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Research Article

Risk Factors of Arterial Damage Assessed by ABI and baPWV among Hemodialysis Patients in Macau

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Cardiovascular disease (CVD) is the leading cause of mortality and morbidity among patients with ESRD on chronic dialysis. Arterial damage is one of the characteristics of CVD. But the association between arterial damage and conventional risk factors for CVD has not yet been fully highlighted in chronic hemodialysis patients. Here we validate the clinical value of assessment of arterial damage by ABI and PWV in chronic hemodialysis patients in Macau.

1. Background

Cardiovascular disease (CVD) is the leading cause of mortality and morbidity among patients with ESRD on chronic dialysis [1]. According to the US Renal Data System and Hong Kong Renal Registry reports, CVD accounts for approximately 40% of mortality in dialysis patients [2, 3].

Arterial damage, including stenosis and stiffness of arteries, is one of the characteristics of CVD. Several noninvasive measurements are introduced to identify the abnormal structure and function of arteries [4]. Ankle-brachial index (ABI) serves as a reliable sign for diagnosing peripheral artery disease. A reduced ABI relates to further development of angina, myocardial infarction, stroke, and need for coronary bypass surgery. And it is a powerful independent predictor of all-cause and cardiovascular mortality in hemodialysis (HD) patients [5, 6]. Pulse wave velocity (PWV), which provides a comprehensive assessment of arterial stiffness, is also a powerful and independent predictor of all-cause and cardiovascular mortality in ESRD patients [7, 8].

Classic cardiovascular risk factors, such as blood pressure, diabetes mellitus, and hyperlipidemia, have been implicated in accelerated arterial damage. However, findings of many studies in general population were inconsistent with respect to risk factors other than age and blood pressure [9]. And the association between arterial damage and those risk factors has not yet been fully highlighted in chronic hemodialysis patients. In consideration of the genetic contribution to arterial damage [10], it is meaningful to validate the clinical value of assessment of arterial damage by ABI and PWV in chronic hemodialysis patients in Macau.

2. Methods

2.1. Subjects. We retrospectively studied all chronic HD patients who were aboriginal inhabitants of Macau (all of the enrolled patients were born in Macau and in most of time they lived in Macau) in this cross-sectional study from March 2012 to June 2012. Patients who met the following criteria were enrolled.

Entry Criteria. These include age of 18 or older, 1 year or more of duration of dialysis, and dry weight being kept stable for at least 3 months before enrollment.

Exclusion Criteria. These include presence of clinically overt congestive heart failure (NYHA class III-IV); persistent hypotension despite pharmacological therapy (defined as systolic blood pressure (SBP) < 90 mmHg or diastolic blood

pressure (DBP) <60 mmHg); atrial fibrillation; acute infectious disease; liver cirrhosis with ascites; tumor; malnutrition; bilateral amputation of lower limbs; and bilateral arteriovenous fistula over arms (patients who had bilateral arteriovenous fistula over arms were not enrolled in this study).

Informed consents were obtained before enrollment. And this study was approved by the Ethical Committee of Centro Hospitalar Conde de São Januário.

2.2. Demographic and Clinical Data Collection

Data Included. The data included were age, gender, body weight, height, blood pressure, illness history, and etiology of ESRD.

2.3. Treatment of Uremia. Four-hour HD thrice weekly and the target of KT/V should be higher than 1.4. EPO and iron were prescribed to keep the pre-HD level of Hb within the range of 10–12 g/dL. Calcium, Vitamin D and its analogy, and phosphate binder were given individually or collectively to meet the target range of calcium, phosphate, and PTH which were 2.1–2.5 mM, 1.13–1.78 mM, and 2–9 times of the upper limit of normal, respectively.

2.4. Biochemical Tests. Fasting blood samples were collected in the morning. Measurements were performed using routine laboratory methods for such serum parameters as creatinine, calcium, phosphate, albumin, blood glucose, HbA1C, total cholesterol, triglyceride, and low- and high-density lipoprotein. Serum intact parathyroid hormone (iPTH) was measured by Nichols immunoradiometric assay.

2.5. ABI Measurement. The ABI was measured using VP-1000 vascular profiler (Nippon Colin Ltd., Komaki, Japan) in HD patients before hemodialysis and at least 15-minute supine rest (the time of measurement was not specialized). ABI was automatically calculated as the ratio of ankle SBP to brachial SBP for each side and the lower value was used for analysis [11]. All the ABI measurements were performed by one experienced operator and the intraobserver coefficient of variation was about 2.68–4.45%.

