)	
case 10	60	f	0.56	83	135	118	70	(-)	○
case 11	60	f	0.60	77	108	108	68	(-)	
case 12	61	m	0.73	84	104	103	60	(-)	
case 13	61	f	[1.8]					(-)	
case 14	63	f	0.51	92	120	102	68	(-)	
case 15	65	m	1.06	55	88	115	58	(-)	
case 16	65	f	0.64	70	88	101	58	(-)	○
case 17	66	f	0.63	71	103	130	76	(-)	○
case 18	67	f	0.69	64	100	116	78	(-)	○
case 19	74	m	0.68	86	98	108	76	(-)	

Case 1 and 13 are caderveric donors. HT: hyper tension treatment

Fig.1

GS index: the rate of global sclerosis

$$\text{GS index} = \frac{\text{globally sclerosed glomeruli N}}{\text{globally sclerosed glomeruli N+ open glomeruli N}}$$

AS index: the degree of small artery (SA) sclerosis (arteriosclerorsis)

$$\text{AS index} = \frac{\text{EL1 SA N+(EL2 SA N}\times 2\text{)+(EL3 SA N}\times 3\text{)}}{\text{All SA N}}$$

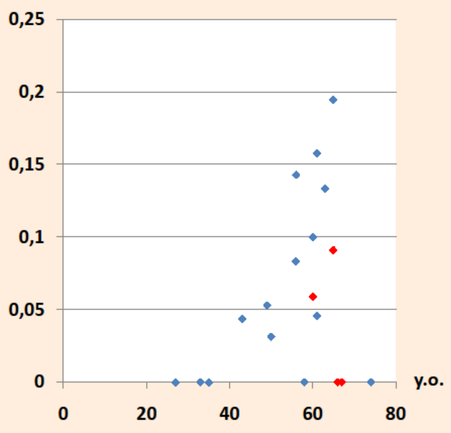
numbers of elastic lamella in small artery
EL1: 1
EL2: 4〜6
EL3: 7〜

AO index: the degree of arteriolar hyalinosis (AH) (arteriolosclerosis)

$$\text{AO index} = \frac{\text{AH1 A N+(AH2 A N}\times 2\text{)+(AH3 A N}\times 3\text{)}}{\text{All A N}}$$

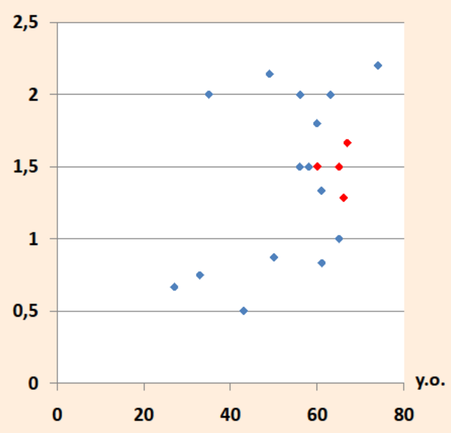
hyalinosis lesion in arteriole
AH1: <1/3 entire circle
AH2:1/3〜2/3 entire cir
AH3:>2/3 entire circle

Fig.2a



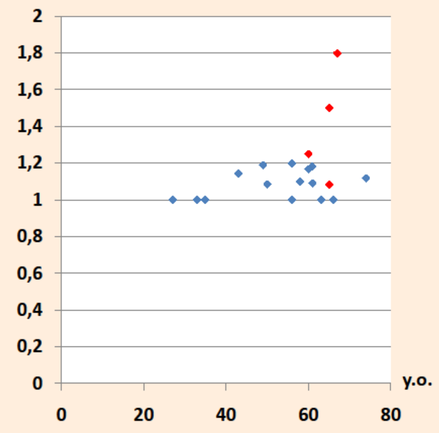
GS and ages showed no significant relationship.

Fig.2b



SA and ages showed a significant relationship. $p < 0.05$.

Fig.2c



AO and ages showed no significant relationship.

Nephrectomized cases

case	years	F/M	S-Cr	eGFR	SBP	DBP	UP	HT
	y.o.		mg/dL	ml/min/1.73m^2	mmHg	mmHg		
case 1	29	m	0.47	122	110	64	(-)	
case 2	37	f	0.47	117	118	78	(-)	
case 3	46	m	4.83	83	116	60	(-)	
case 4	60	f	0.92	91	106	60	(-)	
case 5	72	m	0.94	61	140	86	(-)	
case 6	75	f	0.88	64	124	692	(-)	○
case 7	77	m	0.84	68	122	78	(-)	○
case 8	79	m	0.15	37	138	80	(-)	○

