Letter to Editor

What is the important practical implication of detecting decreased G6PD levels in vitiligo?

Sir/Madam,

Vitiligo is an acquired depigmenting disorder affecting 1% of the world population. Oxidative stress has been proposed to be one of the major players in the pathogenesis of this enigmatic disorder. It has been suggested that oxidative stress produces 'neoantigens', which can ignite an autoimmune attack against melanocytic antigens through 'molecular mimicry'. Moreover, the immune attack against neoantigens can release a plethora of sequestered autoantigens, culminating in the 'breakdown of tolerance'. Oxidative stress can also induce the apoptosis of melanocytes, during which membrane-associated autoantigens may form. On the other hand, oxidative stress can amplify antigen presentation, the release of cytokines, and an immunological attack.[1]

Several studies have shown that the levels of Glucose-6-phosphate dehydrogenase (G6PD), one of the major antioxidant enzymes, are remarkably lower in patients with vitiligo compared to healthy controls.[2-4]

Glucose-6-phosphate dehydrogenase is a rate-limiting enzyme in the pentose phosphate pathway that maintains the level of the coenzyme nicotinamide adenine dinucleotide phosphate (NADPH). The NADPH in turn maintains the supply of reduced glutathione and catalase in the cells that are used to detoxify free radicals and hydrogen peroxide.[5]

Of note, markedly increased levels of hydrogen peroxide in vitiligo skin are suggested to be of crucial importance in the pathophysiology of this complex disorder via a variety of ways, such as, enhancing the production of reactive quinones[6] and disturbing the homeostasis of calcium and the uptake of l-phenylalanine in the epidermis.[7]

Given the already decreased G6PD levels in vitiligo, overexhausting the G6PD-dependent detoxifying enzymes by agents known to induce hemolysis in G6PD-deficient individuals can potentially exacerbate this condition. The high levels of the potent oxidants vicine, divicine, convicine, and isouramil in Fava beans are the typical examples of such agents.[8] Interestingly, antimalarials, known to induce hemolysis in G6PD-deficient individuals through generating free radicals that exhaust the G6PD-dependant detoxifying enzymes, are well-known triggers of vitiligo.[9] We suggest that henna, another inducer of hemolysis in G6PD-deficient individuals, has also got the potential to exacerbate vitiligo. We believe that the vitiligo's stubbornness to therapeutic approaches can be caused at least partly by the many 'unheeded' environmental triggers that this commentary has endeavored to decipher. We also encourage further research on this interesting and very important topic.

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Quick Response Code:	Website: www.advbiores.net
	DOI: 10.4103/2277-9175.156653