Abdominal and pelvic adhesions: are blacks different?

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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 erythematosus 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.


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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 erythematosus [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 erythematosus 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 autimmune 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) - β1, β2 and β3 are the three isoforms expressed in mammal cells. The isoform β1 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


