Association of anthropometric obesity measures with chronic kidney disease risk in a non-diabetic patient population

James O. Burton¹, Laura J. Gray², David R. Webb³, Melanie J. Davies⁴, Kamlesh Khunti², Winston Crasto³, Sue J. Carr¹ and Nigel J. Brunskill¹

¹Department of Infection, Immunity and Inflammation, University of Leicester, Leicester, UK, ²Department of Health Sciences, University of Leicester, Leicester, UK and ³Department of Cardiovascular Sciences, University of Leicester, Leicester, UK

Correspondence and offprint requests to: James O. Burton; E-mail: jb343@le.ac.uk

Abstract

Background. Obesity is a risk factor for both chronic kidney disease (CKD) and cardiovascular disease. The association of simple indices of obesity with CKD remains poorly understood. Evidence suggests that measures of central obesity such as waist circumference (WC) and waist-to-hip ratio (WHR) are more accurate predictors of morbidity and cardiovascular risk than body mass index (BMI). This study aimed to investigate the association of BMI, WC and WHR with CKD risk in a population screened for type 2 diabetes.

Methods. Data were drawn from a population-based screening programme of 6475 volunteers without pre-existing diabetes. A number of investigations and cardiovascular health-related assessments were performed. Participants were categorized into two groups: those with an estimated glomerular filtration rate (eGFR) ≥60 and <60 mL/min/1.73m². Participants were also categorized as low, medium and high risk according to each anthropometric variable.

Results. CKD was independently associated with higher WC and BMI (P < 0.01) but not WHR (P = 0.47). Increasing obesity measured by BMI and WC was associated with a reduction in eGFR for both men and women (P < 0.001). Increasing risk categories for BMI and WC were also associated with lower eGFR in men and women (P < 0.001). Combining anthropometric measures provided no additional measure of risk for underlying CKD.

Conclusions. WC may be a simple and reliable clinical tool for the detection of underlying CKD within primary care. Given the complex interaction between adiposity and uraemia, a combined screening tool using BMI and WC or WHR is unlikely to provide any additional benefit to risk analysis.

Keywords: anthropometrics; body mass index; waist circumference; waist-to-hip ratio; chronic kidney disease

Introduction

Obesity is an escalating global health concern and costs the UK National Health Service (NHS) an estimated £4.2 billion/year [1]. Conservative estimates which extrapolate pre-2003 population trends suggest that in England alone 12 million adults and 1 million children are now clinically obese. This equates to approximately one-third of all adults and one-fifth of all children aged 2–15 years [2].

Obesity precedes the onset of many risk factors for cardiovascular disease (CVD) and cardiovascular mortality such as diabetes, hypertension and dyslipidaemia and shortens life expectancy by an average of 9 years [3–7]. In addition, biological processes have been identified as potential mechanisms leading directly from obesity to renal damage including hormonal factors, inflammation, oxidative stress and endothelial dysfunction [8, 9].

Chronic kidney disease (CKD) is now recognized as an independent risk factor for myocardial infarction and cardiovascular mortality [10, 11]. Studies estimate the adult prevalence of CKD at ~13% and growing [12], partly due to the increase in diabetes, hypertension and increased body mass index (BMI). Obesity is a risk factor for the development of CKD [8, 13] and is then subsequently associated with poor cardiovascular outcome [14].

The best measure of obesity in CKD patients remains controversial. Studies using BMI to evaluate obesity and adverse outcomes in CKD have conflicting results. This confusion may be in part due to the relative role of muscle, fat and bone mass in determining BMI. For instance, in individuals with established CKD, lower BMI may reflect either decreased visceral fat (lower cardiovascular risk) or decreased muscle mass (higher cardiovascular risk).

It is generally recognized that the central deposition of fat (abdominal or visceral obesity) is closely associated with chronic diseases and is a key constituent of the metabolic syndrome [15, 16]. Anthropometric measures of central adiposity other than BMI such as waist circumference (WC) and waist-to-hip ratio (WHR) have been shown in the general population to better predict risk of diabetes, hypertension and CVD [4, 17, 18]. Accordingly, the UK ‘National Institute of Clinical Excellence’ (NICE) now recommends a combined disease risk assessment tool in primary care using BMI and WC for the prediction of
diabetes, coronary heart disease and some cancers [19]. However, the merits of such a tool remain unproven in CKD prediction.

