

Flummoxed by Diamox

Sir,

A 39-year-old female, case of idiopathic intracranial hypertension, presented to the emergency medicine department with breathlessness and diplopia. She had a history of hyperreactive airway disease and had similar symptoms, 3 months back which was relieved by over-the-counter medications. The diplopia was present even on primary gaze. Ten days before the present episode, she had developed worsening diplopia, for which she was started on acetazolamide (Diamox) 500 mg twice daily by a neurologist. On admission, the patient had wheeze and the breathlessness was attributed to the bronchoconstriction.

Routine laboratories showed hypokalemia. Unexpectedly, the arterial blood gas analysis showed high anion gap acidosis [Table 1]. We were consulted for metabolic acidosis. While acetazolamide can cause metabolic acidosis in relatively modest doses,^[1] the anion gap is normal in acetazolamide-induced metabolic acidosis. The patient's albumin level was normal (4.5 g/dl) and the albumin correction was not necessary. Her renal function was normal and there was no hyperlactatemia. We considered the possibility of addition of an unmeasured anion to the circulation and quickly ruled out the known offenders such as salicylate and ethylene glycol. History was revisited and the patient revealed that she had taken a native medication called Pankaja Kasthuri, popular in this part of the country. The chemical composition of Pankaja Kasthuri is unknown. We hypothesize that the high anion gap could have possibly arisen from unmeasured anions in the native medication.

Both acetazolamide and the native medication were stopped and serial arterial blood gas analyses were done [Table 1].

Table 1: Arterial blood values

Parameter	Baseline	Day 1	Day 2	Day 3	Day 4	Day 5
pH	7.034	7.288	7.336	7.381	7.41	7.446
pCO ₂ (mmHg)	36.1	25.6	22.8	25.0	28.7	28.7
HCO ₃ (mmol/L)	9.2	11.9	11.9	14.5	17.8	19.5
AG (mmol/L)	30.4	20.7	21.4	17.9	17.8	16.3
Delta AG	1.24	0.72	0.78	0.62	0.94	0.96

AG: Anion gap

The patient was also treated with oral bicarbonate. Over a period of 4 days, the blood gas abnormalities and the hypokalemia gradually reversed. We calculated the delta anion gap (gap-gap) as

$$\text{Delta anion gap} = (\text{Anion Gap} - 12) / (24 - \text{HCO}_3)$$

In patients with high anion gap acidosis, the delta anion gap helps in detection of coexistent nonanion gap acidosis. If there is a coexistent nonanion gap acidosis, the reduction in bicarbonate is disproportionately more than the increase in anion gap.^[2] In such cases, the delta anion gap is <1. As we see in Table 1, the primary abnormality in day 1 is the high anion gap acidosis, but as the patient improved, the effect of acetazolamide (normal anion gap acidosis) predominated. Thus, we assume that the unmeasured anion in the circulation, which presumably came from the native medication, has a shorter half-life than acetazolamide.

Symptomatic acidosis with acetazolamide is not rare.^[3] However, high anion gap acidosis has not been described with acetazolamide and in our case was possibly induced by the concurrent intake of native medication. The same medication has been associated with hyperprolactinemia.^[4] As this native medication is commonly used in South India, endocrinologists should be aware of its adverse effects.

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.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Karthik Balachandran, Adlyne Reena Asirvatham, Shriraam Mahadevan

Department of Endocrinology, Sri Ramachandra Medical College and Research Institute, Chennai, Tamil Nadu, India

Address for correspondence:

Dr. Shriram Mahadevan,
Department of Endocrinology, Sri Ramachandra Medical College and Research
Institute, Porur, Chennai, Tamil Nadu, India.
E-mail: mshriram@gmail.com

REFERENCES

1. Venkatesha SL, Umamaheswara Rao GS. Metabolic acidosis and hyperventilation induced by acetazolamide in patients with central nervous system pathology. *Anesthesiology* 2000;93:1546-8.
2. Narins RG, Emmett M. Simple and mixed acid-base disorders: A practical approach. *Medicine (Baltimore)* 1980;59:161-87.
3. Heller I, Halevy J, Cohen S, Theodor E. Significant metabolic acidosis induced by acetazolamide. Not a rare complication. *Arch Intern Med* 1985;145:1815-7.
4. Kannan S, Mahadevan S, Sathya A, Sriram U. Prolactin and alternative medicines: A word of caution. *Trop Doct* 2008;38:195-6.

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