LETTER TO THE EDITOR

Minoxidil Poisoning: A Case of Refractory Shock with Remarkable ECG Changes

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Dear Editor,

Minoxidil is widely used as a topical medication for alopecia and has now become an easily available over-the-counter medication that is sold as a cosmetic.[1] However, it is a very potent vasodilator and is used to manage refractory hypertension.[2] Its consumption can lead to life-threatening complications.

A 12-year-old girl was brought to the Emergency Department by her two brothers with complaints of repeated vomiting with some fresh and altered blood. She gave the history of severe headaches involving both sides of the head and giddiness on standing up. There was no history of any pain abdomen or melena, visual disturbance, fever, breathlessness, or palpitation. She had no history of migraine or any cardiac ailment. Both she and her attendants denied a history of ingestion of any poison. On examination she had severe tachycardia (150/min), hypotension (70/38 mm Hg), and tachypnoea but no orthopnoea. She had cold clammy peripheries but there was no pallor or cyanosis. Her systemic examination was unremarkable.

Resuscitative measures were instituted with a rapid intravenous infusion of 1.5 l of normal saline. A Ryle's tube (RT) was inserted, and stomach wash given. The Ryle's tube aspirate was clear and had no abnormal odour. Despite fluid replacement, her hypotension and tachycardia persisted. Electrocardiogram (ECG) done on admission revealed ST segment elevation in aVR, ST depression in all leads and T wave inversion in precordial leads (Fig. 1). Her bedside 2D echo was normal.

Repeated interrogation of the attendants revealed that following an altercation at home, she had consumed 20 mL of Mincize from the cosmetic cupboard. The solution was Minoxidil 5%. A literature search was done for minoxidil poisoning and noradrenaline infusion was started with the aim of maintaining a systolic blood pressure (SBP) >100 mm Hg. Despite high doses of noradrenaline, she remained hypotensive. Her BP was eventually stabilized by adding vasopressin infusion for pressor support. After 12 hours of dual pressor therapy, BP stabilized and vasopressin was tapered off, but noradrenaline support was required for another 72 hour. Her ECG reverted to normal (Fig. 2). She remained asymptomatic thereafter and discharged after a week. Her ECG was normal on discharge (Fig. 3).

Minoxidil is absorbed from the gastrointestinal tract into the bloodstream and peak concentration is achieved within 1 hour of ingestion. Minoxidil must be metabolized to its active metabolite

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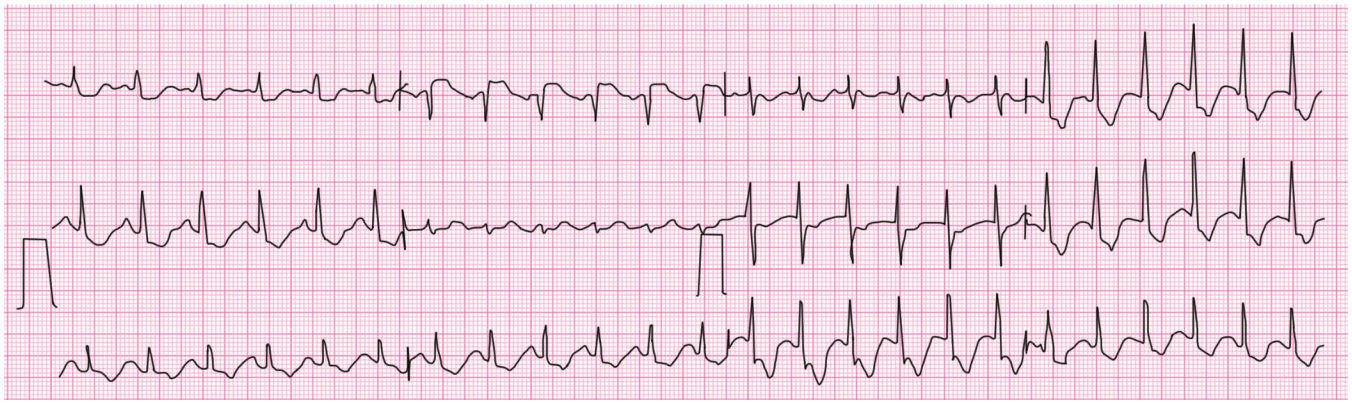