2.6. PWV Measurement. The brachial-ankle PWV (baPWV) was assessed using VP-1000 vascular profiler (Nippon Colin Ltd., Komaki, Japan) in HD patients before hemodialysis and at least 15-minute supine rest (the time of measurement was not specialized; PWV may change significantly after ultrafiltration measurement is taken before hemodialysis), which allowed online pulse wave recording and automatic calculation of PWV. Briefly, baPWV was calculated from $(D1-D2)/T$. $D1$ is the distance between the heart and ankle, $D2$ is the distance between the heart and brachium, and T is the transit between the right brachial arterial wave and right tibial arterial wave. The distances between the sampling points are automatically calculated from the patient's height and are divided by the time interval for the waveform from each measuring point [12]. The baPWV was performed in HD patients before hemodialysis and at least 15-minute supine rest. Two measurements were performed in each arm, and the average value was used for the analysis. All the PWV measurements were performed by one experienced operator and the intraobserver coefficient of variation was about 1.58–3.36%.

2.7. Statistical Analysis. Continuous variables with normal distribution were expressed as means ± standard deviation. Univariate analysis was done to explore relationships between 2 variables by Pearson correlation test for data with bivariate normal distribution and Spearman rank correlation test for nonparametric data. Stepwise multiple linear regression analysis was used to assess the independent determinants of ABI and baPWV. A two-tailed $P < 0.05$ was considered as statistically significant. All statistical analyses were performed with the statistical software Stata 7.0 (Computer Resource Center, USA).

3. Results

3.1. Patient Characteristics. A total of 312 maintenance HD patients (176 males/136 females) were enrolled. Etiology of ESRD is diabetic nephropathy ($n = 111$), lupus nephritis ($n = 4$), primary nephritis ($n = 72$), hypertensive nephrosclerosis ($n = 53$), chronic interstitial nephritis ($n = 3$), and unknown etiology ($n = 41$). Vascular access type is arteriovenous fistula ($n = 254$), permanent catheter ($n = 57$), and arteriovenous graft ($n = 1$). Table 1 shows the demographic and clinical characteristics of the enrolled HD patients. On average of all, ABI was 1.03 ± 0.20 and baPWV was 2037.14 ± 653.14 cm/s.

3.2. Univariate Analysis. Table 2 shows the effects of exposure factors on ABI and baPWV. Risk factors for ABI include age ($P = 0.000$), male ($P = 0.000$), high pulse pressure ($P = 0.037$), and hyperglycemia ($P = 0.007$). Risk factors for PWV include age ($P = 0.000$), high pulse pressure ($P = 0.000$), hyperglycemia ($P = 0.030$), and hyperphosphatemia ($P = 0.030$). The impact of heart rate on arterial stiffness was not probed in consideration of the variability in heart rate.

3.3. Multiple Linear Regression Analysis. In a stepwise multiple linear regression analysis, we employed ABI and baPWV value as a dependent variable, while using age, gender, HD duration, BMI, SBP (systolic blood pressure), DBP (diastolic blood pressure), MAP (mean arterial pressure), PP (pulse pressure), FBG (fasting blood glucose), HbA1c (hemoglobin A1c), cholesterol, triglycerides, LDL-C (low density lipoprotein cholesterol), HDL-C (high density lipoprotein cholesterol), Ca (calcium), P (phosphorus), Ca × P (Calcium-phosphate product), PTH, KT/V (index of urea clearance), and RRF (residual renal function) as independent variables (Table 3).

Risk factors for decreased ABI: every 1 unit there is an increase of age; SBP, DBP, PP, FBG, Ca × P, and ABI will be reduced by 0.005 ($P = 0.000$), 0.002 ($P = 0.001$), −0.002 ($P = 0.002$), 0.004 ($P = 0.000$), 0.001 ($P = 0.011$), and 0.034 ($P = 0.037$), respectively. For male, ABI will be reduced by

Table 1: Baseline characteristics of the study population.