Given this complex interrelationship between obesity, CKD and cardiovascular risk, it is crucial to find robust methods of detecting people in the general population who are most vulnerable. The aim of this study was to evaluate the usefulness of three different anthropometric measurements (BMI, WC and WHR) as potential predictors of CKD in a population-based cohort derived from a major screening programme for type 2 diabetes.

Materials and methods

The aims were tested using screening data from the Leicester arm of the multicentre Anglo-Danish-Dutch study of Intensive Treatment In peOple with screen-detected diabetes in primary care (ADDITION) database. ADDITION-Leicester is a pragmatic randomized controlled trial of multifactorial cardiovascular risk intervention in individuals with screen-detected type 2 diabetes [20].

Patient selection and recruitment

Briefly, 66 320 patients aged between 40 and 75 years old inclusive (25–75 for South Asians) were eligible for inclusion from 20 primary care facilities in Leicestershire, UK and a random selection of 30 950 patients were invited to take part. Once identified, patients were sent individual screening appointments at either a hospital site or a mobile screening unit, located within their community. At the screening visit, informed written consent was obtained. Of the 6749 who were recruited, 6475 had measures of estimated glomerular filtration rate (eGFR) and were included in this analysis.

Patient demographics including age, gender, occupation and ethnicity as well as smoking status, self-reported medications and past medical (including cardiovascular) history were obtained using questionnaires. These data were then collated and verified by a member of the research team during screening at face-to-face interview. The estimated absolute 10-year risk of CVD was calculated using the previously published ‘Ethrisk’ equation, an ethnicity-adjusted Framingham risk score [21]. The risk was only estimated for those participants with complete data on all the composite risk factors required for the calculation.

Biochemical assessment

All samples collected were from fasting subjects and were analysed at a single centre (University Hospitals of Leicester NHS Trust). eGFR was calculated using the four-variable Modification of Diet in Renal Disease Study equation [22, 23]. Urinary albumin was measured on spot urine by the immunoturbidometric method and urinary creatinine by the colorimetric method to gain a urinary albumin to creatinine ratio. Cholesterol, low-density lipoprotein (LDL) cholesterol and triacylglycerol levels were measured using standard enzymatic methods and LDL cholesterol was calculated using the Friedewald formula [24]. HbA1c was measured using the Biord Varisat II system (Bi-Rad Laboratories, Henel Hempstead, UK).

Anthropometric measurements and definitions

These were undertaken by trained staff following standardized procedures. Height was measured to the nearest 0.1 cm and weight (in light indoor clothing) measured to the nearest 0.1 kg. BMI (kg/m2) was defined as weight in kilograms divided by height in metres squared. WC was measured at the midpoint between the lower costal margin and the level of the anterior superior iliac crest to the nearest 0.1 cm. WHR is the ratio of WC to hip size which was defined as the greatest measurement from the anterior superior iliac crest to the top of the thigh.

Anthropometric measurements were categorized as follows: BMI was expressed as normal (<25 kg/m²), overweight (25 < 30 kg/m²) and obese (>30 kg/m²) in both men and women; WC as low (<94 cm), high (94–102 cm) and very high (>102 cm) in men and low (<80 cm), high (80–88 cm) and very high (>88 cm) in women [19] and WHR as low risk (<0.89), moderate risk (0.96–1) and high risk (>1) in men and low risk (<0.8), moderate risk (0.81–0.85) and high risk (>0.85) in women [25].

Statistical analysis

For statistical analysis, patients were first divided into two groups according to eGFR, those ≥60 and those <60 mL/min/1.73m² (as this value equates to CKD Stages 3–5). In addition, patients were categorized as low, medium and high risk according to each anthropometric variable (BMI, WC and WHR) using the values stated above.

Results are presented as the mean ± SD or the median and interquartile range unless otherwise stated. Categorical variables between the two groups were analysed using chi-squared test. One-way and two-way analysis of variance (ANOVA) was used to test the relationship across anthropometric variables between single and multiple groups, respectively. Data were analysed using Stata (version 11) and P < 0.05 reflects statistical significance.

Results

The prevalence of CKD defined as an eGFR <60 mL/min/1.73m² and equating to CKD Stages 3–5 in ADDITION-Leicester was 10.4% (n = 676) and is similar to other epidemiological studies [26]. The distribution of eGFR is shown in Figure 1.

Results according to eGFR category

Patient demographics including clinical, biochemical and anthropometric data are shown in Table 1. The traditional risk factors of CVD (male sex, increasing age, hypertension, hypercholesterolaemia, smoking status, elevated HbA1c and proteinuria as measured by urinary albumin to creatinine ratio) as well as an elevated 10-year Ethrisk CVD score were all associated with an eGFR <60 mL/min/1.73m² (P < 0.0001).