Fig. 1: ECG at time of admission

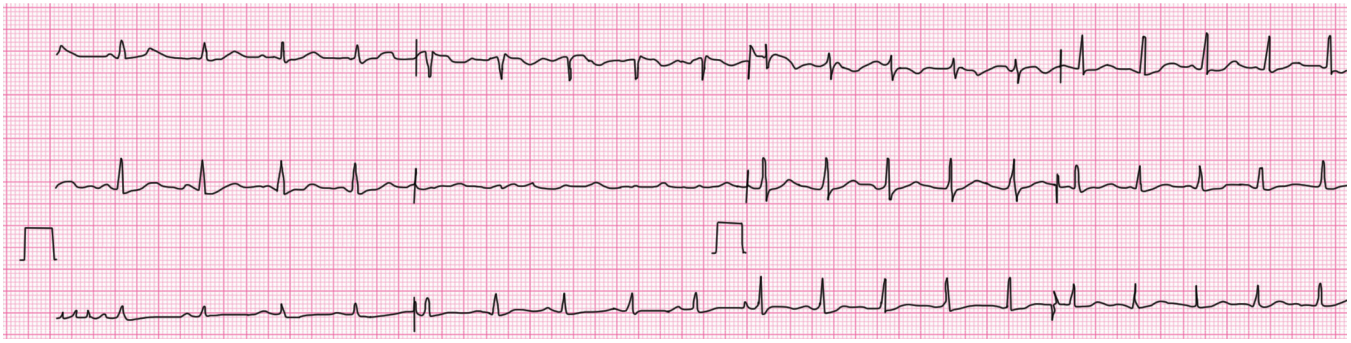

Fig. 2: ECG after 12 hrs when BP is stabilized

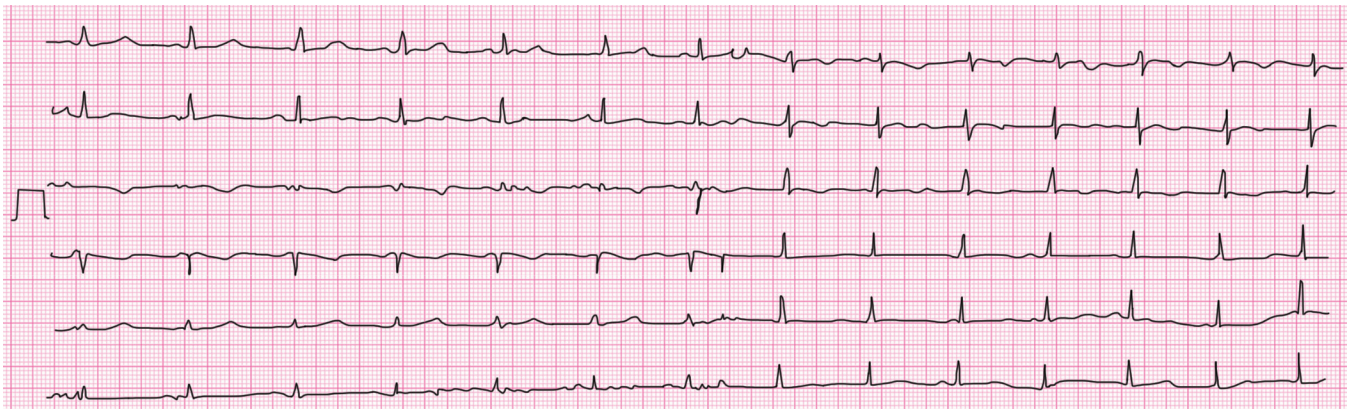

Fig. 3: ECG just prior to discharge

Minoxidil NO sulfate by hepatic sulfotransferase. Minoxidil sulfate activates the ATP-mediated K+ channel. The open K+ channels cause an efflux of K+ from the smooth muscle cells and lead to muscle hyperpolarization and relaxation. Its half-life is 3–4 hours but effects may be prolonged up to 72 hours due to accumulation at the site of action.[3]

Our patient has consumed 20 mL of 5% strength solution equivalent to 1000 mg which is 10 times higher than the maximum permissible oral dose. A review of the literature revealed that minoxidil toxicity manifests as severe hypotension and tachycardia. It presents within a few hours of oral ingestion. The treatment of minoxidil poisoning includes large volumes of intravenous fluids and vasopressor support.[4] We managed our patient with noradrenaline initially. We preferred it to Dopamine as it is less arrhythmogenic and causes lesser tachycardia than dopamine.[5] Due to the prolonged duration of the minoxidil effect, our patient required prolonged vasopressor support for almost 72 hours. Electrocardiogram changes associated with large dose Minoxidil ingestion are similar to that caused by some other drugs that activate the K+ channel. These changes usually normalize with time.

This case highlights the importance of reviewing history when the clinical findings cannot be easily explained. Prior good health should always raise doubt about deliberate self-harm. The availability of potent medications as cosmetics and without black-box warnings raises the risk of such poisoning. There are not too many cases of minoxidil poisoning reported. The high dose ingested by this child led to profound sustained hypotension and the complete recovery was satisfying.

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References

1. Spindler JR. The safety of topical minoxidil solution in the treatment of male pattern baldness; the results of a 27-center trial. Clin Dermatol 1998;6(4):200–212. DOI: 10.1016/0738-081x(88)90088-0.
2. Pettinger WA. Minoxidil and the treatment of severe hypertension. N Engl J Med 1980;303(16):902–906. DOI: 10.1056/NEJM198010163031607.
3. Brunton LL, Chabner BA, Knollmann BC. Goodman & Gilman's the Pharmacological Basis of Therapeutics, 12th edition. New York: McGraw-Hill Companies, Inc.; 2011. pp. 781.
4. Rose SR, Tomaszewski C. Evaluation of oral and topical minoxidil exposures reported to US Poison Centres from 1985–1991. Drug Investig 1994;8(2):81–94. DOI: https://doi.org/10.1007/BF03257431.
5. Shenoy S, Ganesh A, Rishi A, Doshi V, Lankala S, Molnar J, et al. Dopamine versus norepinephrine in septic shock: a meta-analysis. Crit Care 2011;15(suppl 1): P89. DOI: 10.1186/cc9509.