

Opinion

Abdominal and pelvic adhesions: are blacks different?

Emmanuel Nzau-Ngoma^{1,&}, Jean-Marie Mbuyi-Muamba², Justin Esimo Mboloko¹, Bienvenu Massamba Lebwaze³, Willy Arung⁴

¹Department of Obstetrics and Gynecology, University Hospital of Kinshasa, DR Congo, ²Department of Internal Medicine, University Hospital of Kinshasa, DR Congo, ³Department of Pathology, University Hospital of Kinshasa, DR Congo, ⁴Department of Surgery, University Hospital of Lubumbashi, DR Congo

[&]Corresponding author: Emmanuel Nzau-Ngoma, Department of Obstetrics and Gynecology, University Hospital of Kinshasa, DR Congo

Key words: Abdominal adhesions, pelvic adhesions, disparities, abdominal surgery

Received: 20/03/2016 - Accepted: 07/04/2016 - Published: 14/04/2016

Abstract

Studies conducted mostly in the United States bring evidence on racial disparities between blacks and whites in various pathologies including asthma and other allergies whose pathophysiology relates in part to innate immunological characteristics such as variation in host defense genes. There are various other pathologies evoking that black people could have an immune overreaction in response to diverse aggressions. For example, the prevalence of systemic lupus erythematous and systemic sclerosis has been found to be higher in black Americans compared to Caucasians suggesting, at least in part, racial disparities in immunological reactions. So, these arguments borrowed from other fibrotic disorders suggest a hypothetic racial disparity in abdominal and pelvic adhesions. Data on this condition according to racial disparity are scarce and should incite further researches to bring new findings on that question. As a result, a disparity between races will motivate the identification of genetic support which will give new insights in prevention and therapy of adhesions.

Pan African Medical Journal. 2016; 23:186 doi:10.11604/pamj.2016.23.186.9381

This article is available online at: http://www.panafrican-med-journal.com/content/article/23/186/full/

© Emmanuel Nzau-Ngoma et al. The Pan African Medical Journal - ISSN 1937-8688. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/2.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.



Opinion

Abdominal and pelvic adhesions (APA) although may cause few or no detrimental effects, in an important proportion their burden in terms of short and long term consequences is considerable with associated morbidity, mortality, prolonged operating time, visceral injuries during surgery and expense. Given the complexity of their pathophysiology, there is no unique effective therapy that is sufficient to reduce their incidence. Although many researchers' teams and scientific organizations around the world are committed to address various issues regarding adhesions, in Africa there is a literature deficiency though some insights on the similarity of APA with other fibrotic disorders suggest a concerned pattern in people of African origin. The present paper addresses the APA with the opinion that black people could experience a high burden of abdominal and pelvic adhesions. We present here few arguments from other racial diseases disparities to support the same possible disparity regarding APA that should be of interest for researchers. Studies conducted mostly in the United States (US) bring evidence on racial disparities between blacks and whites in various pathologies including asthma and other allergies whose pathophysiology relates in part to innate immunological characteristics such as variation in host defense genes [1]. Indeed, it is well established in the US that the prevalence, morbidity, and mortality of allergic airway diseases, such as asthma, is higher among blacks [2]. This finding was first thought to be exclusively related to socioeconomic characteristics, but well-designed studies demonstrated phenotypic and genotypic variations between blacks and whites associated to this disparity [3]. There are various other pathologies evoking that black people could have an immune overreaction in response to diverse aggressions. For example, the prevalence of systemic lupus erythematous [4] and systemic sclerosis [5], two autoimmune diseases, has been found to be higher in black Americans compared to Caucasians suggesting, at least in part, racial disparities in immunological reactions. One of the most plausible theories supporting genetic differences between blacks and whites and explaining noticeable disparities related to an array of pathologies is the longtime parasitic exposure of African Ancestries. In that theory, the exposure to severe parasitic pathologies contributed to gene selection responsible of individuals' survival. In fact, some observations suggested that the systemic lupus erythematous susceptibility among blacks is related to genes protecting against severe malaria [6]. In the absence of continual exposure to Plasmodium falciparum; parasite responsible of malaria, these genes may contribute to hyperimmune responses typical of systemic automimmune diseases. Another illustration is given by the susceptibility to nephrosclerosis due to the presence of Apolipoprotein L1, a protein associated with relative immunity to trypanosomal infection [7] but causing damages in the kidney in the absence of parasitic infection.

Adhesions share an array of characteristics with many other diseases whose pathophysiology lies on fibroblastic activity. Many studies mostly from American authors showed a higher prevalence of some of those pathologies, namely leiomyoma and skin scar anomalies (keloid and hypertrophic scar) in black people than in other races [8]. Likewise, skin scar anomalies have been found to be most prevalent in black people compared to white people. According to APA, data are very scarce in the literature, but their pathophysiological similarity to other fibrotic disorders suggests the same pattern of disproportionate prevalence in black people. Transforming Growth factor (TGF) - $\beta 1$, $\beta 2$ and $\beta 3$ are the three isoforms expressed in mammal cells. The isoform $\beta 1$ is the most powerful fibrotic agent studied and is involved into proliferation, differentiation, migration and remodeling of extracellular matrix [9].

