ORIGINAL ARTICLE

The change in the prevalence of obesity and new-onset diabetes in Chinese peritoneal dialysis patients over 25 years

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ABSTRACT

Background. The global prevalence of both obesity and end-stage kidney diseases (ESKDs) has increased in recent decades. Given the complicated interaction between obesity and ESKD, we examined the change in the prevalence of obesity in incident Chinese peritoneal dialysis (PD) patients over the past 25 years.

Methods. We reviewed the anthropometric measures of incident PD patients in a single Hong Kong center from 1995 to 2019. The results are reported in five 5-year periods. Patients with and without diabetes were analyzed separately, and the incidence of new-onset diabetes after PD was explored.

Results. We reviewed 1681 patients. Their mean age was 58.4 ± 12.5 years; 931 patients (55.4%) had pre-existing diabetes. From 1995–99 to 2015–19, the prevalence of obesity or overweight at the initiation of PD increased progressively for every 5-year period (from 21.9% to 26.2, 37.9, 42.7 and 47.3%, $P < 0.001$ for linearity). The increase in the prevalence of obesity or overweight was more pronounced in diabetic patients (from 33.7% to 59.6%) than non-diabetic ones (from 13.2% to 32.3%). Among nondiabetics patients, the incidence of new-onset diabetes after started on PD showed an insignificant rising trend during that period (from 18.0, 19.7, 17.8 and 22.4% to 23.3%, $P = 0.106$). The incidence of new-onset impaired fasting glucose or diabetes was significantly higher in obese or overweight patients than the others (56.9% versus 51.4%, $P < 0.001$).
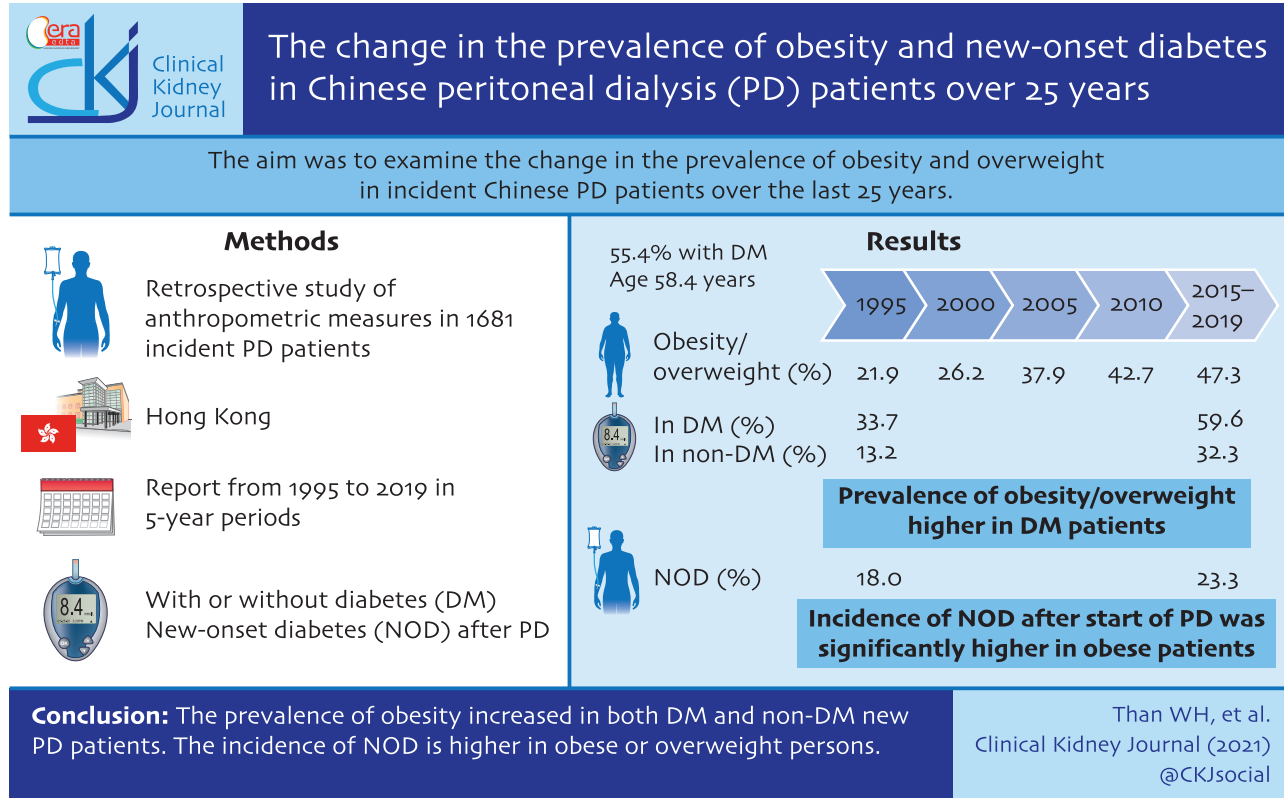
Conclusions. The prevalence of obesity has increased substantially in both diabetic and nondiabetic new PD patients in Hong Kong over the past 25 years. The incidence of new-onset diabetes was significantly higher in new PD patients with pre-existing obesity or overweight than those without obesity. The prognostic implication and impacts on the healthcare system deserve further studies.

