Original article

Urinary albumin excretion in latent autoimmune diabetes in adults (LADA) is more similar to type 2 than type 1 diabetes: Results of the Nord-Trøndelag Health Study 1995–1997

M.A. Radtke a,∗, T.I. Lund Nilsen c, K. Midthjell d, V. Grill a,b

a Department of Cancer Research and Molecular Medicine, Faculty of Medicine, Norwegian University of Science and Technology, NTNU, Olav Kyrres gate 17, 7006 Trondheim, Norway
b Saint Olav University Hospital, Olav Kyrres gate 17, 7006 Trondheim, Norway
c Human Movement Science Programme; Faculty of Social Science and Technology Management, Norwegian University of Science and Technology, 7006 Trondheim, Norway
d Department of Public Health, Faculty of Medicine, Norwegian University of Science and Technology, 7006 Trondheim, Norway

Received 7 July 2008; received in revised form 30 November 2008; accepted 2 December 2008
Available online 6 May 2009

Abstract

Aim. – As it is unclear, whether or not, urinary albumin excretion (UAE) differs between patients classified as latent autoimmune diabetes in adults (LADA) and other forms of diabetes, our study aimed to investigate the distribution of the albumin-to-creatinine ratio (ACR) in LADA compared with those in the “classical” types 1 (T1D) and 2 (T2D) diabetes.

Methods. – We used data from the Nord-Trøndelag Health Study (HUNT) (n = 64,931) of 1995–1997. ACR (mg/mmol) was measured in three urine samples from all diabetic patients (n = 1525) and from 5% of the non-diabetic study population (n = 2104). We calculated the geometric means and 95% confidence intervals (CI) using a general linear model.

Results. – The unadjusted mean ACR in LADA was similar to that in T2D (1.45, CI: 1.23–1.71 vs 1.41, CI: 1.33–1.49, respectively) but was significantly higher than those in T1D (0.99, CI: 0.83–1.19; P = 0.002) and non-diabetics (0.72, CI: 0.69–0.74; P < 0.001). These results remained similar even after multiple adjustments.

Conclusion. – In this cross-sectional study, the ACR in LADA and in T2D were similar and higher than in T1D. This similarity between LADA and T2D makes it unlikely that the autoimmune processes that operate in LADA promote albuminuria.

© 2009 Elsevier Masson SAS. All rights reserved.

Keywords: Autoimmunity; Latent autoimmune diabetes in adults; LADA; Diabetes epidemiology; Diabetic nephropathy; Albuminuria; Microalbuminuria

Résumé


On ne sait pas si l’élimination urinaire d’albumine (EUA) est différente chez les patients atteints d’un diabète auto-immun latent de l’adulte de celle des patients atteints d’autres formes de diabète.

Objectif. – Étude de la distribution du rapport albumine/créatinine chez des patients atteints de LADA, comparés à des patients atteints de diabète de type 1 (DT1) et de type 2 (DT2) « classiques ».

Méthodes. – Nous avons utilisé les données de la Nord-Trøndelag Health Study (n = 64931), réalisée entre 1995 et 1997. Le rapport albumine–créatinine (mg/mmol) a été mesuré dans trois prélèvements d’urine de tous les diabétiques (n = 1525) et de 5 % de la population non diabétique de l’étude. Nous avons calculé la moyenne géométrique et l’intervalle de confiance à 95 % (IC) avec un modèle linéaire général.

Résultats. – Non corrigée, la moyenne du rapport albumine–créatinine des sujets atteints de LADA était similaire à celle des DT2 (1.45 ; IC 1.23–1.71, vs 1.41 ; IC 1.33–1.49) mais significativement supérieure à celle des DT1 (0.99 ; IC 0.83–1.19, P = 0.002) et à celle des non diabétiques (0.72 ; IC 0.69–0.74, P = < 0.001). Ces résultats n’étaient pas modifiés après correction pour de multiples paramètres.

* Corresponding author.
E-mail address: maria.radtke@ntnu.no (M.A. Radtke).

1262-3636/$ – see front matter © 2009 Elsevier Masson SAS. All rights reserved.

© 2018 Elsevier Masson SAS. Tous droits réservés. - Document téléchargé le 31/12/2018 Il est interdit et illégal de diffuser ce document.
Conclusion. – Dans cette étude transversale, le rapport albumine–créatinine des patients atteints de LADA était proche de celui des DT2 mais supérieur à celui des DT1. Cette similitude de résultats entre LADA et DT2 rend peu vraisemblable la responsabilité du processus auto-immun à l’origine du LADA dans le développement de l’atteinte rénale.