With respect to anthropometric data, univariate analysis revealed that the patients with CKD defined as an eGFR <60 mL/min/1.73m² were not statistically heavier (P = 0.32) but that these patients had a significantly higher BMI (P < 0.0001) and WC (P = 0.05). There was a negative association with WHR which was actually less in the lower eGFR group (P < 0.01).

Logistic regression confirmed that eGFR <60 mL/min/1.73m² was associated with a higher BMI [odds ratio (OR) 1.04, 95% confidence interval (CI) 1.02–1.05, P < 0.0001] and WC (OR 1.01, 95% CI 1.00–1.01, P = 0.04) but a
lower phenotypically more healthy WHR (OR 0.86, 95% CI 0.78–0.95, P < 0.01). However, after adjustment for: age, gender, ethnicity, albuminuria, CVD including BP and fasting glucose; logistic regression analysis confirmed that the lower eGFR was independently associated with increasing BMI (OR 1.04, 95% CI 1.02–1.06; P < 0.0001) and with a higher WC (OR 1.01, 95% CI 1.01–1.02; P < 0.002) but was no longer significantly associated with WHR (OR 1.05, 95% CI 0.92–1.21; P = 0.47).

Similarly, when taking eGFR as a continuous rather than a categorical variable, regression analysis adjusted for those variables above showed that a declining eGFR was associated with both an increasing BMI ($r^2 = 0.2$, $F(7, 5742) = 206.8$; $P < 0.0001$) and WC ($r^2 = 0.2$, $F(7, 5741) = 206.2$; $P < 0.001$) but not WHR ($r^2 = 0.2$, $F(7, INS > 5737) = 204.2$; $P = 0.62$).

The sensitivity analyses for various cut points of WC are given in Table 2 with eGFR cut-off values corresponding to accepted CKD classifications. Screening people with a WC of 95 cm (45% of the population) gives a sensitivity of 52.1% for an eGFR of <60 mL/min/1.73m², 68.1% for <45 mL/min/1.73m² and 80% for <30 mL/min/1.73m². For each level of eGFR, the specificity of a cut point of 95 cm is ~50% meaning that 50% are correctly identified as not having CKD.

Results according to anthropometric variable

Using ANOVA, increasing tertiles of obesity measured by BMI and WC was associated with a statistically significant reduction in eGFR for both men and women ($P < 0.001$). A higher category WHR was also associated with a lower eGFR in men ($P < 0.001$) but did not reach statistical significance in women ($P = 0.12$) (Table 3).

When analysing anthropometric measures as continuous variables using adjusted regression analysis, an increasing BMI was associated with a lower eGFR ($r^2 = 0.05$, $F(7, 5742) = 41.04$; $P < 0.0001$), as was an increasing WC ($r^2 = 0.2$, $F(7, 5741) = 174.9$; $P < 0.001$) but not WHR ($r^2 = 0.4$, $F(7, 5737) = 602.9$; $P = 0.62$).

Relationship between eGFR, BMI and central obesity measures

The interrelationship between eGFR, BMI and WC was tested using a two-way ANOVA and is shown in Figure 2a. Although an increasing WC was associated with a significant reduction in eGFR across BMI categories ($P < 0.0001$), the overall interaction between BMI and WC was not statistically significant ($P = 0.76$).

The interrelationship between eGFR, BMI and WHR was also tested and is shown in Figure 2b. Similarly, an increasing WHR was associated with a significantly lower eGFR across all BMI categories ($P < 0.0001$). However, the overall interaction between BMI and WHR was not statistically significant ($P = 0.65$).

Discussion

In this study of a randomly selected population screened for the presence of diabetes, CKD as defined by an eGFR of <60 mL/min/1.73m² was directly associated with both an increased BMI and a larger WC but not an increased WHR. Additionally, increasing obesity measured by BMI and WC was associated with a reduction in eGFR for both men and women but WHR in men alone. Given current epidemic rates of obesity and increasing incidence and prevalence of CKD leading to established renal failure in the general population, identification of simple clinical measures that could be used as screening tools to identify individuals most likely to have CKD is crucial.