GRAPHICAL ABSTRACT

The change in the prevalence of obesity and new-onset diabetes in Chinese peritoneal dialysis (PD) patients over 25 years

The aim was to examine the change in the prevalence of obesity and overweight in incident Chinese PD patients over the last 25 years.

Methods

Retrospective study of anthropometric measures in 1681 incident PD patients

Hong Kong

Report from 1995 to 2019 in 5-year periods

With or without diabetes (DM) New-onset diabetes (NOD) after PD

Results

55.4% with DM
Age 58.4 years

	1995	2000	2005	2010	2015–2019
Obesity/overweight (%)	21.9	26.2	37.9	42.7	47.3
In DM (%)	33.7				59.6
In non-DM (%)	13.2				32.3

Prevalence of obesity/overweight higher in DM patients

NOD (%)	18.0	23.3

Incidence of NOD after start of PD was significantly higher in obese patients

Conclusion: The prevalence of obesity increased in both DM and non-DM new PD patients. The incidence of NOD is higher in obese or overweight persons.

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INTRODUCTION

The number of patients with chronic kidney disease (CKD) around the world is approximately 850 million [1, 2]. From 1990 to 2016, the global incidence and prevalence of CKD increased by 89% and 87%, respectively [3]. In many western countries, the prevalence of end-stage kidney disease (ESKD) has increased in the past two decades [4, 5], and the burden of ESKD will probably continue to increase in the coming years because of the change in demographic, clinical and lifestyle factors, and the improvements in the provision of dialysis facilities [6]. For example, it has been estimated that in the USA, the incidence rate of ESKD will rise 11–18% between 2015 and 2030, and the number of prevalent patients is expected to rise from 690 000 to 1 115 000 during this period [6]. More importantly, the incidence and prevalence of ESKD will probably increase more drastically for many developing countries [5].

In many developed countries, population changes in age, obesity and diabetes prevalence, and ESKD survival are believed to be the major causes of the increasing prevalence of ESKD [6]. Notably, the prevalence of obesity has increased in the general population over the past two decades [7]. Although the prevalence of obesity is highest in the USA, Europe and the Eastern Mediterranean region [7], urbanization of rural areas has resulted in an increase in the prevalence of obesity in many countries

[8–10]. In China, the prevalence of obesity rose from 6.5% in 1997 to 14.8% in 2011, and the prevalence of overweight rose from 29.1% to 41.0% during that period [11].

Morbid obesity in itself causes obesity-related glomerulopathy, but, more commonly, obesity may increase the rate of progression of CKD from other causes [12, 13]. It has been estimated that obesity contributes to 18.4% of new onset of advanced CKD [14], and, above 27 kg/m^2, each unit increase in body mass index (BMI) leads to a 7% increase in the risk of development of ESKD among living kidney donors [15]. On the other hand, CKD also causes malnutrition and muscle wasting [16, 17]. Many CKD patients have progressive weight loss with a gradual decline in kidney function [18]. Given that complicated interaction, the change in the prevalence of obesity in new ESKD patients has not been well studied. The objective of this study is to describe the change in the prevalence of obesity in new ESKD patients in Hong Kong over the past two decades.

MATERIALS AND METHODS

Overall design

This is a retrospective analysis of data collected from two observational cohort studies, which were approved by the Joint Chinese University of Hong Kong—New Territories East Cluster Clinical Research Ethics Committee (approval numbers CREC-

2008.276 and CRE-2015.250). All study procedures were in compliance with the declaration of Helsinki. A total of 1681 incident adult peritoneal dialysis (PD) patients were reviewed. In essence, the two cohorts included all incident PD patients of our unit from 1995 to 2019. Patients who were planned to have elective living donor transplants or transferred to other renal centers within 6 months of PD were excluded. In general, anthropometric measures were performed at the time of PD catheter insertion, while dialysis adequacy and nutritional assessment, as well as fasting plasma glucose level, were performed around 4 weeks after patients were stable on PD. For this study, baseline demographic and clinical data, dialysis adequacy and nutritional assessment were collected by chart review.

Assessment of anthropometric measures

The body weight was measured at baseline and, if the patient was still on PD, 2 years afterward. BMI was calculated by the conventional formula:

$$BMI = body\ weight\ (kg)/[body\ height\ (m)]^2$$

The BMI was further categorized according to the Asia Pacific version of the World Health Organization classification [19, 20]. In essence, BMI $<18\ kg/m^2$ for underweight; between 18 and 22.9 kg/m^2 for normal weight; between 23 and 24.9 kg/m^2 for marginal overweight; between 25 and 29.9 kg/m^2 for overweight; and $\geq 30\ kg/m^2$ for obesity [19, 20].

