GUEST EDITORIAL

Anaemia (part 2)

According to the World Health Organization, anaemia is defined as 'a condition in which the number of red blood cells or their oxygen-carrying capacity is insufficient to meet physiologic needs'.[1]

The number of anaemic people worldwide is estimated to be a staggering 2 billion, of whom ~50% have iron-deficiency anaemia (IDA).[1] Of major concern are the more serious consequences of IDA on the cognitive and physical development of children and the work productivity of adults. Furthermore, the increased risk of maternal, child and postoperative morbidity due to severe anaemia has been well documented.

Given the vastness of the subject, a systematic approach to diagnosis is crucial. In this series, anaemia has been divided into two causative categories, i.e.:

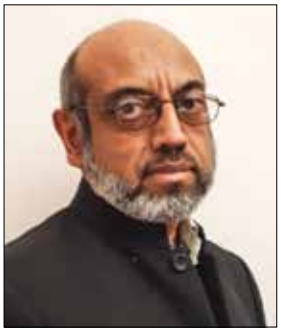
- Decreased bone marrow production or output of red cells, discussed in the previous issue of this CME series.[2]
- Peripheral loss (bleeding, sequestration or haemolysis) of red cells, which is the subject of discussion in the current issue.

Of the causes of anaemia due to peripheral loss of red cells, haemolytic anaemia represents the largest group. Understanding the pathophysiology of intra- and extravascular haemolysis is of paramount importance, as the clinical presentation and management of the two varieties differ in many respects.

Globally, the prevalence of inherited haemolytic anaemias (including thalassaemia, glucose-6-phosphate dehydrogenase deficiency, globin chain variants, e.g. sickle cell disease (SCD)), is highest in areas endemic to malaria. A striking overlap between the geographical distribution of inherited haemolytic anaemias and malaria endemicity led researchers to explore the effect of this co-existence. In vitro studies have demonstrated decreased parasite survival in the red cells of affected individuals (mostly heterozygous). These experiments led investigators to conclude that selective survival from partial protection against malaria has ensured persistence of these conditions. SCD serves as a classic example to illustrate the point. In some parts of Africa, the childhood mortality for the homozygous state (SS) exceeds 60%. As a consequence of such a severe clinical entity, the sickle gene is expected to dwindle to extinction as a result of its progressive dilution over subsequent generations. However, the sickle gene is anything but extinct, and is prevalent in polymorphic frequencies in some geographical regions. The primary reason for this observation is attributed to partial protection enjoyed by the heterozygous state against malaria, and it is this selective survival advantage that has led to persistence of the sickle gene. Various mechanisms for protection against malaria have been proposed, of which two are widely accepted: (*i*) K^+ is an absolute requirement for parasite surival. Sickle cells have decreased intracellular K^+ (from K^+ loss due to red cell membrane damage), thus compromising parasite survival; (*ii*) the intracellular pH in parasite-infected red cells drops, which triggers the sickling process. The parasitised sickle cells are then selectively removed by splenic macrophages.

Transformation of the South African (SA) political landscape after the first democratic election in 1994 occasioned relaxation of SA immigration laws. This led to an influx of visitors for business, leisure and employment purposes from various parts of the world. Consequently, healthcare workers are now exposed to inherited haemolytic conditions previously not highly prevalent in SA.

Physiological states of anaemia, e.g. pregnancy, are not included for discussion in this CME series.



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1. World Health Organization. The Global Prevalence of Anaemia in 2011. Geneva: WHO, 2015.
2. Alli N, Vaughan J, Patel M. Anaemia: Approach to dagnosis. S Afr Med J 2017;107(1):23-27. http://dx.doi.org/10.7196/SAMJ.2017.v107i1.12147

S Afr Med J 2017;107(2):95. DOI:10.7196/SAMJ.2017.v107i2.12224