The relationship between obesity and CKD is complex and the effects on outcome are controversial. Adipose

---

Table 1. Patient demographics for the entire cohort and split into two groups according to eGFR*

<table>
<thead>
<tr>
<th></th>
<th>All (N = 6475)</th>
<th>eGFR ≥60 (n = 5799)</th>
<th>eGFR &lt;60 (n = 676)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>55.9 (10.8)</td>
<td>55.0 (10.7)</td>
<td>63.9 (8.4)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Sex, male (%)</td>
<td>3079 (47.6)</td>
<td>2865 (49.4)</td>
<td>214 (31.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>78.0 (16.1)</td>
<td>77.9 (16.1)</td>
<td>78.6 (15.6)</td>
<td>0.32</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.1 (5.0)</td>
<td>28.0 (5.0)</td>
<td>29.0 (5.1)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>94.0 (13.2)</td>
<td>93.8 (13.1)</td>
<td>94.9 (13.2)</td>
<td>0.04</td>
</tr>
<tr>
<td>WHR</td>
<td>0.89 (0.08)</td>
<td>0.89 (0.08)</td>
<td>0.88 (0.08)</td>
<td>0.003</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>137.0 (19.6)</td>
<td>136.6 (19.5)</td>
<td>140.9 (20.4)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>85.4 (10.6)</td>
<td>85.4 (10.5)</td>
<td>85.4 (11.2)</td>
<td>0.93</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non</td>
<td>3739 (58.2)</td>
<td>3355 (58.2)</td>
<td>384 (57.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Ex</td>
<td>1745 (27.2)</td>
<td>1519 (26.4)</td>
<td>226 (34.0)</td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>944 (14.7)</td>
<td>889 (15.4)</td>
<td>55 (8.3)</td>
<td></td>
</tr>
<tr>
<td>Creatinine (µmol/L)</td>
<td>86.1 (16.1)</td>
<td>83.6 (12.8)</td>
<td>107.1 (24.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>5.7 (0.6)</td>
<td>5.7 (0.6)</td>
<td>5.8 (0.5)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>5.5 (1.1)</td>
<td>5.5 (1.1)</td>
<td>5.7 (1.2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LDL (mmol/L)</td>
<td>3.5 (0.9)</td>
<td>3.5 (0.9)</td>
<td>3.6 (1.0)</td>
<td>0.01</td>
</tr>
<tr>
<td>HDL (mmol/L)</td>
<td>1.36 (0.4)</td>
<td>1.35 (0.4)</td>
<td>1.42 (0.4)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ACR [median (IQR)]</td>
<td>0.7 (0.5–1.1)</td>
<td>0.7 (0.5–1.1)</td>
<td>0.8 (0.5–1.5)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Ethrisk 10-year CVD risk (%)</td>
<td>14.1 (11.3)</td>
<td>13.9 (11.2)</td>
<td>16.4 (12.0)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*Statistical relationship between these two groups is shown. Data are expressed as either mean (SD); median (IQR) for non-normally distributed data or count (percentage). BP, blood pressure; ACR, albumin:creatinine ratio.
tissue is established as a hormonally active organ that releases large numbers of bioactive adipokine proteins. These adipokines target numerous tissues and organs and regulate not only energy homeostasis and body weight but also insulin resistance, plasma lipids, endothelial health, coagulation and inflammation [27]. While the multisystem effects of adipose tissue are still not completely understood, there is good evidence to suggest that obesity has a direct impact on renal function [8, 28]. However, it also seems likely that reduced renal clearance contributes further to the accumulation of adipokines that may offer a novel pathway to explain the marked prevalence of disordered metabolism seen in uraemic patients [27]. This again underscores the importance of identifying those who are at risk of both end-organ damage and the subsequent accumulation of circulating humoral factors that may contribute to the further progression of CKD.

Most of the current literature uses BMI to classify obesity and identify its associations with morbidity and mortality as BMI correlates with body fat in most individuals. Indeed, our results show that CKD is associated with increased BMI and that it remains a useful clinical tool in this cohort of individuals. There is also some evidence to show that it is easier and more reliable to measure in primary care than other anthropometric variables that can have significant operator variability [29].

However, BMI assesses entire body mass without differentiating between the different components of subcutaneous and visceral fat, muscle and bone. As a result, it is possible to achieve a clinically significant reduction in WC and improvement of metabolic profile using diet and exercise but without observing any weight loss or change in BMI [30]. Studies have suggested that visceral adiposity assessed using computerized tomography (CT) scanning remains more strongly associated with an adverse metabolic risk profile and may be a better predictor of CVD than standard anthropometric measurements including BMI [31, 32]. However, such scanning methods are impractical on a large scale, which highlights the importance of identifying a simple and inexpensive surrogate marker of fat distribution.