Dialysis adequacy, nutrition and inflammation markers

The method of dialysis adequacy assessment has been described previously [21]. In essence, 24-h urine and dialysate collection were performed for the calculation of the total K_t/V. Nutritional status was represented by serum albumin level, subjective global assessment (SGA) overall score, comprehensive malnutrition-inflammation score (MIS), normalized protein nitrogen appearance (NPNA) and fat-free edema-free body mass (FEBM). For SGA, the four-item seven-point scoring system validated in PD patients was used [22]. The calculation of MIS included 10 items, each scored from 0 to 3, with a maximum score of 30 [23]. NPNA was calculated by the modified Bergstrom's formula [24]. FEBM was determined by the creatinine kinetic method according to the formula described by Forbes and Bruining [25] and further presented as the percentage of ideal body weight (IBW). We also measured the serum C-reactive protein (CRP) by a commercially available ultra-sensitive assay (Roche Diagnostics GmbH, Mannheim, Germany).

Pulse wave velocity study and bio-impedance spectroscopy study

From 2005, arterial pulse wave velocity (PWV) and multifrequency bio-impedance spectroscopy was performed for all new PD patients, generally around 1 month after they were stable on dialysis. PWV was measured by an automatic computerized recorder and analyzed using the Complior @ SP program (Artech Medical, France) by the method described previously [26]. We used the multi-frequency bioimpedance spectroscopy device (Body Composition Monitor, Fresenius Medical Care, Germany) as described previously [27, 28]. Data on lean tissue mass (LTM), adipose tissue mass (ATM) and volume of overhydration (OH) were retrieved for analysis in this study.

Glucose study

We reviewed the fasting plasma glucose level 1 month after the patient was on a stable PD regimen, with a constant degree of glucose exposure from the PD solutions, and was clinically euvolemic. The glucose level was categorized according to the American Diabetes Association classification and has been described previously [29]. In essence, fasting blood glucose $<5.6\ mmol/L$ denotes normal; impaired fasting glucose for 5.6 to $<7.0\ mmol/L$; diabetes for 7.0 to $<11.1\ mmol/L$; and overt diabetes for $\geq 11.1\ mmol/L$.

Statistical methods

Statistical analysis was performed by SPSS for Windows software version 25.0 (IBM, Armonk, NY, USA). The normality of the distribution was checked by the Shapiro–Wilk test. Summary statistics were described in frequency (%) for categorical variables and mean ± standard deviation or median (interquartile range) for continuous variables as appropriate. The longitudinal trend of baseline demographics and clinical data over different eras were compared by analysis of variance (ANOVA) and chi-square test. A value of P < 0.05 for the weighted linear term during ANOVA with linear polynomial contrast or that for the linear association in the chi-square test was considered statistically significant for linearity of the trend. Correlation or association between variables was checked by Spearman's rank correlation or Mann–Whitney U test as appropriate. The body weight or BMI between baseline and at Year 2 after baseline were compared by Student paired *t*-test. The prevalence or incidence and association between two categorical variables were calculated from Pearson's chi-square test or Fisher's exact test. Summary findings of Year 2 follow-up were calculated in the nested cohort. BMI category change was verified by McNemar's test. A value of P < 0.05 was considered statistically significant. All probabilities were two-tailed.

RESULTS

We reviewed 1681 incident PD patients. Their mean age was 58.4 ± 12.5 years; 958 patients (57.0%) were men; and 931 patients (55.4%) were diabetic. Mean BMI at baseline was 24.2 ± 4.4 kg/m^2, and mean body weight was 63.1 ± 13.7 kg. The trends for baseline demographics and clinical data during the study period are summarized in Table 1.

Prevalence of obesity over the years

Both body weight and BMI increased in new PD patients over the years. Waist and hip circumferences also had similar trends during this period (Table 1). The distribution of BMI categories at the initiation of PD over the years is shown in Figure 1. From 1995–99 to 2015–19, the prevalence of baseline obesity in new PD patients increased from 3.1% to 11.6%, and that of overweight increased from 18.8% to 35.7%.

We further evaluated the trend of anthropometric measures and the prevalence of obesity for diabetic and nondiabetic patients separately (Table 2). In essence, both groups showed a significant trend of increasing body weight and BMI over the study period and the prevalence of overweight and obesity increased over the years in both diabetic and nondiabetic patients. Of note, the increase in the prevalence of overweight and obesity was more pronounced in diabetic patients (from 33.7% in 1995–99 to 59.6% in 2015–19) than nondiabetic ones (from 13.2% to 32.3% during the same period).

