Blood pressure change in the tyrosine-kinase inhibitors treatment of dialysis patients with renal cell carcinoma

Kentaro Nakai[1)], Hideki Fujii[1)], Masato Fujisawa[2)], Shinichi Nishi[1)]

1) Division of Nephrology and Kidney Center, Kobe University Graduate School of Medicine
2) Department of Organs Therapeutics, Division of Urology, Kobe University Graduate School of Medicine

OBJECTIVES

◆ **Malignant tumor** is more frequently occurred and a higher risk of death in dialysis patients than in general population[1-3]. Especially, **dialysis patients with renal cell carcinoma** (RCC) have a significantly higher relative risks compared to general population (3.6 to 24.1 SIR) [4].

◆ Recently, **molecular targeted therapy** is recommended for patients with advanced RCC. However, there are few reports about the molecular targeted therapy for dialysis patients with RCC. Furthermore, **the rise in blood pressure** is one of the adverse effects due to the **tyrosine-kinase inhibitors (TKI) treatment**[5,6], but is not fully understood in dialysis patients.

METHODS

◆ Among **dialysis patients** who were admitted in our hospital from 2010 to 2013, those with **kidney cancer** were enrolled. Of 32 cases, three patients were excluded from the present study. Two patients received only best supportive care because their general condition was too poor to receive an invasive surgery, 1 patient did not have sufficient clinical data because he was treated on an outpatient basis.

◆ Finally, we compared clinical characteristics, treatment strategy and blood pressure change over a 1-month period between patients with only surgery (control group, n=16) and those with surgery plus TKI treatment (TKI group, n=13). Their mean age at start of treatment was 61 ± 8 years, 76% were men, and dialysis vintage was 11.9 ± 11.4 years. Eleven of 29 patients were died and the median overall survival time was 65 months.

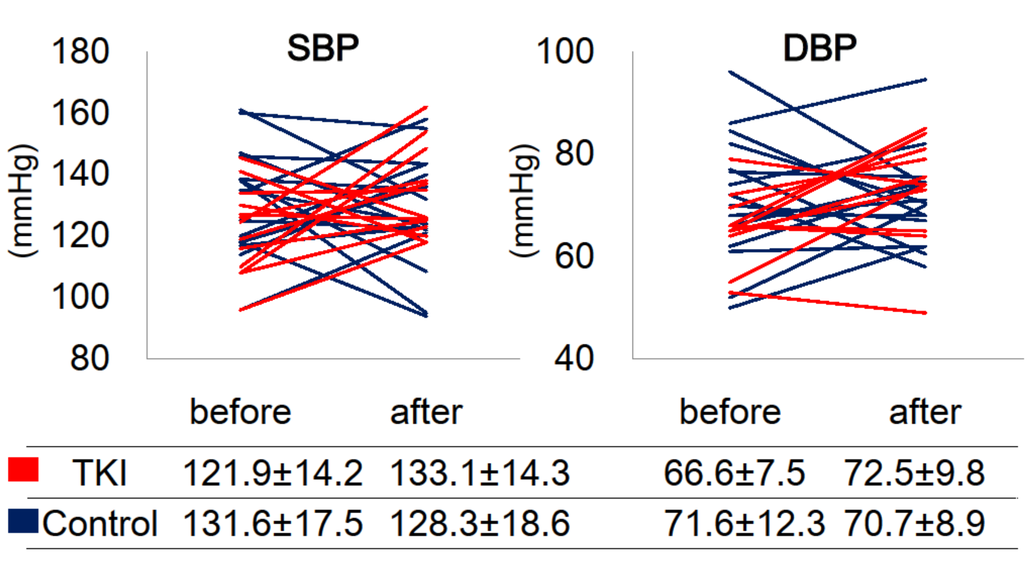
	TKI (n=13)	Control (n=16)	p value
Sex (male: female)	11:2	11:5	ns
Age (years old)	63±6	60±9	ns
Dialysis vintage (years)	8.9±11.9	15.0±10.6	ns
Primary kidney disease (%)			ns
Diabetes mellitus	2 (15)	2 (13)	
Glomerulonephritis	5 (38)	7 (44)	
Antihypertensive agents (%)	8 (62)	10 (63)	ns
Stage (I / II / III / IV; %)	**15 / 0 / 15 / 70**	**75 / 6 / 6 / 13**	**p<0.01**
Observation period (months)	43.2±33.1	29.0±14.1	ns
Death (%)	**8 (62)**	**3 (19)**	**p<0.05**