Mots clés : Auto-immunité ; Diabète auto-immun latent de l’adulte ; LADA ; Épidémiologie du diabète ; Néphropathie diabétique ; Albuminurie ; Microalbuminurie

1. Abbreviations

HUNT 2 22nd Nord-Trøndelag Health Study
LADA latent autoimmune diabetes in adults
ACR albumin-to-creatinine ratio
MA microalbuminuria
MDRD modification of diet in renal disease
GFR glomerular filtration rate
GLM general linear model
OR odds ratio
ACE angiotensin-converting enzyme
CV cardiovascular
UAE urinary albumin excretion

2. Introduction

Over the last two decades, a new major type of diabetes has become recognized—namely, latent autoimmune diabetes in adults (LADA). LADA patients are clinically diagnosed as non-insulin-dependent diabetics, but show evidence of autoimmunity in the form of islet-directed antibodies such as those against glutamic acid decarboxylase (GAD).

LADA constitutes a large category of diabetes, accounting for approximately 10% of the diabetic population [1,2]. Yet, the natural history of LADA has been considerably less well characterized than those of other types of diabetes. Indeed, one aspect that needs further clarification is the development of diabetic complications in LADA.

An increase in albuminuria is a recognized sign of early nephropathy. Several studies have described the prevalence of nephropathy in type 1 (T1D) and type 2 (T2D) diabetes using albuminuria as a marker [3–6]. Measuring albumin excretion in LADA patients would also provide information on the extent of incipient and overt nephropathy in this form of diabetes. However, the detailed characterization of albumin excretion in LADA patients is, to our knowledge, so far lacking. It is therefore unclear as to whether or not LADA patients display albuminuria to the same extent as the “classical” types (1 and 2) of diabetes.

For this reason, using data gathered from a large population-based health survey in Norway to study urinary albumin excretion (UAE) in LADA, we compared the mean and distribution of albumin-to-creatinine ratios (ACR) and the prevalence of microalbuminuria (MA) in LADA and in the “classical” T1D and T2D diabetes. For further comparisons, we also included a non-diabetic, non-hypertensive group from the study population.

3. Materials and methods

3.1. Subjects

In Nord-Trøndelag County, Norway, all inhabitants aged 20 years or older were invited to participate in a large health survey, carried out between 1995 and 1997 (HUNT 2). A total of 64,931 (70.4%) of the eligible population participated. All of these volunteers underwent a clinical examination and filled out a questionnaire. The latter was included with the invitation to participate and contained items on a range of lifestyle- and health-related topics, including self-reported diabetes. The clinical examination included standardized measurements of height, weight and blood pressure and a non-fasting blood sample.

The participants who reported diabetes in the initial questionnaire were asked to participate in an additional study and to fill in an additional questionnaire, including questions on diabetes such as duration, medication and complications. In addition, these diabetics were included in a screening investigation for microalbuminuria (MA). The MA screening also included a 5% random sample from the non-diabetic population [7].

3.2. Microalbuminuria screening

For the MA screening, participants received three plastic receptacles, transport tubes and one pre-stamped envelope bearing the postal address of the analyzing laboratory. They all also received written instructions on how to collect their morning urine as well as being informed on the technique by a trained nurse. All were asked to fill in a questionnaire on urinary tract infections during the previous week, persistent haematuria during the previous year and the date of their most recent menstrual period. MA was defined as an ACR superior or equal to 2.5 mg/mmol to an ACR inferior or equal 30 mg/mmol in at least two of the three urine samples.

3.3. Blood sampling and laboratory procedures

A non-fasting blood sample was drawn from all subjects who participated in the primary investigation (n = 64,931). From the participants with self-reported diabetes, one additional blood sample was taken and analyzed for HbA1C. These participants were also asked to provide a further blood sample after an overnight fast to obtain data on fasting glucose, fasting C-peptide and anti-GAD antibodies.