Table 1. The trend of baseline demographic and clinical data over the study period

Era	1995–99	2000–04	2005–09	2010–14	2015–19	P-value
No. of patients	223	329	330	342	457	
M:F	127:96	173:156	187:143	192:150	279:178	0.084[a]
Age, years	54.0 ± 12.8	57.9 ± 13.5	58.3 ± 12.3	59.0 ± 12.0	60.8 ± 11.7	<0.001[b]
Body weight, kg	58.9 ± 9.9	58.8 ± 11.3	62.6 ± 13.4	64.3 ± 14.6	67.6 ± 14.9	<0.001[b]
Body height, cm	160.8 ± 7.6	159.6 ± 8.6	160.5 ± 8.4	161.3 ± 8.7	162.6 ± 8.2	<0.001[b]
BMI, kg/m^2	22.8 ± 3.5	23.1 ± 4.0	24.2 ± 4.3	24.5 ± 4.5	25.4 ± 4.5	<0.001[b]
Waist circumference, cm	85.8 ± 6.3	89.6 ± 11.4	92.3 ± 11.2	94.2 ± 12.5	94.4 ± 12.2	<0.001[b]
Hip circumference, cm	96.3 ± 4.3	94.4 ± 7.4	97.9 ± 8.4	99.3 ± 10.1	98.6 ± 11.8	0.004[b]
Blood pressure, mmHg						
Systolic	145.2 ± 22.3	145.5 ± 21.9	141.8 ± 21.3	143.1 ± 21.1	147.9 ± 20.9	0.016[b]
Diastolic	74.8 ± 11.8	76.4 ± 13.2	75.0 ± 12.8	76.5 ± 12.2	78.3 ± 13.3	0.004[b]
Underlying renal diseases, n (%)						<0.001[a]
Diabetic nephropathy	69 (30.9)	127 (38.6)	136 (41.2)	169 (49.4)	264 (57.8)	–
Glomerulonephritis	90 (40.4)	89 (27.1)	84 (25.5)	78 (22.8)	78 (17.1)	–
Hypertensive nephropathy	11 (4.9)	21 (6.4)	32 (9.7)	31 (9.1)	49 (10.7)	–
Polycystic kidney disease	9 (4.0)	10 (3.0)	10 (3.0)	8 (2.3)	9 (2.0)	–
Urological causes	9 (4.0)	19 (5.8)	12 (3.6)	15 (4.4)	7 (1.5)	–
Other specific causes	5 (2.2)	14 (3.9)	17 (5.1)	9 (2.6)	10 (2.2)	–
Idiopathic	30 (13.5)	50 (15.2)	39 (11.8)	32 (9.4)	40 (8.8)	–
Comorbidities, n (%)						–
Diabetes	95 (42.6)	160 (48.6)	182 (55.2)	207 (60.5)	287 (62.8)	<0.001[a]
IHD	53 (23.8)	80 (24.3)	88 (26.7)	78 (22.8)	94 (20.6)	0.179[a]
CVA	45 (20.2)	81 (24.6)	94 (28.5)	76 (22.2)	65 (14.2)	0.004[a]
Charlson's score	5.2 ± 2.3	6.0 ± 2.7	6.2 ± 2.7	6.1 ± 2.6	6.0 ± 2.2	0.007[b]
Residual GFR (mL/min/1.73 m^2)	2.2 ± 1.9	3.6 ± 2.2	4.0 ± 2.8	3.6 ± 2.7	4.3 ± 3.4	<0.001[b]

Data are presented as mean ± standard deviation or n (%). P-values for the trend of linearity; calculated by
[a]Chi-square test and
[b]ANOVA.
IHD, ischemic heart disease; CVA, cerebrovascular disease; GFR, glomerular filtration rate (1 month after dialysis).
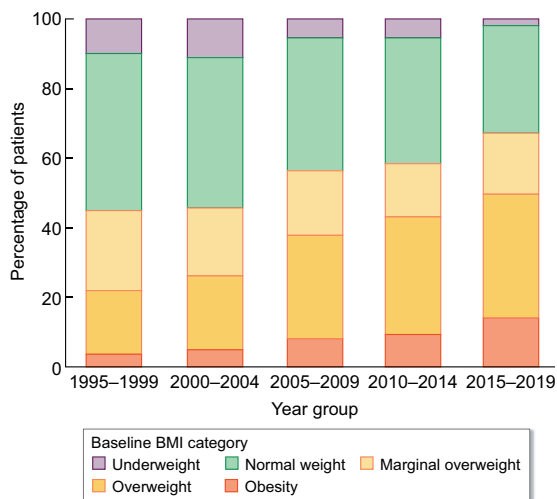

FIGURE 1: The distribution of BMI categories at the initiation of PD over different eras.

Nutritional and biochemical status

The relation between BMI and nutritional and biochemical parameters was explored and summarized in Table 3. In essence, most nutritional parameters, including blood pressure, fasting plasma glucose, lipid profile, serum albumin, subjective scoring and anthropometric measures by creatinine kinetics and multi-frequency bio-impedance, had modest but significant correlations with BMI, while total K_t/V and carotid-radial PWV had modest but significant inverse correlations with BMI. Notably, serum CRP level did not correlate with BMI.

