Abstract

Introduction: Hemodialysis patients cause coronary artery diseases to a higher rate and have a poor survival prognosis. Brachial ankle pulse wave velocity (baPWV) is a reliable measurement of arterial stiffness, and could predict mortality in patients with end-stage renal diseases. It is generally thought that atherosclerosis is not improved by hemodialysis treatments. We retrospectively examined whether hemodialysis treatments improved atherosclerosis or not, using the value of baPWV/systolic blood pressure (SBP) as an arteriosclerotic index.

Methods: We examined the relationship between the value of baPWV, SBP and platelet counts of 14 hemodialysis patients and assessed the change of baPWV for 13 patients who were measured baPWV more than twice.

Results: 7KH YDOXH RI ED3:9 DQG 6%3 VKRZHG WKH VLJQL;FDQW SRVLWLYH FR the value of baPWV/SBP was used as an arteriosclerotic index. Value of 10 hemodialysis patients showed the

Keywords: Atherosclerosis; Hemodialysis; Decreased platelets count; Klotho gene

Introduction

Patients receiving maintenance hemodialysis for end stage renal disease have a worse survival and morbidity prognosis. e number of hemodialysis patients is annually increasing worldwide. Cardiovascular mortality rates among hemodialysis patients are approximately from 40% to 50% of deaths [1,2]. e cardiovascular mortality rates of the hemodialysis patients are higher than those of the general population by at least 10 times to 20 times. When a hemodialysis period becomes longer, the atherosclerosis is accelerated and patients develop cardiovascular diseases to a higher rate [1,3,4].

Patients with chronic kidney diseases died from much higher CVD than renal disease itself [5]. It is generally thought that atherosclerosis is not improved by hemodialysis treatments. Patients with CKD usually have traditional cardiovascular risk factors such as hypertension, hypercholesterolemia and diabetes mellitus.

Arterial sti ness can be assessed by noninvasive measurement of brachial ankle pulse wave velocity (baPWV), which is a simple and reproducible method [6,7]. baPWV re ects arterial wall structural components such as collagen and elastin, transmural pressure and

smooth muscle tone, which mainly regulates arterial vessel distensibilition author: Hidekazu Takeuchi, Nagasaki-ken Tomie Hospital 499 Tomie-chou, Gotou-city, Nagasaki 853-0205, Japan, Tel: +81-959-86-1121; and function [8,9]. baPWV is reported to be a crucial independent mail: takeuch-h@r8.dion.ne.jp determinant of cardiovascular risk [10,11]. And baPWV could predict Received April 08, 2013; Published July 11, 2013 mortality in patients with hypertension [12], type 2 diabetes [13], and Citation: Takeuchi H (2013) A Novel Risk Factor of Aggravating Atherosclerosis end-stage renal diseases [14].

in Hemodialysis Patients; the Decreased Platelet Counts. 2: 735 doi: 10.4172/

e objective of the present study was retrospectively to elucidate VFLHQWL 355UHSRUWV whether hemodialysis treatments improve atherosclerosis or not, using pyright: © 2013 Takeuchi H. This is an open-access article distributed under the value of baPWV/systolic blood pressure (SBP) as an arteriosclerosic distribution, and reproduction in any medium, provided the original author and index and to clarify what the essential factor is. source are credited.

Page 2 of 5

signi cance [14], we used the value of baPWV/SBP to evaluate arterivalue of baPWV/SBP. e change was shown in Table 3. Among three stin Tw l8sg3cance [it baherosclerosis ofr hee preurpe patients (23%) who were not improved, the value of baPWV/SBP of two patients (Table 3; No.11 and No.12) was exacerbated slightly, but the baPWV/SBP value of the third patient (Table 3; No.13) was

Among 10 patients with improved baPWV/SBP values, their platelet counts were more than 100,000/ I. However, three patients whose baPWV/SBP values were not improved had the platelet counts less than 90,000/ I. And there was statistically signi cant di erence between their platelet counts of the two groups (Figure 2; p<0.001). e three worsened patients were not infected by hepatitis C virus or hepatitis B virus.

Discussion

aggravated largely.

