Health inequalities across patients with early inflammatory arthritis of different ethnicities: what could be the driving factors?

Bergstra, S.A.

RHEUMATOLOGY

Rheumatology 2023;62:7–8
https://doi.org/10.1093/rheumatology/keac383
Advance access publication 4 July 2022

Editorial

Health inequalities across patients with early inflammatory arthritis of different ethnicities: what could be the driving factors?

This editorial refers to the article 'Worse outcomes linked to ethnicity for early inflammatory arthritis in England and Wales: a national cohort study', published by Adas MA, Norton S, Balachandran S *et al.*, 2023;62:169–180.

The treatment options and disease outcomes of inflammatory rheumatic conditions, such as RA, have dramatically improved over the past decades [1]. However, not all patients have benefitted similarly from these improvements. Differences in treatment outcomes have been described for different rheumatic conditions across countries, areas and neighbourhoods, but also across patient groups with different racial or ethnic backgrounds [2–5]. In the study of Adas *et al.* that is published in this edition, the authors describe a national cohort study performed in England and Wales, in which they assess different indicators of quality of care and treatment outcomes across ethnicities in early inflammatory arthritis (EIA) [6].

Based on a large group of patients, with representation from most rheumatology departments in England and Wales, the authors demonstrated that compared with white patients, several ethnic minority groups with confirmed EIA were less likely to achieve disease remission (measured as DAS28 <2.6) three months after start of treatment. The proportion of patients achieving remission was 37% in white patients, 31% in Asian patients, 27% in mixed patients and only 23% in black patients, which can be considered a substantial difference within only 3 months of treatment. For individual patients, not being able to achieve remission at 3 months may have important consequences regarding their long-term disease outcomes in these patients, as achieving early favourable outcomes has been identified as a strong indicator for favourable long-term outcomes [7].

Next to the identification of this inequity across ethnic groups, it is important to identify potential drivers of this difference, which may help healthcare providers and policy makers to take steps to reduce these health inequalities. These drivers are likely multifactorial, and may include, among others, differences in patients' genetic (e.g. ACPA) and environmental (e.g. smoking) risk factors, differences in socioeconomic indicators or health behaviour (e.g. seeking medical advice or treatment adherence), differences in work or living environment (e.g. air pollution) or (unconscious) differences in provided treatment [8, 9].

The authors discuss some of these potential drivers, with a focus on quality metrics of RA care as defined by NICE. Although they show that approximately half of the EIA patients are not referred and treated within the ideal timeframe as defined by these quality metrics, this does not differ between different ethnic minorities. The results do show that patients of mixed ethnicity less often receive disease-specific education and are less often treated towards a target of low disease activity or remission, but this cannot explain lower remission rates in patients with black or Asian ethnicity. Thus, improving NICE quality metrics is unlikely to reduce inequalities in achieving remission in these groups.

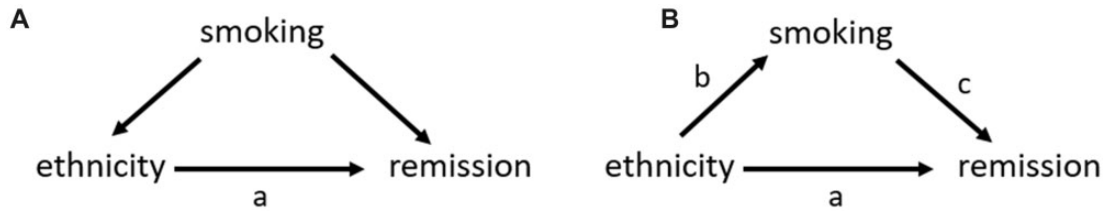
An alternative proposed explanation is sub-optimal initial treatment. Most patients included (70% overall) receive a working diagnosis of RA, with a higher proportion of RA patients with black (80%) and a lower proportion of RA patients with mixed (60%) ethnicity. However, the proportion of patients receiving methotrexate, potentially in combination with glucocorticoids, is lower for ethnic minorities than for white patients, which may explain (part of) the observed difference in achieving remission.

A complicating factor is that different ethnic groups differed in their baseline characteristics. Differences in age, gender, smoking behaviour and RF and/or ACPA positivity may as well explain differences in health outcomes as medication use [10]. The authors treated these baseline characteristics as confounders for the relationship between ethnic minority and the different outcomes (e.g. remission, quality of care indicators), and indeed reported differences in adjusted and unadjusted results. However, a definition of a confounder is a variable that influences both the dependent variable (e.g. achieving remission) and the independent variable, in this case ethnicity, without being in the causal pathway between the independent and the dependent variable. It is difficult to argue that baseline characteristics such as gender, smoking or ACPA positivity would influence one's ethnicity.

Moreover, treating these variables as confounders does not allow us to evaluate them as potential drivers of health inequalities across ethnicities.

Instead, it would be interesting to treat these variables as potential mediators. This would allow us to interpret both the direct effect between ethnicity and each outcome, as well as the indirect effect between ethnicity, potential mediator and outcome (Fig. 1). It is even possible to evaluate multiple potential mediators simultaneously, to gain more insight into which of the measured factors contribute most strongly to health inequalities across ethnicities.

Fig. 1 Confounding *vs* mediation



In (**A**) we are interested in the relationship (a) between ethnicity and achieving remission. By adjusting for smoking, we aim to observe the unconfounded relationship (a) ethnicity and remission. However, when treating smoking as a confounder for this relationship according to the provided definition, we implicitly assume that it influences both ethnicity (which is impossible in real life) and remission. In (**B**) we are also interested in the relationship (a) between ethnicity and smoking. Ethnicity is assumed to influence smoking, and smoking is assumed to influence the chance of achieving remission. Therefore, smoking acts here as a mediator for this relationship. By also examining the relationship (b) between ethnicity and smoking and the relationship (c) between smoking and remission, we can also evaluate both the direct effect (a) between ethnicity and remission, and the indirect effect (b and c) between ethnicity and remission via smoking.

For now, the question 'Which of the proposed factors contributes most to health inequalities in EIA?' will remain unanswered. Based on the current evidence, multiple factors may play a role, and it is evident that intervening and adjusting for some of these factors (e.g. starting treatment) is easier than for others (e.g. ACPA status). Moreover, whereas some factors can be directly influenced by healthcare providers (e.g. providing educational materials), this is not the case for other factors (e.g. air pollution).

Nevertheless, the authors provide important insights in existing health inequalities across different ethnic minorities, which are likely not restricted to England and Wales.

Studies such as these increase the attention for potential health inequalities across ethnicities, which may increase awareness among healthcare providers. This enables us, even if we do not yet know the exact drivers of these health inequalities, to ensure that patients of different ethnicities receive equal care concerning the factors healthcare providers can directly influence in daily practice.

Funding: No specific funding was received from any bodies in the public, commercial or not-for-profit sectors to carry out the work described in this article.

Disclosure statement: The author has declared no conflicts of interest.

Data availability statement

There are no new data associated with this article.

Sytske Anne Bergstra[1]

[1]*Department of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands*
Accepted 27 June 2022
Correspondence to: Sytske Anne Bergstra, Department of Rheumatology, C1-R, Leiden University Medical Center, Albinusdreef 2, 2333 ZA, Leiden, The Netherlands.
E-mail: s.a.bergstra@lumc.nl

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