Items	Total (n = 312)
Age (years)	61.47 ± 14.42
Gender (female)	45.39% (136)
Occupation	25.96% (81)
Married	76.6% (239)
Ever smoker	3.52%
Previous CVD history	17.3% (54)
Diabetes mellitus	35.58%
HD duration (months)	66.97 ± 66.72
SBP (mmHg)	147.88 ± 26.52
DBP (mmHg)	78.71 ± 15.13
PP (mmHg)	69.17 ± 21.32
MAP (mmHg)	101.76 ± 16.92
BMI (kg/m^2)	22.32 ± 4.29
RAS inhibitor	33.97%
Hyperlipidemia	20.19%
Serum albumin (g/L)	43.58 ± 4.52
Cholesterol (mmol/L)	4.36 ± 1.06
Triglycerides (mmol/L)	1.79 ± 1.21
LDL cholesterol (mmol/L)	2.26 ± 0.87
HDL cholesterol (mmol/L)	1.30 ± 0.45
Hemoglobin (g/dL)	11.57 ± 1.56
Hematocrit (%)	34.41 ± 5.26
Calcium (mmol/L)	2.46 ± 0.21
Phosphate (mmol/L)	1.61 ± 0.51
Calcium-phosphate product [(mmol/L)^2]	3.92 ± 1.22
Intact-PTH (pg/mL)	366.98 ± 423.97
CRP	1.09 (0.12, 25.2)
Kt/V	1.54 ± 0.34
ABI	1.03 ± 0.20
PWV (cm/s)	2037.14 ± 653.14

0.059, as compared to female (P = 0.002). Liner correlation between LDL-C and ABI was found in patients whose level of LDL-C was higher than 2.6 mmol/L. Every 1 unit increase of LDL-C, ABI will be reduced by 0.088 (P = 0.006).

Risk factors for increased baPWV: every 1 unit there is an increase of age; BMI, MAP, PP, Ca × P, and baPWV will be increased by 12.028 (P = 0.000), 23.779 (P = 0.003), 3.664 (P = 0.044), 5.982 (P = 0.017), and 7.183 (P = 0.026), respectively. Liner correlation between LDL-C and baPWV was found in patients whose level of LDL-C was higher than 2.6 mmol/L. Every 1 unit increase of LDL-C, baPWV will be increased by 214 (P = 0.0022).

4. Discussion

A multitude of traditional and uremia-specific risk factors may cause abnormality of structure and function of arteries [13], which would lead to arterial stenosis and stiffness. ABI and PWV are introduced in the present study in view of their potential advantages in predicting arterial damage before the onset of clinical diseases.

Although carotid-femoral PWV (cfPWV) is considered as a well-established index of central arterial stiffness [14], there are some obvious limitations [15]. Namely, it is difficult for clinical operator to use pressure transducers on target arteries. And it is unacceptable for some subjects to expose inguinal area during the acquisition of femoral pressure waveforms. BaPWV was used in place of cfPWV in the present study in consideration of the fact that baPWV reflects both central and peripheral arterial stiffness [16].

It was indicated that arterial structure was in part influenced by genetic factors [10]. To avoid the interference of heredity and environment as far as possible, we conducted this study only in aboriginal inhabitants of Macau.

Men have a higher risk of getting heart disease than women who are still menstruating. Gender is one of the CVD risk factors that cannot be changed. Data showed that, for male, ABI will be reduced by 0.059, as compared to female (P = 0.002).

Age-related structural change of arteries mainly involves the media of aorta. Consistent with the previous study that there was a linear relationship between age and stiffness of central artery [17], our study also revealed age-related peripheral arterial disease in hemodialysis patients.

A negative correlation between pulse pressure and ABI, while a positive one between pulse pressure and baPWV, was confirmed, respectively, in this study, as the result of the large epidemiological study in general population [18]. Hypertension may cause arterial damage and inversely sclerotic change of arteries may induce hypertension. So it is not difficult to understand that hypertension serves as both a risk factor and a marker of impaired vascular compliance.

Hypercholesterolemia, as one of classic cardiovascular risk factors, has been implicated in accelerated arterial stiffening. However, it was not the determinant of accelerated arterial stiffness in normotensive subjects and in treated hypertensive subjects [19]. In our study, the correlation between LDL-C and arterial damage cannot be demonstrated in univariate analysis in which subjects were divided in 2 groups according to the level of LDL-C with the cut point of 2.6 mmol/L. But in multiple regression analysis, in patients with LDL-C higher than 2.6 mmol/L, a linear relationship was found between LDL-C and ABI or baPWV. The possible explanation may be that the relationship between LDL-C and ABI or baPWV is not completely linear, and both high and low level of LDL-C would relate to arterial damage in hemodialysis patients. In keeping with this point, it was reported that there was increased risk of CVD in dialysis patients with the lowest level of blood lipids [20].