According to the results of the present study, hemodialysis treatments improved atherosclerosis of 10 hemodialysis patients, but atherosclerosis of three hemodialysis patients (23%) were not improved. When atherosclerosis of hemodialysis patients was evaluated, it was proper to consider the in uence of hypertension on arterial wall and to use the value of baPWV/SBP as an arteriosclerotic index for hemodialysis patients.

Cardiovascular risk factors such as hypertension, in ammation, oxidative stress have been studied intensely [15]. In addition to these common cardiovascular risk factors, other factors such as uramic milieu and hemodialysis procedure itself such as heparin and dialysis

Page 3 of 5

counts of the improved group (Table 3; No.1; 100,000/ I) were larger than the largest platelet counts of the worsened group (Table 3; No.11; 86,000/ I).

Presently, although the hemodialysis patients were treated with medical interventions such as beta-blockers, statins and erythropoietin [4], decreased platelet counts (<90,000/ I) were not the target of the treatment. e decreased platelet counts may have been implicated in exacerbating atherosclerosis in patients with hemodialysis treatments and they may predict the bad prognosis.

e reason why platelet counts in the worsened group were less than those of the improved group remains elusive.

Patients with chronic kidney disease (CKD) have higher risks of cardiovascular disease and mortality than the normal population [18]. Klotho is the gene associated with attenuating the progression of hypertension, anti-aging, mineral metabolism, and vitamin D metabolism and encodes a single-pass transmembrane protein that forms a complex with multiple broblast growth factor receptors, and is most abundant in the renal tubules [19]. A defect in Klotho gene expression in mice results in shortened lifespan and aging-like phenotypes [20-23]. Recent studies also showed that Klotho functions as a renoprotective factor [24,25]. Klotho expression is decreased in patients with acute or chronic kidney diseases in response to reactive oxygen species [26].

Previous studies indicated that uremic toxins increased the oxidative stress and reduced cell viability [27,28]. An animal study showed that

	No.	baPWV (cm/s)		
_				
_				
_				
_				
_				
_				
_				
—				
e risk factors for hemodialysis	5			
st report to demonstrate that				
00/1) were associated with th	e			
modialysis patients. In Figure 2 d group (No.11~No.13) had the	2, Ə			

membrane are known to be possible risk factors for hemodialysis patients [16,17].

As far as we know, this is the rst report to demonstrate that the decreased platelet counts (<90,000/I) were associated with the exacerbation of atherosclerosis in hemodialysis patients. In Figure 2, the box plot showed that the worsened group (No.11~No.13) had the signi cantly (p<0.001) lower values in the serum level of platelet counts than those of the improved group (No.1~No.10) and the least platelet

Citation: Takeuchi H (2013) A Novel Risk Factor of Aggravating Atherosclerosis in Hemodialysis Patients; the Decreased Platelet Counts. 2: 735 doi: V F L H Q W L785 U H S R U W V

Page 5 of 5

- Adijiang A, Niwa T (2010) An oral sorbent, AST-120, increases Klotho expression and inhibits cell senescence in the kidney of uremic rats. Am J Nephrol 31: 160-164.
- Kuro-o M, Matsumura Y, Aizawa H, Kawaguchi H, Suga T, et al. (1997) Mutation of the mouse klotho gene leads to a syndrome resembling ageing. Nature 390: 45-51.
- Arking DE, Krebsova A, Macek M Sr, Macek M Jr, Arking A, et al. (2002) Association of human aging with a functional variant of klotho. Proc Natl Acad Sci U S A 99: 856-861.
- Arking DE, Atzmon G, Arking A, Barzilai N, Dietz HC (2005) Association between a functional variant of the KLOTHO gene and high-density lipoprotein cholesterol, blood pressure, stroke, and longevity. Circ Res 96: 412-418.
- Arking DE, Becker DM, Yanek LR, Fallin D, Judge DP, et al. (2003) KLOTHO allele status and the risk of early-onset occult coronary artery disease. Am J Hum Genet 72: 1154-1161.
- 34. Kempe DS, Ackermann TF, Fischer SS, Koka S, Boini KM, et al. (2009) \$FFHOHUDWHG VXLFLGDO HU\WKURF\WH GHDWK LQ 458: 503-512.