HT: hyper tension treatment

Fig.3

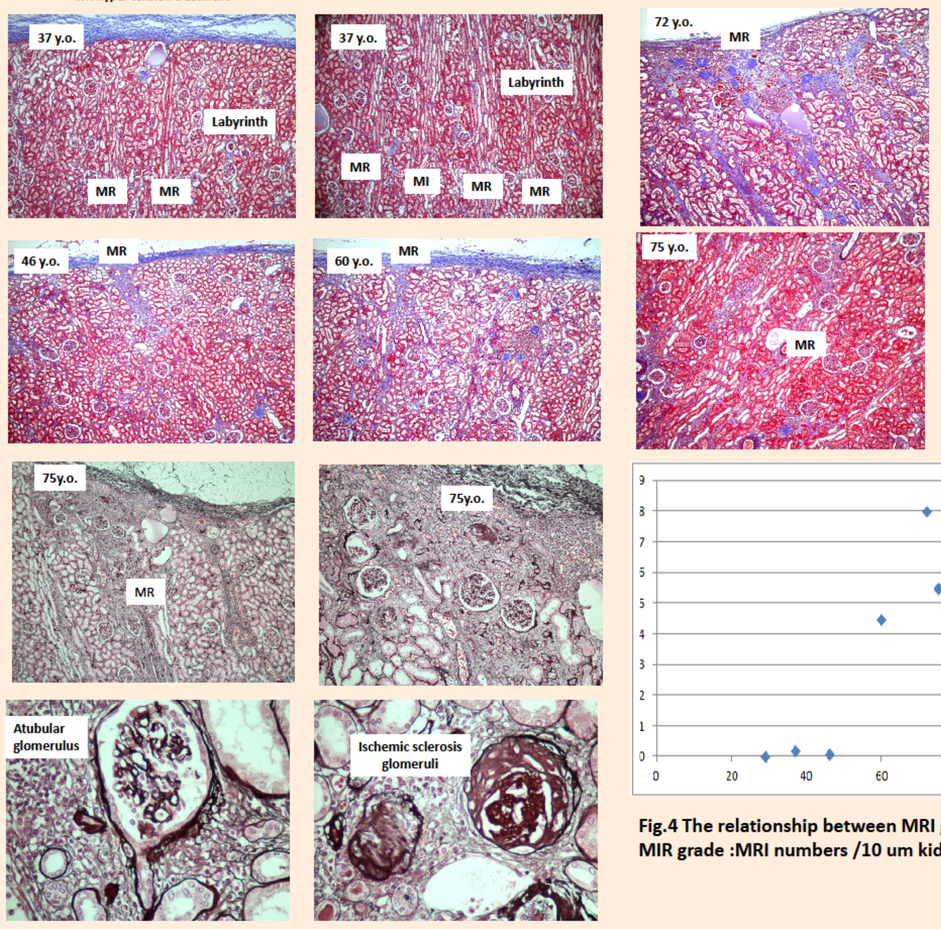


Fig.4 The relationship between MRI grade and ages. MIR grade :MRI numbers /10 um kidney surface

24h-Ccr of donors significantly decreased according to the ages . At the evaluation of pre-perfusion biopsies, all open glemeruli were minor glomerular abnormalities but the GS index increased from 0 to 0.20 according to the ages (Fig.2a). AS index showed the rising tendency according to the ages (Fig.2b), while AO index did not increase according to the ages except for hypertensive patients (Fig.2c). In the observation of the large renal sections from nephrectomized kidneys, the similar changes were noted in GS index, AS index and AO index. According to the ages the fibrotic changes appeared in medullary rays (MR) (Fig.3). The medullary ray fibrosis was limited in cortex under the renal capsule in middle age cases less than 60 years-old. In higher age cases the medullary ray fibrosis spread from superficial cortex to deep medulla in linear forms. The degree of medullary ray fibrosis increased according to the ages (Fig.4). Many open and sclerosed glomeruli were included in the fibrotic medullary ray lesions. Glomeruli in the medullary may fibrosis seemed to be atubular glomeruli and ischemic glomerui.

CONCLUSIONS

From the donor kidney biopsies we could only noticed arteriosclerosis (AS) progressed as aging. The GS index did not significantly elevate as aging, thus we did not prove the main reason of GFR decline was the progression of GS in donor biopsies. In large renal sections medullary ray fibrosis progressed according to the ages. We considered that the main reason of GFR decline without urinary abnormalities in aging depended to medullary injury injuries(MRI) including atubular glomeruli and ischemic glomeruli in medullary ray fibrotic lesion. A tubular and ischemic glomeruli involved in post-glomerular MRI may not cause proteinuria.

COI

I have no interest conflict to this poster presentation.

51ERA-EDTA
383--MP
L) Renal pathology. L3) Renal histopathology.
SHINICHI NISHI
DOI: 10.3252/pso.eu.51era.2014
Poster presented at: 51 ERA-EDTA CONGRESS AMSTERDAM THE NETHERLANDS MAY 31 JUNE 3 2014
PosterSessionOnline.com