To compare with hemoglobin A1c (HbA1c), test of blood glucose gives a more accurate picture of diabetes control in dialysis patients. A lower level of HbA1c would be found in dialysis patients, because shorter life span of red cells reduces the time that glucose interacts with hemoglobin in the bloodstream [21]. This explains why blood glucose, instead of HbA1c, was associated with ABI and baPWV in the present study.

Our research suggests that serum calcium had no correlation with arterial damage, while calcium-phosphate product showed a risk trend of arterial damage, similar to that seen

Table 2: Effects of exposure factors on ABI and baPWV: univariate analysis.

	Group		ABI				PWV (cm/s)			
	Group 1 (n)	Group 2 (n)	Group 1	Group 2	Z value	P value	Group 1	Group 2	Z value	P value
Age (year)	≤65 (200)	>65 (100)	1.127 ± 0.145	1.035 ± 0.194	4.478	0.0000	1821 ± 539.7	2085 ± 490.4	−4.987	0.0000
Gender	Male (128)	Female (172)	1.065 ± 0.150	1.119 ± 0.177	−4.117	0.0000	1971 ± 591.2	1863 ± 490.4	1.147	0.2516
Smoking	No (248)	Yes (52)	1.093 ± 0.168	1.109 ± 0.171	−0.608	0.5429	1912 ± 536.9	1898 ± 545.7	0.252	0.8008
HD duration (year)	≤3 (119)	>3 (155)	1.112 ± 0.177	1.097 ± 0.154	0.920	0.3576	1943 ± 532.7	1872 ± 520.9	1.392	0.1639
BMI (kg/m^2)	≤25 (231)	>25 (69)	1.095 ± 0.166	1.098 ± 0.177	0.366	0.7142	1887 ± 521.3	1985 ± 586.3	−1.230	0.2185
SBP (mmHg)	≤140 (107)	>140 (192)	1.103 ± 0.152	1.095 ± 0.172	0.479	0.6322	1799 ± 464.4	1971 ± 567.3	−2.491	0.0127
DBP (mmHg)	≤90 (234)	>90 (66)	1.086 ± 0.175	1.130 ± 0.139	−1.332	0.1828	1925 ± 543.4	1856 ± 516.6	1.342	0.1795
MAP (mmHg)	≤105 (169)	>105 (131)	1.082 ± 0.182	1.114 ± 0.146	−0.900	0.3682	1850 ± 516.5	1986 ± 556.1	−1.841	0.0656
PP (mmHg)	≤60 (107)	>60 (193)	1.122 ± 0.152	1.081 ± 0.175	2.087	0.0369	1769 ± 507.2	1987 ± 539.3	−4.339	0.0000
FBG (mmol/L)	≤6.1 (163)	>6.1 (137)	1.118 ± 0.152	1.070 ± 0.182	2.526	0.0115	1850 ± 508.9	1975 ± 566.6	−2.173	0.0298
HbA1c (%)	<6.5 (37)	≥6.5 (84)	1.035 ± 0.237	1.048 ± 0.175	0.515	0.6067	2014 ± 589.4	2072 ± 602.9	−1.257	0.2086
Cholesterol (mmol/L)	≤5.8 (261)	>5.8 (39)	1.095 ± 0.168	1.101 ± 0.170	−0.173	0.8625	1910 ± 524.3	1907 ± 626.3	0.402	0.6879
Triglycerides (mmol/L)	≤1.7 (170)	>1.7 (130)	1.087 ± 0.172	1.107 ± 0.163	−0.799	0.4241	1918 ± 498.9	1899 ± 586.0	0.457	0.6479
LDL-C (mmol/L)	≤2.6 (196)	>2.6 (104)	1.098 ± 0.169	1.093 ± 0.167	0.441	0.6590	1939 ± 536.8	1853 ± 536.9	1.666	0.0957
HDL-C (mmol/L)	≤1.4 (177)	>1.4 (123)	1.085 ± 0.184	1.112 ± 0.141	−1.167	0.2431	1908 ± 569.6	1911 ± 489.9	−0.132	0.8950
Ca (mmol/L)	<2.5 (171)	≥2.5 (129)	1.110 ± 0.173	1.080 ± 0.161	2.678	0.0074	1910 ± 549.6	1907 ± 523.3	−0.111	0.9117
P (mmol/L)	≤1.78 (203)	>1.78 (97)	1.078 ± 0.180	1.130 ± 0.135	−1.905	0.0568	1954 ± 577.6	1815 ± 429.7	1.998	0.0457
CaXP $(mmol/L)^2$	≤4.44 (211)	>4.44 (89)	1.084 ± 0.179	1.125 ± 0.136	−1.111	0.2665	1943 ± 572.5	1830 ± 436.7	1.515	0.1297
PTH (pg/mL)	≤300 (180)	>300 (120)	1.086 ± 0.180	1.112 ± 0.148	−0.547	0.5844	1916 ± 557.3	1899 ± 508.6	0.550	0.5822
KT/V	≤1.4 (114)	>1.4 (185)	1.091 ± 0.168	1.102 ± 0.163	−0.558	0.5770	1867 ± 487.6	1934 ± 568.1	−0.628	0.5300
RRF	0 (171)	>0 (129)	1.089 ± 0.154	1.105 ± 0.185	−1.383	0.1667	1871 ± 490.4	1910 ± 537.5	−1.379	0.1680