Although anthropometric measures like WC and WHR offer better assessment of central adiposity than BMI, they still do not allow for accurate differentiation between subcutaneous and visceral fat deposition. WHR is thought to correlate more strongly with deposition of visceral rather than subcutaneous fat (which correlates better with BMI) [33] and general population studies have shown that WHR is a more powerful predictor than BMI of underlying pathologies such as CVD. One example of a recent prospective study of >25 000 participants in the UK demonstrated that WHR was independently and more consistently predictive of coronary heart disease than either BMI or WC [34]. Similar evidence exists looking at the relationship with WHR and kidney function [35]. So why do the results of this study seem to suggest that WHR is not as predictive of underlying CKD within this cohort as WC or BMI? Firstly, it is worth noting that in each of the above studies, WC was still independently associated with the risk of coronary heart disease but just less consistently than WHR. Similarly, in addition to demonstrating that waist and hip circumferences measure different aspects of body composition and fat distribution, the study of Seidell et al. [33] also showed that they have independent and often

### Table 2. Sensitivity analyses for various cut points of WC with eGFR values corresponding to accepted CKD classificationsa

<table>
<thead>
<tr>
<th>WC value (cm)</th>
<th>% Screened</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>80</td>
<td>85</td>
<td>86.5 (83.7–89.0)</td>
<td>13.0 (12.2–13.9)</td>
<td>89.4 (76.9–96.5)</td>
<td>13.1 (12.3–13.9)</td>
<td>80.0 (28.4–99.5)</td>
<td>13.1 (12.2–13.9)</td>
</tr>
<tr>
<td>85</td>
<td>76</td>
<td>76.8 (73.4–79.9)</td>
<td>24.4 (23.3–25.5)</td>
<td>85.1 (71.7–93.8)</td>
<td>24.3 (23.3–25.4)</td>
<td>80.0 (28.4–99.5)</td>
<td>24.3 (23.2–25.3)</td>
</tr>
<tr>
<td>90</td>
<td>62</td>
<td>65.8 (62.1–69.4)</td>
<td>37.9 (36.7–39.2)</td>
<td>76.6 (62.0–87.7)</td>
<td>37.6 (36.4–38.8)</td>
<td>80.0 (28.4–99.5)</td>
<td>37.5 (36.3–38.7)</td>
</tr>
<tr>
<td>95</td>
<td>47</td>
<td>52.1 (48.2–55.9)</td>
<td>53.4 (52.1–54.6)</td>
<td>68.1 (52.9–80.9)</td>
<td>52.9 (51.7–54.2)</td>
<td>80.0 (28.4–99.5)</td>
<td>52.8 (61.6–54.0)</td>
</tr>
<tr>
<td>100</td>
<td>32</td>
<td>35.5 (31.9–39.2)</td>
<td>68.4 (67.2–69.6)</td>
<td>42.6 (28.3–57.8)</td>
<td>68.1 (66.9–69.2)</td>
<td>60.0 (14.7–94.7)</td>
<td>68.0 (66.9–69.1)</td>
</tr>
<tr>
<td>105</td>
<td>19</td>
<td>23.1 (20.0–26.4)</td>
<td>80.7 (79.6–81.7)</td>
<td>14.9 (6.2–28.3)</td>
<td>80.2 (79.2–81.2)</td>
<td>40.0 (5.3–85.3)</td>
<td>80.3 (79.3–81.3)</td>
</tr>
</tbody>
</table>

aNumber screened represents the percentage of subjects with a WC equal to or greater than the given cut point.

### Table 3. Differences in eGFR according to anthropometric categories of obesitya

<table>
<thead>
<tr>
<th>eGFR (men)</th>
<th>Low risk WC</th>
<th>High risk WC</th>
<th>Very high risk WC</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>74.7 (12.9)</td>
<td>77.0 (12.9)</td>
<td>73.1 (11.5)</td>
<td>72.5 (13.1)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>79.3 (13.6)</td>
<td>77.1 (13.0)</td>
<td>73.0 (12.5)</td>
<td>72.8 (12.9)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>73.8 (12.6)</td>
<td>75.1 (12.9)</td>
<td>73.0 (12.5)</td>
<td>74.5 (13.7)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

aP-values refer to statistical relationship between increasing obesity measures and eGFR using ANOVA. Data are expressed as mean (SD).
opposite effects on CVD risk factors. The investigators went on to suggest that the specific effects of each measure are poorly captured in the WHR. In contrast to this, WC alone correlated highly with both visceral and subcutaneous fat.