The trends of baseline biochemical and nutritional parameters during the study period are summarized in Table 4. For the entire study cohort, there was a modest but significant improvement in some nutritional parameters (hemoglobin, MIS and NPNA) but not the others (serum albumin, SGA score and LTM by bioimpedance study). Notably, during the study period, there was a modest increase in absolute FEBM, but a marked reduction in the percentage of FEBM when adjusted for the IBW. When only obese or overweight patients were analyzed, most of the nutritional indices did not change during the study period, except for a slight increase in NPNA and reduction in the percentage of FEBM. The changes in baseline nutrition and biochemical status of diabetic and nondiabetic patients are further analyzed separately and summarized in Supplementary data, Table S1.

Acquired glucose intolerance

From 1995–99 to 2014–19, the post-dialysis fasting plasma glucose of nondiabetic patients gradually increased (from 5.9 ± 2.0, 5.9 ± 1.5, 5.8 ± 1.3 and 6.1 ± 1.7 to 6.4 ± 2.2 mmol/L for each consecutive 5-year period, P = 0.016 for linearity) The incidence of new-onset diabetes also showed a modest rising trend (from 18.0, 19.7, 17.8 and 22.4% to 23.3% for each consecutive 5-year period, P = 0.106 for linearity), but the trend did not reach

Table 2. The trend of anthropometric measures and the prevalence of obesity over the study period in new PD patients with and without diabetes

Year	Diabetic						Non-diabetic					
	1995–99	2000–04	2005–09	2010–14	2015–19	P-value	1995–99	2000–04	2005–09	2010–14	2015–19	P-value
No. of cases	95	159	182	207	287	<0.001[a]	128	169	148	135	170	<0.001[a]
Body weight, kg	61.7 ± 10.0	61.4 ± 11.5	65.5 ± 13.8	68.0 ± 14.4	71.1 ± 14.9	<0.001[b]	56.8 ± 9.3	56.4 ± 10.7	59.0 ± 12.0	58.6 ± 13.0	61.7 ± 12.9	<0.001[b]
BMI, kg/m^2	24.0 ± 3.5	24.1 ± 3.9	25.3 ± 4.1	25.7 ± 4.5	26.5 ± 4.5	<0.001[b]	21.9 ± 3.1	22.1 ± 4.0	22.8 ± 4.2	22.7 ± 3.8	23.7 ± 4.1	<0.001[b]
BMI category, n (%)	–	–	–	–	–	<0.001[a]	–	–	–	–	–	<0.001[a]
Underweight	8 (8.4)	6 (3.8)	3 (1.6)	6 (2.9)	0 (0.0)	–	14 (10.9)	30 (17.8)	15 (10.1)	13 (9.6)	9 (5.3)	–
Normal weight	30 (31.6)	63 (39.6)	54 (29.7)	57 (27.5)	60 (20.9)	–	71 (55.5)	79 (46.7)	73 (49.3)	67 (49.6)	82 (48.2)	–
Marginal overweight	25 (26.3)	34 (21.4)	37 (20.3)	35 (16.9)	56 (19.5)	–	26 (20.3)	30 (17.8)	23 (15.5)	17 (12.6)	24 (14.1)	–
Overweight	28 (29.5)	45 (28.3)	70 (38.5)	81 (39.1)	123 (42.9)	–	14 (10.9)	26 (15.4)	29 (19.6)	35 (25.9)	40 (23.5)	–
Obese	4 (4.2)	11 (6.9)	18 (9.9)	28 (13.5)	48 (16.7)	–	3 (2.3)	4 (2.4)	8 (5.4)	3 (2.2)	15 (8.8)	–

Data are presented as mean ± standard deviation or n (%). P-values for the trend of linearity; calculated by
[a] chi-square test and
[b] ANOVA.

statistical significance. There was a modest but significant correlation between post-dialysis fasting plasma glucose blood and baseline BMI ($r = 0.142$, $P < 0.001$) in nondiabetic patients, and the incidence of post-dialysis diabetes also increases with baseline BMI categories (Figure 2).

Change in body weight after 2 years

After 2 years, 297 patients died, 98 had kidney transplants, 88 were converted to hemodialysis and 33 were transferred to other centers. There were 954 patients who remained on PD in our center and the change in their body weight was explored. For this nested cohort, their average body weight increased from 61.7 ± 13.2 to 63.0 ± 13.3 kg (paired Student's *t*-test, $P < 0.001$), and BMI increased from 23.9 ± 4.3 to 24.4 ± 4.4 kg/m^2, $P < 0.001$. The change in BMI category over 2 years is summarized in Table 5. Specifically, 638 patients (66.9%) remained in the same category, 102 patients (10.7%) had down-grading of their BMI categories and 214 patients (22.4%) had up-grading in their BMI categories.

From 1995–99 to 2014–19, the mean BMI change after 2 years of PD gradually increased (0.01 ± 0.09, 0.01 ± 0.07, 0.02 ± 0.07, 0.03 ± 0.10 and 0.04 ± 0.09 kg/m^2 for each consecutive 5-year period respectively, $P < 0.001$ for linearity). The mean BMI change was slightly higher for diabetic than nondiabetic patients (0.03 ± 0.08 versus 0.02 ± 0.08 kg/m^2, $P = 0.027$).

