The girl with the “saturation gap”

Sir,

A 21-year-old female presented with a complaint of dyspnea for the past 6 h. She was tachypneic and in obvious respiratory distress on inspection. Her oxygen saturation was 76% (at room air), pulse rate was 120/min, blood pressure was 150/90, and respiratory rate was 26/min. No adventitious sounds were heard on auscultation. Immediate resuscitative measures were started in the form oxygen supplementation and noninvasive ventilation support to decrease the respiratory distress. Bedside chest radiograph and echocardiography were within normal limits. She was a known case of mononeuritis multiplex and was on treatment with dapsone, methotrexate, and prednisolone apart from some other symptomatic medicines. Her complete blood count and urine examination were unyielding. Arterial blood gas (ABG) analysis was done which showed pH 7.48, $pO_2$ 268.1 (on oxygen), $pCO_2$ 24.5, bicarbonate levels as 18.9, $SpO_2$ as 96.3%. The blood withdrawn for ABG was brownish. Furthermore, seeing the discrepancy between $SaO_2$ (which was 96.3%) in ABG and $SpO_2$ (76%) in finger pulse oximetry, methemoglobinemia was suspected. A repeat ABG was done for methemoglobin (MetHb) levels, which revealed 28.2% MetHb. As the detailed history was being taken from the family members simultaneously, it was revealed that the girl had ingested around 10 tablets of dapsone a few hours back with intent of suicide. With a working diagnosis of dapsone-induced methemoglobinemia, she was managed with intravenous methylene blue 4 ml 8 hourly along with other resuscitative measures. Gradually, her MetHb levels reduced to 10.3% in the next few days along with improvement in serial ABG reports [Table 1]. She was started on antidepressants after having appropriate consultation with the psychiatrist. The patient was maintaining saturation 96% at room air on the 4th day (pulse oximetry) and was discharged in a satisfactory condition.

| Table 1: Serial arterial blood gas measurements |
|-----------------------------------------------|
| Parameter                                      | ABG at admission (on oxygen) | ABG at 6 h (on oxygen) | ABG on day 2 (on oxygen) | ABG at discharge (on room air) |
| pH                                            | 7.48                          | 7.44                   | 7.43                      | 7.42                           |
| $pO_2$                                         | 268.1                         | 101.6                  | 98.1                      | 97.4                           |
| $pCO_2$                                        | 24.5                          | 27.1                   | 31.2                      | 37.2                           |
| $HCO_3^-$                                      | 18.9                          | 20.7                   | 22.2                      | 23.4                           |
| Base excess                                    | -5.5                          | -4.5                   | -3.4                      | -1.8                           |
| $SO_2$ (ABG) (%)                               | 96                            | 99                     | 99                        | 96                             |
| $SpO_2$ (pulse oximetry) (%)                   | 76                            | 82                     | 94                        | 96                             |
| MetHb (%)                                      | 28.2                          | 24.4                   | 17.6                      | 10.2                           |

ABG: Arterial blood gas, MetHb: Methemoglobin
Letters to Editor

Dear Sir,

Hemoptysis is defined as the expectoration of blood from the lung parenchyma or airways. It is one of the most common and challenging symptoms encountered in clinical practice. Massive or recurrent hemoptysis is considered to be of major concern that troubles both patients and the treating physicians. Most physicians are facing difficulties in managing this symptom in routine clinical practice. Massive or recurrent hemoptysis is partly contributed by different causes of hemoptysis, with the common ones being active or healed pulmonary tuberculosis (TB), bronchiectasis, aspergilloma, lung abscess, and bronchogenic carcinoma. A systematic and timely management protocol needs to be established to focus on a more definitive treatment.

MetHb is formed when iron in the hemoglobin (Hb) changes from ferrous (Fe^{2+}) to ferric (Fe^{3+}) state. In healthy adults, 99% Hb is in Fe^{3+} state and remaining 1% is MetHb.[1] Increased fraction of MetHb impairs the oxygen-carrying capacity of Hb and hence leads to hypoxemia. MetHb concentration above 20% is associated with dizziness, fatigue, palpitations and above 40% is associated with arrhythmias, dyspnea, and seizures.[1] The diagnosis is usually made by a precipitating/trigger factor in history, and a typical “saturation gap” between oxygen saturation noted in pulse oximetry and ABG analysis.[2] Pulse oximetry can detect only oxyhemoglobin and deoxyhemoglobin. In the presence of MetHb, the oxygen saturation falls when detected via pulse oximetry but not when detected through ABG analysis. This saturation gap is very suggestive of the presence of an alternative form of Hb, especially MetHb.[2] In addition, the chocolate brown color of blood (detected while sampling) is again indicative of MetHb.[3] Apart from supportive care, specific treatment includes intravenous methylene blue 1–2 mg/kg body weight. In vivo, methylene blue is converted to leukomethylene blue which subsequently acts as an electron donor to MetHb and reduces its concentration.[4] Hemodialysis may be the last resort in refractory cases.[2] Methemoglobinemia is an uncommon side effect of dapsone treatment, incidence reported in around 5% of cases. In addition, other drugs, namely antimalarials, sulfonamides, and prilocaine are also reported to cause similar adverse outcomes which the clinicians should be aware of.[5]

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Ankita Chouksey, Alkesh Kumar Khurana, Abhishek Goyal

Department of Pulmonary Medicine, All India Institute of Medical Sciences, Bhopal, Madhya Pradesh, India
E-mail: lungcancer@rediffmail.com

REFERENCES
1. Ward KE, McCarthy MW. Dapsone-induced methemoglobinemia. Ann Pharmacother 1998;32:549-53.
2. Burke P, Jahangir K, Kolber MR. Dapsone-induced methemoglobinemia: Case of the blue lady. Can Fam Physician 2013;59:958-61.
3. Donnelly GB, Randlett D. Images in clinical medicine. Methemoglobinemia. N Engl J Med 2000;343:337.
4. Ashurst JV, Wasson MN, Hauger W, Fritz WT. Pathophysiologic mechanisms, diagnosis, and management of dapsone-induced methemoglobinemia. J Am Osteopath Assoc 2010;110:16-20.
5. Trindade MA, Careta MF. Acute methaemoglobinemia induced by dapsone case report in leprosy treatment. Hansenol Int 2008;31:31-4.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Access this article online
Quick Response Code:  
Website: www.lungindia.com
DOI: 10.4103/lungindia.lungindia_160_18

How to cite this article: Chouksey A, Khurana AK, Goyal A. The girl with the “saturation gap”. Lung India 2018;35:448-9.

© 2018 Indian Chest Society | Published by Wolters Kluwer - Medknow