Fasting glucose was measured at each site of investigation by HemoCue®, while HbA1C was analyzed using Architect c8000/ci8200 (Abbott) at Levanger Hospital. In addition, C-peptide was measured by radioimmunoassay (RIA); Diagnostic
The diabetic groups in the study were classified as:

- T1D: those who started insulin treatment within 6 months of diabetes diagnosis and anti-GAD antibodies superior or equal to 0.08 or anti-GAD antibodies inferior to 0.08 combined with fasting levels of circulating C-peptide inferior to 150 pmol/L;
- T2D: those with anti-GAD antibodies inferior to 0.08 and dietary or oral therapy or insulin treatment started more than 12 months after diabetes diagnosis;
- LADA: those with anti-GAD antibodies superior or equal to 0.08 and lifestyle changes and oral therapy or insulin treatment started more than 12 months after diabetes diagnosis or insulin therapy started within 12 months of diagnosis but with fasting C-peptide superior to 150 pmol/L.

3.5. Attendance and inclusion procedure

Of the 1952 subjects who reported having diabetes in the initial questionnaire, 1437 (74%) attended the additional blood sampling in the overnight fasting state and answered the additional questionnaire. Of these, we were able to classify the specific types of diabetes (see below) and somewhat lower in the non-diabetic group (73%). Ultimately, only those who delivered all three urine samples were included. We also excluded those with self-reported urinary tract infections, haematuria or menstruation, as the urine samples were not examined for microscopic haematuria or asymptomatic bacteriuria. (The inclusion of such conditions would probably have led to random misclassifications, at least among the diabetic groups. However, asymptomatic bacteriuria is not thought to influence albumin excretion except in the presence of pyuria [11].)

We excluded participants with manifest proteinuria from most of the analyses, as our objective was to study the distribution of albumin excretion at the level of microscopic haematuria or below. Manifest proteinuria (defined as a mean ACR >30 mg/mmol) was present in 47 participants (2.0%). Those who presented with proteinuria were distributed as follows: T1D: \( n = 3 \) (3.2% of all T1D patients included in the analysis); T2D: \( n = 40 \) (4.2%); LADA: \( n = 4 \) (3.7%); and non-diabetics: \( n = 4 \) (0.2%). Thus, a total of 221 participants were excluded from the final analysis because of proteinuria. However, these participants did not differ from those without manifest proteinuria in terms of age or diabetes duration (results not shown).

In total, 60% of all patients with self-reported diabetes were included in the final analyses.

3.6. Statistical analyses

The ACR data were logarithmically transformed because of the skewed distribution. We compared the unadjusted geometric means and 95% confidence intervals (CI) of the ACR between the different study groups. A general linear model was used to compare the beta coefficients of the adjusted ACR. The covariates were entered in sequential models as follows:

- model 1: age and gender;
- model 2: age, gender and diabetes duration;
- model 3: age, gender, diabetes duration and HbA1c;
- model 4: age, gender, diabetes duration, HbA1c, systolic blood pressure, use of ACE inhibitors or angiotensin II receptor blockers, body mass index (BMI) and high density lipoprotein (HDL) cholesterol.

\( P \) values were calculated for differences relative to data from the LADA group while, in the analyses adjusted for diabetes duration and HbA1c, we included only the diabetic groups. To test for effect modification, the analyses were stratified according to median values of age and diabetes duration. We also calculated the odds ratios (OR) and 95% CI for MA across the different groups.

All analyses were performed using the statistical software SPSS, version 13.0 for Windows (SPSS Inc., 1989–2004).

3.7. Consent

Participation in the study was voluntary and all participants gave their written consent to do so. The study was approved by the regional ethics committee and by the Norwegian Data Inspectorate.

4. Results

4.1. Baseline characteristics

The baseline characteristics of the study participants are given in Table 1. It is evident that features such as BMI, age and lipid parameters were similar among the LADA and T2D patients.

4.2. Distribution of albumin excretion

Fig. 2 shows the ACR distribution curves for the different study groups. Again, it is clear that the curves for T2D and LADA are similar and skewed towards higher levels of ACR than are the curves for T1D and the non-diabetic group.
Fig. 1. Classification and inclusion of participants with self-reported diabetes. Excluded from the analyses were those with self-reported urinary tract infections (UTIs) within the previous week, menstruation at the time of sample collection or persistent haematuria or manifest proteinuria.

4.3. Albumin-to-creatinine ratios

The unadjusted mean ACR in LADA was similar to the mean ACR in T2D but higher than in the T1D and non-diabetic group. A similar relationship of LADA with both “classical” types of diabetes was demonstrable in the various adjusted models presented in Table 2. Thus, the similarity of LADA to T2D and its differences compared with T1D were maintained even after stepwise adjustments for age, gender, diabetes duration, HbA1c, BMI, systolic blood pressure and use of ACE inhibitors or angiotensin II receptor blockers. After making additional adjustments for HDL cholesterol, the difference between LADA and T1D was reduced but there was still a clear trend of a higher mean ACR in the LADA group ($P = 0.125$).