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; PP: pulse pressure; FBG: fasting blood glucose; HbA1c: hemoglobin A1c; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; Ca: calcium; P: phosphorus; CaXP: calcium-phosphorus product; KT/V: index of urea clearance; RRF: residual renal function, defined as the numerical average of the urinary clearance of urea and creatinine.

Table 3: Multiple linear regression analysis of the factors associated with arterial damage.

	ABI			baPWV		
	Coefficient	*t* value	*P* value	Coefficient	*t* value	*P* value
Age	−0.005	−6.570	0.000	12.028	5.010	0.000
HD duration (year)	0.001	0.970	0.332	−1.827	−0.580	0.564
Gender (male)	−0.059	2.43	0.002	19.560	0.270	0.784
BMI (kg/m^2)	−0.001	−0.380	0.706	23.779	3.040	0.003
SBP (mmHg)	−0.002	3.240	0.001	0.208	0.100	0.917
DBP (mmHg)	0.002	3.050	0.002	0.527	0.260	0.797
MAP (mmHg)	0.001	1.320	0.187	3.664	2.020	0.044
PP (mmHg)	−0.004	−4.590	0.000	5.982	2.410	0.017
FBG (mmol/L)	−0.001	−2.600	0.011	0.539	0.400	0.687
HbA1c (%)	−0.009	−0.930	0.353	25.780	0.860	0.394
Cholesterol (mmol/L)	0.001	0.090	0.929	−7.664	−0.550	0.584
Triglycerides (mmol/L)	0.005	0.440	0.661	47.998	1.550	0.123
LDL-C (mmol/L)	−0.026	−1.890	0.060	9.675	0.230	0.816
HDL-C (mmol/L)	0.015	0.510	0.612	126.769	1.430	0.155
Ca (mmol/L)	−0.001	−0.440	0.662	0.441	0.240	0.809
P (mmol/L)	−0.0559	3.000	0.003	88.645	1.47	0.142
CaXP $(mmol/L)^2$	−0.034	2.100	0.037	7.183	0.130	0.026
PTH (pg/mL)	0.001	1.590	0.113	0.061	0.600	0.551

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; PP: pulse pressure; FBG: fasting blood glucose; HbA1c: hemoglobin A1c; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; Ca: calcium; P: phosphorus; CaXP: calcium-phosphorus product.

with serum phosphorus in the univariate analysis. And the risk associated with calcium-phosphate product was independent of PTH. These findings are consistent with the previous national study that revealed the association of calcium-phosphate product with mortality in hemodialysis patients [22]. It is especially true in dialysis patients that elevated calcium-phosphate product promotes the accumulation of calcium-phosphate crystals in the media or intima of the arterial wall, which would result in arterial damage. Both high and low level of PTH may influence the metabolism of calcium and phosphate. The relationship between PTH and ABI or baPWV is not completely linear either.