Secondly, it is important to try and understand the interrelationship between the fat distribution and the uraemic milieu. It is unclear whether there is a differential association between abdominal fat compartments and renal function due to the inability of the WHR to detect
visceral as compared with subcutaneous fat. Given the body of literature linking metabolic risk factors with both visceral fat and CKD, it is intuitive to believe that individuals with more visceral abdominal obesity would be more likely to have CKD. However, that is not the case; CT studies designed to investigate the association between central adipose distribution and CKD showed no differentiation between visceral versus subcutaneous fat deposition [36] and renal impairment.

This is entirely in keeping with our results. For this cohort of relatively young patients, it is reasonable to expect that BMI will represent an unhealthy phenotype and therefore be associated with CKD. Similarly, it is not surprising that CKD was independently associated with a higher WC, given that this represents a more accurate measure of central adiposity. The fact that CKD did not significantly correlate with WHR may represent either the inability of this particular measure to accurately predict risk in all models; the fact that the uraemic state alters the traditional models for disease risk in the general population or indeed a combination of both. In addition, studies that have shown a positive association with WHR (but not other measures) and CKD did not adjust their models for albuminuria as those data were not available [28]. This cohort represents people without pre-existing diabetes and is corrected for albuminuria meaning that this may be more representative of the true independent effects of different anthropometric measures on CKD risk.

This relationship is also further evidenced when separating obesity measures into tertiles of increasing risk; the higher the categorical risk the lower the eGFR. This was statistically significant for BMI and WC in men and women but WHR in men alone and clinically more significant for WC than BMI. This is completely in keeping with published evidence of prospective data looking at the relationship of anthropometric measures and the development of incident CKD where WC, irrespective of general adiposity, was a more important determinant of risk than WHR and BMI [37]. With respect to gender differences, redistribution of fat occurs in different ways between men and women as they get older. In women, WHR increases with age which mostly reflects a reduction in fat deposits in the hips making this variable more difficult to interpret across a wider age range [38]. This may explain why we observed no significant association between WHR and eGFR in our general population cohort.

Current clinical guidelines promote using WC (both independently and in combination with BMI) in preference to WHR for risk assessment and management in clinical practice of disorders, such as coronary heart disease, type 2 diabetes, osteoarthritis and some cancers [19]. Our results suggest that WC may also be a superior tool to WHR in the assessment of CKD risk. Despite showing an association between reduced eGFR and increasing WC irrespective of BMI category, any further stratification of risk using combined tools of BMI and either WC or WHR failed to demonstrate a consistent association with eGFR. This may well represent the unique relationship between the uraemic milieu and associated disease risk. Indeed, evidence and traditional risk models extrapolated from the general population (like Framingham) completely fail to explain the increased prevalence of CVD in patients with CKD. In spite of this, the authors believe that regardless of an individual’s BMI, an increased WC could be a potential clinical marker of underlying CKD risk that is easily collected in the primary care setting.

The principal limitation of this study is its cross-sectional design and lack of longitudinal data so causality cannot be inferred. However, patients with CKD were more obese and increasing obesity was associated with declining eGFR so whether obesity is causative or not it may represent a potential clinical measure to assess risk of underlying renal disease. There is evidence that adiposity can differ for a given BMI or WC between ethnic groups [39]. As a result, some of our South Asian population may have been categorized as lower than their actual risk, however, the validity of current anthropometric cut points in different ethnic populations in this setting was outside the scope of this study. Despite the relatively large number of study participants (6475), individual groups used for analysis were still small. Similarly, although changes in eGFR were statistically significant, in absolute terms, they are still slight but comparable to other published data and we believe to be clinically relevant.

In conclusion, both BMI and WC but not WHR were independently associated with a lower eGFR in a randomly screened population without pre-existing diabetes. WC may therefore be a simple and reliable clinical tool for the detection of underlying covert CKD within primary care. Given the complex interaction between adiposity and uraemia, a combined screening tool using BMI and WC or WHR is unlikely to provide any additional benefit.

Acknowledgements. We would like to thank all the Leicester contributors to the ADDITION database, which was supported by the Department of Health and an unrestricted grant from Servier UK.

Conflict of interest statement. None declared.

References

1866 J.O. Burton