DISCUSSION

In this study, we found that obesity is increasingly prevalent in new PD patients of Hong Kong over the past 25 years, and this phenomenon is present in both diabetic and nondiabetic patients. The incidence of new-onset diabetes was also significantly higher in new PD patients with pre-existing obesity or overweight than those without obesity.

The increasing prevalence of obesity in new PD patients that we observed is in line with that of the general population around the world [8–10]. In China, the prevalence of obesity rose from 6.5% in 1997 to 14.8% in 2011, and the prevalence of overweight rose from 29.1% to 41.0% during that period [11], which is slightly higher than that observed in our patients. Although the link between obesity and the risk of ESKD was well documented [12], it is probable that another group of patients with advanced CKD develop muscle wasting and protein-energy malnutrition [16, 17], which results in a lower prevalence of obesity as compared with the general population.

In our study, an increasing prevalence of overweight or obesity is present in both diabetic and nondiabetic new PD patients. Although the relative increase was more marked in the nondiabetic group (from 13.2% to 32.3%, i.e. relative increase by 144.7%) than the diabetic ones (from 33.7% to 59.6%, i.e. relative increase by 76.9%), the absolute magnitude of increase was more pronounced in the diabetic group (25.9% versus 19.1%). In other words, our observation suggests that obesity was increasingly more common among diabetic patients with advanced CKD. One possible reason is that a higher proportion of diabetes is caused by obesity and insulin resistance nowadays [30]. On the other hand, it is also possible that insulin therapy, which commonly causes weight gain, is increasingly being used for patients with advanced diabetic kidney disease because of the emphasis on tight diabetic control in the recent decade [31, 32].

Our finding that obesity is increasingly more frequent in new Chinese PD patients implies that a fixed PD regimen may result in inadequate dialysis in a considerable portion of

Table 3. The relation between baseline BMI and clinical and biochemical parameters

Variables	BMI		BMI category/obesity group	
	Spearman's r	P-value	P of one-way ANOVA	P for trend of linearity
Age	0.036	0.137	<0.001	0.014
Systolic blood pressure	0.137	<0.001	<0.001	<0.001
Diastolic blood pressure	−0.001	0.959	0.002	0.694
Charlson's comorbidity	0.161	<0.001	<0.001	<0.001
Serum CRP	0.007	0.832	0.235	0.803
Glucose profile				
Fasting blood glucose	0.239	<0.001	<0.001	<0.001
HbA1c	0.194	<0.001	0.015	0.001
Serum lipid profile				
Cholesterol	−0.206	<0.001	<0.001	<0.001
Triglyceride	0.067	0.063	0.393	0.083
LDL	−0.155	<0.001	<0.001	<0.001
HDL	−0.347	<0.001	<0.001	<0.001
Dialysis adequacy				
Total K_t/V	−0.150	<0.001	<0.001	<0.001
Residual GFR	0.229	<0.001	<0.001	<0.001
Nutrition status				
Serum albumin	0.045	0.067	0.095	0.052
Hemoglobin	−0.014	0.560	0.529	0.798
NPNA	0.350	<0.001	<0.001	<0.001
SGA	0.093	0.007	0.110	0.022
MIS	−0.133	<0.001	0.001	<0.001
FEBM (absolute)	0.021	0.380	0.409	0.679
FEBM (% of IBW)	−0.442	<0.001	<0.001	<0.001
Bioimpedence spectroscopy				
OH	0.260	<0.001	<0.001	<0.001
Lean total mass	0.304	<0.001	<0.001	<0.001
ATM	0.618	<0.001	<0.001	<0.001
Arterial PWV				
Carotid-to-radial	−0.124	0.001	0.008	0.001
Carotid-to-femoral	0.012	0.720	0.264	0.878

LDL, low-density lipoprotein; HDL, high-density lipoprotein; GFR, glomerular filtration rate.

patients. The consideration is particularly important in this part of the world, where a routine three 2-l exchange is used [33, 34]. Theoretically, obese patients require a higher volume of PD exchange to achieve adequate solute clearance, particularly when their residual renal function is gradually lost. In fact, this is probably the reason that the survival advantage of PD (as compared with hemodialysis) is lost in obese patients, and they have a higher risk than nonobese patients of being converted to hemodialysis [35].

In addition to studying the change in body weight, we tried to explore the change in nutritional status and body composition in our new PD patients over the years, as summarized in Table 4. Although the data appear complicated, the general trend was that some nutritional parameters but not others improved over time. Notably, the increase in skeletal muscle mass (as represented by FEBM by creatinine kinetics and LTM by bioimpedance study) was only modest, suggesting that the increase in body weight was largely accounted for by adipose tissue and probably fluid retention. It is also interesting to note that although there is evidence from Table 4 that the magnitude of fluid overload increased in the new PD patients over the years, the residual renal function actually increased during the same period (Table 1), arguing against the possibility of delayed initiation of dialysis.