Several limitations of this study must be taken into consideration when interpreting the data. Firstly, cross-sectional design of the study did not allow us to determine causality. Secondly, due to the technical limitation of baPWV measurement, patients with atrial fibrillation or amputated extremity were excluded. However, these patients generally are at high risk of arterial damage. Thirdly, the age on the start of dialysis was not analyzed due to relatively small sample size. Fourthly, the long term impact of different vascular access on arterial stiffness is an interesting question and needs further study.

5. Conclusions

In conclusion, the presented data clearly showed that age, pulse pressure, LDL-C, fasting blood glucose, and calcium-phosphate product are the risk factors of arterial damage in hemodialysis patients in Macau.

Conflict of Interests

The authors declare that they have no conflict of interests.

Authors' Contribution

Mingxin Li and Jing Xin are equal contributors.

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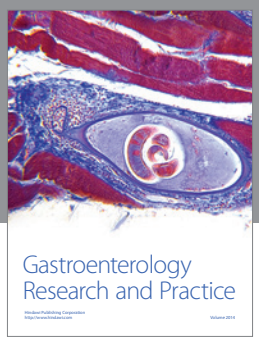

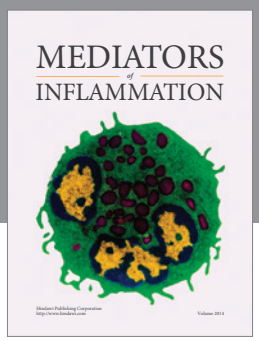

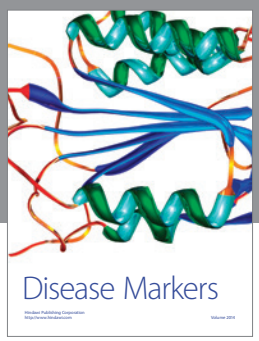

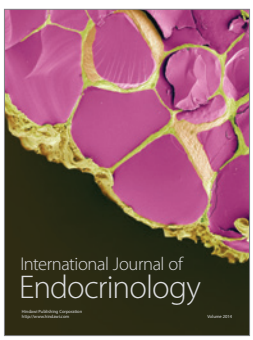

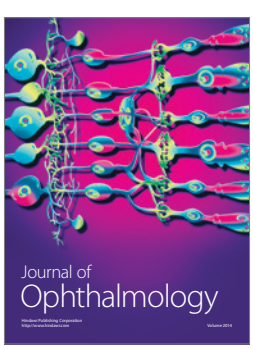

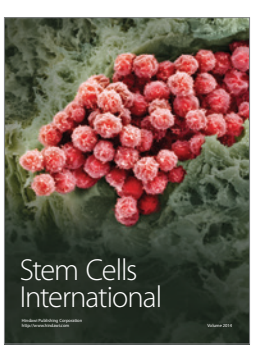

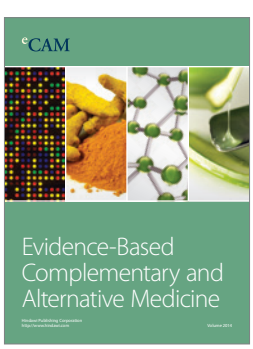

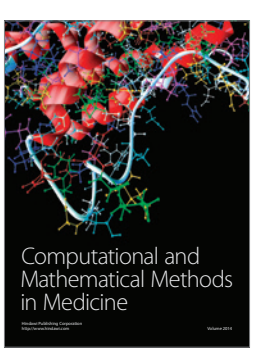

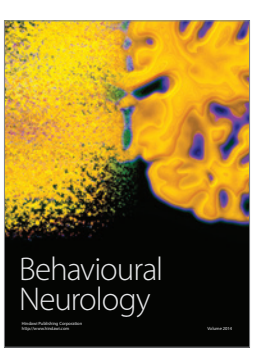

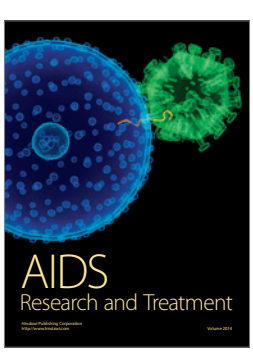

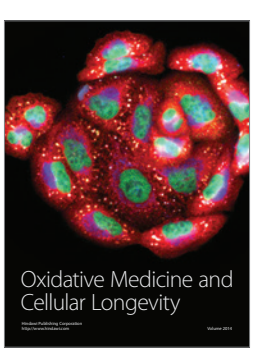

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