RESULTS

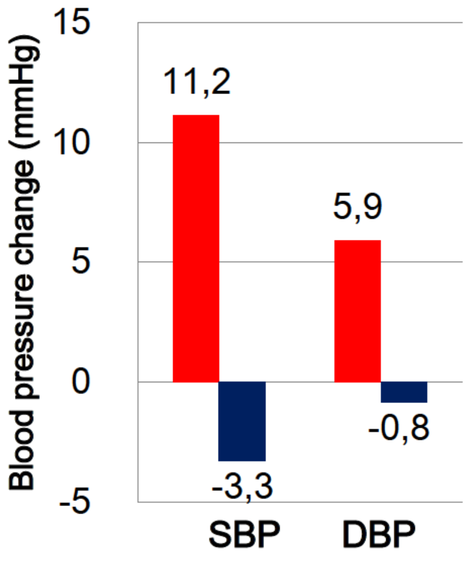
◆ The change of blood pressure before and after treatment tended to be higher in the TKI group compared to the control group.

◆ The percentage of patients who need management of hypertension was 69.2% in the TKI group and 25.0% in the control group (p<0.05).

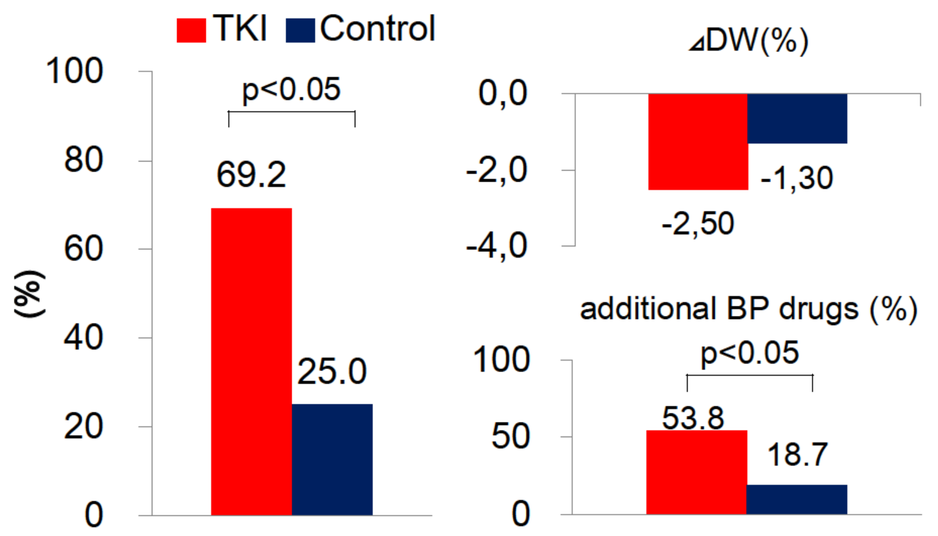
【The change of Blood Pressure before and after treatment】



	SBP before	SBP after	DBP before	DBP after
TKI	121.9±14.2	133.1±14.3	66.6±7.5	72.5±9.8
Control	131.6±17.5	128.3±18.6	71.6±12.3	70.7±8.9



【The need for management of hypertension】
(defined as either the additional BP drugs or the decrease of DW by 3% or more)



CONCLUSIONS

◆ Our study showed that **TKI treatment** was associated with **blood pressure elevation** especially in dialysis patients.

◆ Therefore, we should pay more attention to the change of blood pressure and control it appropriately in these patients.

All authors declare that they have no conflict of interest.

REFERENCES:

1. Stewart JH, et al. The pattern of excess cancer in dialysis and transplantation. Nephrol Dial Transplant. 24(10): 3225-31, 2009.
2. Maisonneuve P, et al. Cancer in patients on dialysis for end-stage renal disease: an international collaborative study. Lancet. 354(9173): 93-9, 1999.
3. Iseki K, et al. Evidence for increased cancer deaths in chronic dialysis patients. Am J Kidney Dis. 22(2): 308-13, 1993.
4. Holley JL. Screening, diagnosis, and treatment of cancer in long-term dialysis patients. Clin J Am Soc Nephrol. 2(3): 604-10, 2007.
5. Kandula P, et al. Proteinuria and hypertension with tyrosine kinase inhibitors. Kidney Int. 80(12): 1271-7, 2011.
6. Lankhorst S, et al. Mechanism of hypertension and proteinuria during angiogenesis inhibition: evolving role of endothelin-1. J Hypertens. 31(3): 444-54, 2013.

52 ERA
745--FP
F2) Hypertension. Clinical.
Kentaro Nakai
DOI: 10.3252/pso.eu.52era.2015
ePosters supported by F. Hoffmann- La Roche Ltd.
52nd ERA-EDTA CONGRESS London 2015 UNITED KINGDOM MAY 28 '31
PosterSessionOnline.com