4.4. Effect modification

Because diabetes duration and age differed between the diabetes groups, we looked for the effect modification with these factors. However, no effect modification was seen with diabetes

Table 1
Baseline characteristics of the study participants.

<table>
<thead>
<tr>
<th></th>
<th>T1D</th>
<th>T2D</th>
<th>LADA</th>
<th>Non-diabetics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases (n)</td>
<td>93</td>
<td>961</td>
<td>109</td>
<td>2104</td>
</tr>
<tr>
<td>Age (years)</td>
<td>50.3 (±15.5)*</td>
<td>67.7 (±10.8)</td>
<td>67.2 (±11.6)</td>
<td>49.0 (±48.9)*</td>
</tr>
<tr>
<td>Gender (male/female) (%)</td>
<td>60/40</td>
<td>49/51</td>
<td>54/46</td>
<td>46/54</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>26.0 (±3.9)*</td>
<td>29.5 (±4.8)*</td>
<td>28.4 (±4.4)</td>
<td>26.1 (±3.8)*</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>86.5 (±11.4)*</td>
<td>96.5 (±11.5)*</td>
<td>93.9 (±10.7)</td>
<td>85.6 (±11.4)*</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>0.84 (±0.07) *</td>
<td>0.90 (±0.08)</td>
<td>0.89 (±0.08)</td>
<td>0.84 (±0.08)*</td>
</tr>
<tr>
<td>C-peptide (pmol/L)</td>
<td>54 (±82.1)*</td>
<td>825 (±504.3)*</td>
<td>609 (±535.6)</td>
<td>–</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>8.86 (±1.54)</td>
<td>7.87 (±1.63)*</td>
<td>8.55 (±2.16)</td>
<td>–</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>142/79 (±20/10)*</td>
<td>156/86 (±23/13)</td>
<td>154/83 (±22/12)</td>
<td>136/80 (±21/12)*</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>5.57 (±1.11)*</td>
<td>6.25 (±1.28)*</td>
<td>5.91 (±1.30)</td>
<td>5.89 (±1.21)</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.64 (±0.34) *</td>
<td>1.21 (±0.49)</td>
<td>1.28 (±0.50)</td>
<td>1.39 (±0.39)</td>
</tr>
<tr>
<td>GFR (ml min⁻¹·[1.73 m²]⁻¹)</td>
<td>94.7 (±21.9) *</td>
<td>82.6 (±20.4)</td>
<td>83.3 (±22.8)</td>
<td>96.2 (±21.2)</td>
</tr>
<tr>
<td>Current smoker (%)</td>
<td>25*</td>
<td>16</td>
<td>9</td>
<td>29*</td>
</tr>
<tr>
<td>Diabetes duration (years)</td>
<td>22.3 (±14.77) *</td>
<td>8.64 (±7.56)</td>
<td>10.1 (±8.16)</td>
<td>–</td>
</tr>
<tr>
<td>Anti-GAD antibodies</td>
<td>0.33 (±0.51)</td>
<td>–</td>
<td>0.37 (±0.52)</td>
<td>–</td>
</tr>
<tr>
<td>Insulin (yes/no)</td>
<td>93*</td>
<td>174/787*</td>
<td>38/71</td>
<td>–</td>
</tr>
<tr>
<td>ACEI or angiotensin II receptor blockers (%)</td>
<td>17.2</td>
<td>15.6</td>
<td>16.5</td>
<td>–</td>
</tr>
<tr>
<td>Antihypertensive treatment (%)</td>
<td>29.0</td>
<td>43.6</td>
<td>39.4</td>
<td>–</td>
</tr>
<tr>
<td>Oral antidiabetic treatment (%)</td>
<td>–</td>
<td>51.4</td>
<td>43.3</td>
<td>–</td>
</tr>
</tbody>
</table>

T1D: type 1 diabetes; T2D: type 2 diabetes; LADA: latent autoimmune diabetes in adults; BMI: body mass index; GFR: glomerular filtration rate; ACEI: ACE inhibitors; *P<0.05 for (unadjusted) differences compared with LADA; missing data for the variables: BMI: 2.7% in the LADA group; HbA1c: 3.2% in T1D; 2.7% in LADA; <2% for all other variables.
Table 2
Comparison of albumin-to-creatinine ratio (ACR) between study groups.