In this study, the incidence of new-onset diabetes showed a modest but insignificant increasing trend in new PD patients over the years. Since new-onset diabetes is significantly more common in new PD patients with pre-existing obesity, the increasing prevalence of obese PD patients probably explains the trend of new-onset diabetes. Our observation is consistent with the previous reports [36, 37]. For example, Dong et al. [37] showed that a high BMI was a major contributing factor to new-onset diabetes and impaired glucose tolerance in PD patients. The absolute incidence of new-onset diabetes was much higher in this study than in previous reports [29, 36], which probably reflects the increase in the incidence in recent years. Although new-onset diabetes is well reported to be associated with a poor outcome in PD patients [29], it remains unknown whether the use of a glucose-free PD regimen would reduce the risk of new-onset diabetes or its long-term clinical outcome. Nonetheless, our result suggests that routine screening for new-onset impaired fasting glucose should be performed in incident PD patients, preferably 4–8 weeks after they are stabilized on dialysis.

There are several strengths but also inadequacies of this study. The period of review was substantial, and because of the PD-first policy in Hong Kong, our result basically represents the entire population of new ESKD patients in Hong Kong. With the

Table 4. Baseline biochemical and nutritional data over the study period

Year	All patients						Patients with overweight or obesity					
	1995–99	2000–04	2005–09	2010–14	2015–19	P-value	1995–99	2000–04	2005–09	2010–14	2015–19	P-value
No. of cases	223	329	330	342	457		49	86	125	147	226	<0.001[a]
Hemoglobin, g/dL	9.3 ± 1.5	9.1 ± 1.5	9.4 ± 4.4	9.4 ± 1.5	9.8 ± 1.5	0.084[a]	9.1 ± 1.7	8.9 ± 1.3	9.7 ± 6.8	9.4 ± 1.4	9.6 ± 1.5	0.172[b]
Albumin, g/L	27.9 ± 4.6	31.1 ± 5.6	34.2 ± 4.8	34.3 ± 4.9	29.1 ± 5.7	<0.001[b]	28.4 ± 4.2	31.1 ± 4.9	34.3 ± 4.5	34.9 ± 4.7	29.5 ± 5.5	0.235[b]
SGA	5.2 ± 0.9	5.4 ± 1.0	5.7 ± 1.0	5.2 ± 0.9	5.4 ± 0.8	0.104[b]	5.8 ± 0.5	5.2 ± 1.0	5.8 ± 1.0	5.4 ± 0.8	5.5 ± 0.8	0.235[b]
MIS	9.4 ± 2.7	7.4 ± 3.4	6.3 ± 3.8	7.0 ± 3.6	5.5 ± 3.2	0.211[b]	10.0 ± 2.8	7.1 ± 3.1	6.1 ± 3.3	6.2 ± 3.1	6.0 ± 3.3	0.414[b]
NPNA, g/kg/day	0.9 ± 0.2	1.1 ± 0.3	1.2 ± 0.3	1.1 ± 0.2	1.2 ± 0.3	0.001[b]	1.1 ± 0.3	1.2 ± 0.3	1.3 ± 0.2	1.2 ± 0.3	1.3 ± 0.3	0.125[b]
FEBM												
Absolute, kg	25.5 ± 7.0	26.8 ± 8.0	26.4 ± 8.1	27.1 ± 9.7	27.2 ± 9.1	0.025[b]	25.3 ± 6.7	26.7 ± 8.6	27.3 ± 8.4	26.5 ± 7.4	27.4 ± 9.5	0.004[b]
% of IBW	48.8 ± 12.0	42.3 ± 10.3	40.3 ± 10.0	41.1 ± 12.0	38.0 ± 10.6	<0.001[b]	43.4 ± 10.7	37.1 ± 10.9	35.8 ± 8.3	36.5 ± 8.5	35.1 ± 9.9	0.226[b]
Serum lipid, mmol/L												
Total cholesterol	5.2 ± 1.6	5.2 ± 1.2	5.0 ± 1.3	4.9 ± 1.3	4.7 ± 1.7	0.001[b]	3.8 ± 0.3	4.7 ± 0.9	4.7 ± 1.4	4.7 ± 1.4	4.3 ± 1.2	<0.001[b]
Triglyceride	2.0 ± 2.1	2.0 ± 1.4	1.9 ± 1.2	1.8 ± 1.3	1.6 ± 1.4	0.012[b]	1.1 ± 0.5	2.2 ± 1.5	2.0 ± 1.4	1.9 ± 1.4	1.6 ± 1.0	0.337[b]
LDL	3.0 ± 1.1	3.0 ± 1.0	2.9 ± 1.0	2.8 ± 1.1	2.6 ± 1.2	0.058[b]	2.2 ± 0.1	2.7 ± 1.0	2.7 ± 1.0	2.7 ± 1.1	2.6 ± 1.4	0.143[b]
HDL	1.5 ± 0.5	1.4 ± 0.6	1.4 ± 0.9	1.3 ± 0.6	1.4 ± 0.9	0.278[b]	1.2 ± 0.5	1.2 ± 0.3	1.2 ± 0.4	1.3 ± 0.8	1.4 ± 1.3	0.909[b]
Bio-impedance						–						0.248[b]
OH, L	–	–	3.4 ± 2.3	3.9 ± 3.1	4.9 ± 3.9	<0.001[b]	–	–	3.9 ± 2.6	4.4 ± 3.4	5.7 ± 4.1	0.001[b]
ATM, kg	–	–	21.3 ± 9.0	20.3 ± 11.0	21.7 ± 11.4	0.169[b]	–	–	24.7 ± 8.8	26.9 ± 11.6	27.0 ± 12.3	0.688[b]
LTM, kg	–	–	38.8 ± 10.1	38.9 ± 10.8	39.2 ± 10.1	0.657[b]	–	–	36.3 ± 7.0	40.8 ± 11.5	42.3 ± 10.8	0.065[b]
PWV, cm/s						–						–
Carotid-to-radial	–	–	10.1 ± 1.5	10.6 ± 1.7	10.2 ± 1.7	0.908[b]	–	–	10.1 ± 1.6	10.5 ± 1.6	10.0 ± 1.7	0.149[b]
Carotid-to-femoral	–	–	10.3 ± 2.3	11.4 ± 2.5	11.1 ± 1.9	0.017[b]	–	–	10.8 ± 2.4	11.5 ± 2.4	10.9 ± 1.9	0.472[b]