The aforementioned disproportionate prevalence in a variety of different fibrotic disorders has been attributed to an over-expression or activation of TGF- β signaling pathways and their effects on related systems [10]. Eiser [10] hypothesized that heritage polymorphism in blacks may not only result in higher prevalence considering Leiomyoma or keloids but also in other diseases characterized by aberrant fibrosis. We could add APA in the list of disorders mentioned by this author. All those observations instill the idea that there could be a possible overreaction of blacks in terms of inflammation, then fibrosis in an amount of pathologies including APA too. One could hypothesize that immunological response can be stronger in black people than in whites facing the same level of aggression. Indeed, pelvic adhesions secondary to Pelvic Inflammatory Disease (PID) may result in most severe adhesions in blacks than in whites. Observations of physiologic differences on APA among races can be important keys to allow better profiling of risk patients and will give new insights to investigate genetic candidates responsible of that difference. Finally, this information will help for specific recommendations related to management of pathologies with fibrosis as pathophysiological support, such as APA. For instance, the awaiting time to begin infertility exploration could be turned shorter in Sub Saharan Africa when we consider that fibrosis through APA ranks first in the infertility etiologies and so, waiting longer can have deleterious consequences given the limited access to modern therapies in the setting.

Competing interests

The authors declare no competing interest.

Authors' contributions

All authors have read and agreed to the final version of this manuscript and have equally contributed to its content and to the management of the case.

References

- 1. Gao L, Tsai YJ, Grigoryev DN, Barnes KC. Host defense genes in asthma and sepsis and the role of the environment. Curr Opin Allergy Clin Immunol. 2007; 7(6): 459-467. **PubMed | Google Scholar**
- El-Ekiaby A, Brianas L, Skowronski ME, Coreno AJ, Galan G, Kaeberlein FJ, Seitz RE, Villaba KD,Dickey-White H, McFadden ER Jr. Impact of race on the severity of acute episodes of asthma and adrenergic responsiveness. Am J Respir Crit Care Med. 2006; 174(5): 508-513. PubMed | Google Scholar
- Vergara CMurray T,Rafaels N, Lewis R, Campbell M, Foster C, Gao L, Faruque M, Oliveira RR, Carvalho E, Araujo MI, Cruz AA, Watson H, Mercado D, Knight-Madden J, Ruczinski I, Dunston G, Ford J, Caraballo L, Beaty TH, Mathias RA, Barnes KC. African Ancestry is a Risk Factor for Asthma and High Total IgE Levels in African Admixed Populations. Genet Epidemiol. 2013; 37(4): 393-401. PubMed | Google Scholar
- Danchenko N, Satia JA, Anthony MS. Epidemiology of systemic lupus erythematosus: a comparison of worldwide disease burden. Lupus. 2006; 15(5): 308-318. PubMed | Google Scholar

- Nietert PJ, Mitchell HC, Bolster MB, Shaftman SR, Tilley BC, Silver RM. Racial variation in clinical and immunological manifestations of systemic sclerosis. J Rheumatol. 2006; 33(2): 263-268. PubMed |Google Scholar
- Waisberga M, Tarasenkoa T, Vickersa BK, Scotta BL, Willcocksb LC, Molina-Cruzc A, Piercea MA, Huangd C, Torres-Veleze FJ, Smithb KGC, Barillas-Muryc C, Millerc LH, Piercea SK, Bollanda S. Genetic susceptibility to systemic lupus erythematosus protects against cerebral malaria in mice. PNAS. 2011; 108(3): 1122-1127.PubMed | Google Scholar
- Freedman BI. APOL1 and nephropathy progression in populations of African ancestry. Semin Nephrol. 2013 Sep; 33(5): 425-32. PubMed | Google Scholar

- Chike-Obi CJ, Cole PD, Brissett AE. Keloids: pathogenesis, clinical features, and management. Semin Plast Surg. 2009; 23(3): 178-184. PubMed | Google Scholar
- Bergström R, Savary K, Morén A, Guibert S, Heldin CH, Ohlsson R, Moustakas A. Transforming growth factor β promotes complexes between Smad proteins and the CCCTC-binding factor on the H19 imprinting control region chromatin. J Biol Chem. 2010; 285(26): 19727- 19737. PubMed | Google Scholar
- Eiser AR. Does over-expression of transforming growth factorbeta account for the increased morbidity in African-Americans?: possible clinical study and therapeutic implications. Medical Hypotheses. 2010; 75(5): 418-421. PubMed | Google Scholar