<table>
<thead>
<tr>
<th></th>
<th>LADA</th>
<th>Type 1 diabetes</th>
<th>Type 2 diabetes</th>
<th>Non-diabetics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted mean ACR (CI)</td>
<td>1.45 (1.23–1.71)</td>
<td>0.99 (0.83–1.19)</td>
<td>1.41 (1.33–1.49)</td>
<td>0.72 (0.69–0.74)</td>
</tr>
<tr>
<td>β (P value)</td>
<td>1.00</td>
<td>0.68 (0.002)</td>
<td>0.97 (0.757)</td>
<td>0.49 (&lt;0.001)</td>
</tr>
<tr>
<td>Model 1a</td>
<td>1.00</td>
<td>0.82 (0.110)</td>
<td>0.97 (0.734)</td>
<td>0.60 (&lt;0.001)</td>
</tr>
<tr>
<td>Model 2b</td>
<td>1.00</td>
<td>0.69 (0.024)</td>
<td>1.00 (0.985)</td>
<td>–</td>
</tr>
<tr>
<td>Model 3c</td>
<td>1.00</td>
<td>0.66 (0.013)</td>
<td>1.04 (0.687)</td>
<td>–</td>
</tr>
<tr>
<td>Model 4d</td>
<td>1.00</td>
<td>0.77 (0.125)</td>
<td>1.03 (0.801)</td>
<td>–</td>
</tr>
</tbody>
</table>

Data are expressed as unadjusted means with 95% confidence intervals (CI) and β-coefficient with P values relative to LADA. p values in bold

a Age, gender.
b Age, gender, diabetes duration.
c Age, gender, diabetes duration, HbA1c.
d Age, gender, diabetes duration, HbA1c, systolic blood pressure, use of ACE inhibitors or angiotensin II receptor blockers, HDL cholesterol, body mass index.

duration, as the mean ACR was (not significantly) lower in the T1D patients with either shorter or longer diabetes durations (results not shown). However, when grouping by median age (55 years), we found some evidence of an effect modification although, again, it was not significant. In subjects aged inferior to 55 years, the mean UAE (adjusted for gender, diabetes duration, HbA1c, BMI, systolic blood pressure, HDL cholesterol and use of ACE inhibitors or angiotensin II receptor blockers) in T2D was 106% compared with 63% in T1D (P = 0.133) in relation to that in LADA. This difference was attenuated in the superior 55 age-group, with an adjusted UAE in T1D of 88% (not significant) in relation to that in LADA.

4.5. Prevalence of microalbuminuria

The prevalence of MA was 23% in the LADA group, 16% in the T1D group and 25% in the T2D group. Compared with LADA, those with T2D had an OR for MA of 1.13 (95% CI: 0.71–1.81), those with T1D had an OR of 0.65 (95% CI: 0.32–1.32) and those in the non-diabetic group had an OR of 0.17 (95% CI: 0.10–0.27). Adjusting for age, gender, diabetes duration, HbA1c, systolic blood pressure, use of ACE inhibitors or angiotensin II receptor blockers, HDL cholesterol or BMI had no impact on the OR.

4.6. Additional analyses

Inclusion of participants with manifest proteinuria had no impact on the differences observed between groups except for larger CI (results not shown). In addition, adjustments for smoking or for waist circumference instead of BMI also had no influence on the results.

5. Discussion

The main finding of our study is that LADA patients display albuminuria to a degree that is similar to that of T2D patients: the adjusted mean UAE did not differ between the LADA and T2D groups. This is in line with the phenotype similarities seen between the present population of LADA patients and those with T2D.

In contrast, we found that UAE was higher in the LADA than in T1D patients, even after stepwise adjustments for multiple possible confounders. Only by adding HDL cholesterol to the adjusted model was the difference between LADA and T1D reduced (P = 0.125). Yet, adjusting for total cholesterol had no effect on the level of significance. It may be that the more favourable lipid profiles in our T1D patients versus our LADA and T2D patients had a limited impact on UAE in some direct or indirect manner.

In addition, adjusting for age and diabetes duration did not influence the differences between LADA and T1D. Still, it remains possible that a longer diabetes duration and/or younger age among T1D patients could be of importance in the lower ACR seen in T1D because of an effect modification. For this reason, we tested this by splitting the diabetes groups into longer and shorter diabetes durations, and into ages below and above 55 years. The results offer some support for an effect modification by age. Thus, only in the younger age group did we find a difference between LADA and T2D, on one hand, compared with T1D, on the other.
A comparison of the entire range and distribution of UAE reveals another aspect of similarity between LADA and T2D. In the range of albumin concentrations between zero and those approaching MA, the distribution ACR curves for both LADA and T2D were similarly shifted to the right compared with T1D (and non-diabetics). However, the distribution curves of the all diabetic groups became similar at higher levels of albuminuria. This is reflected in the OR for MA, the latter being only non-significantly lower in T1D compared with LADA (P = 0.23 for differences).