Data are presented as mean ± standard deviation. P-values for the trend of linearity; calculated by [a]chi-square test and [b]ANOVA; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

Table 5. Change in the BMI category after 24 months

24 months	Baseline				
	Underweight, n (%)	Normal weight, n (%)	Marginal overweight, n (%)	Overweight, n (%)	Obesity, n (%)
Underweight	38 (4.0)	17 (1.8)	0	1 (0.1)	0
Normal weight	28 (2.9)	275 (28.8)	37 (3.9)	9 (0.9)	1 (0.1)
Marginal overweight	1 (0.1)	64 (6.7)	80 (8.4)	27 (2.8)	1 (0.1)
Overweight	0	20 (2.1)	67 (7.0)	194 (20.3)	9 (0.9)
Obesity	0	0	2 (0.2)	32 (3.4)	51 (5.3)

McNemar–Bowker test, $P < 0.001$.
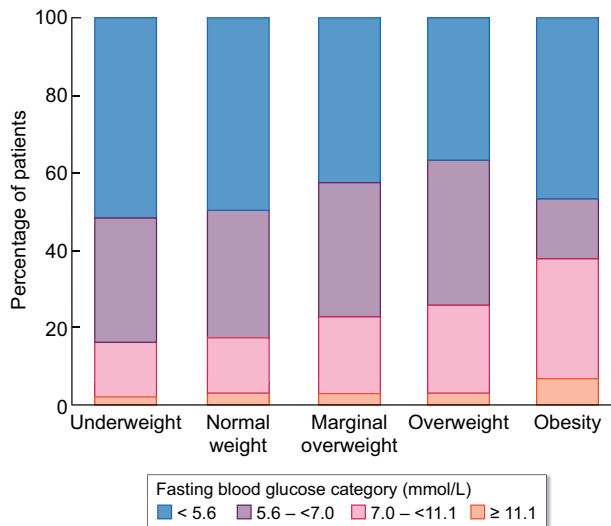
FIGURE 2: The distribution of post-dialysis fasting plasma glucose categories in incident nondiabetic PD patients according to their baseline BMI categories.

well-established hospital computer database system, the data on anthropometric measures and biochemistry were extensive and missing data were minimized. Unfortunately, because of the limitations in our database, we only report BMI at baseline and after 2 years of PD. In theory, it would be interesting to analyze the rate of body weight gain and probably the fluctuation of body weight within 2 years. Furthermore, body weight or BMI are crude measures of obesity. Recent evidence suggests that subcutaneous and visceral adipose tissue may have different physiological implications [17, 38, 39], and fluid retention may confound the measurement of body weight. Notably, data on body composition (multi-frequency bio-impedance) and arterial PWV were not available before 2005 and it was, at least theoretically, possible that the increase in body weight of new PD patients over the years was the result of more severe fluid retention.

Our results lead to several questions that require further study. As discussed above, what we actually showed was a progressive increase in body weight and BMI in new PD patients. The change in BMI after 2 years is primarily driven by the change in body weight, but the exact changes in body composition and ATM have not been evaluated. Although the prognostic impact of obesity in PD patients has been examined by multiple groups [17, 40], the prevalence and impact of body weight change after started on PD have not been extensively studied.

The complex interactions between body fat mass, fluid accumulation and cardiovascular disease in PD patients also deserve further study.

CONCLUSION

The incidence of obesity increased substantially in both diabetic and nondiabetic new PD patients in Hong Kong over the past 25 years. The incidence of new-onset diabetes was significantly higher in new PD patients with pre-existing obesity or overweight than those without obesity. Their prognostic implications and impacts on the healthcare system deserve further studies.

SUPPLEMENTARY DATA

Supplementary data are available at ckj online.

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