The differences in albuminuria that we found between LADA and T2D versus ‘classical’ T1D remain, at present, unexplained. It may be possible that aspects of insulin resistance, which are not encompassed by the factors adjusted for in our analyses, are of importance. Such an explanation has also been suggested by the recent findings in adolescents, which show significantly higher rates of UAE in T2D than in T1D [12,13].

So, how representative are the present study’s findings? For T2D, the prevalence of MA in our study was 25% and the mean disease duration was 8.6 years. These figures are in reasonable agreement with those of the UK Prospective Diabetes Study (UKPDS) [6]. As for T1D, the prevalence of MA in our study was 16% and the mean disease duration was 22 years. This frequency is lower than in previous studies, which reported prevalences of 20 to 30% after 15 years of diabetes [5,14]. That our T1D patients were population-based rather than hospital-based may explain the discrepancy.

To our knowledge, only the study of Isomaa et al. [15] can be compared with our data for LADA and risk of MA. Isomaa et al. studied the prevalences of macro- and microvascular complications, including microalbuminuria, in patients with LADA, T1D and T2D. The latter and LADA patients were from the Botnia study whereas their T1D patients were gathered from hospital registers. Isomaa et al. found that the prevalence of MA was similar across all three diabetes groups (27% for LADA, 29% for T2D and 24% for T1D) [15]. In the present study, the prevalence of MA in LADA was similar (23%), but higher than in the T1D group (16%). This difference between studies cannot be explained by differences in glycaemic control, BMI or diabetes duration. As already mentioned above, the fact that our T1D patients were population-based could be the reason for the difference.

Incidentally, a relatively low use of ACE inhibitors or angiotensin II receptor blockers was seen in our diabetic groups (Table 1). Such low use is probably due to the fact that these drugs were not the first-line choices for antihypertensive or renoprotective treatment at the time of our data collection.

Our study has both strengths and limitations. One of its strengths pertains to the type of survey we used. The HUNT surveys covered an entire homogeneous population within a given geographical area with a relatively high participation rate (70.4%). Also, the population in Nord-Trøndelag can be considered to be a reasonable representation of the entire population of Norway. Furthermore, the prevalence of diabetes (total and T2D, T1D and LADA separately) is similar to that of other population studies in the Scandinavian countries. Moreover, the database used in the assessment of albuminuria is, in our opinion, excellent. (This database has so far led to reports on the prevalence of MA in the general population, in the hypertensive population and in the overall diabetic population [7,16–20].)

As for limitations, the HUNT survey did not include those with T1D below the age of 19 at the time of the investigation. This means that albuminuria in the younger age group with T1D could not be assessed. In addition, the fact that the diagnosis of diabetes was based on the participants’ own admission suggests that undiagnosed cases were not included. (Nevertheless, self-reported diagnoses of diabetes have proven to be correct in 96.4% of cases in the HUNT survey [21].) However, we acknowledge uncertainty about the group of participants with self-reported diabetes that could not be classified because of a lack of follow-up data. Finally, the inherent limitations of a cross-sectional study need to be borne in mind.

6. Conclusion

In this cross-sectional study, the ACR for LADA was increased to a level closer to that of T2D than T1D even after multiple adjustments for phenotype differences. The similar patterns of albumin excretion seen in both LADA and T2D indicate that the autoimmune activity in LADA does not include any damaging effects on the kidneys.

Conflict of interests

The authors have none to declare.

Acknowledgements

The Nord-Trøndelag Health Study (HUNT study) is a collaboration by the HUNT Research Centre, Faculty of Medicine, Norwegian University of Science and Technology, Verdal; the Norwegian Institute of Public Health, Oslo; and the Nord-Trøndelag County Council and Central Norway Regional Health Authority. We thank the Nord-Trøndelag Hospital Trust, the health service and the people in Nord-Trøndelag for their endurance and participation. We also thank Sofia Carlsson and Stein Hallan for their helpful advice and discussions.

M.R. is a recipient of a research fellowship from the Central Norway Regional Health Authority and Norwegian University of Science and Technology.

References


