Glibenclamide: A second wind for refractory hyperkalemia

Sir,

In the clinical case reported by Sodhi *et al*.[1] the authors have discussed heparin-induced hyperkalemia, a less known problem,[2] and expressed the nonresponsiveness to conventional antihyperkalemic measures. Under such circumstances, we would like to recall the role of sulfonylurea inhibitor glibenclamide[3] in the reversal of refractory hyperkalemia.

This is a novel approach that succeeded remarkably despite failure of all conventional treatments and it accords with interpretation of blockade of excessive K_{ATP} channel activity. The K_{ATP} channels are composed of two components, an inwardly rectifying potassium channel (Kir) pore subunit and the regulatory sulfonylurea-receptor (SUR). K_{ATP} channels are present in various tissues, including the pancreas, kidney, heart, skeletal muscle and vascular smooth muscle, and it opens in response to hypoxia, metabolic acidosis, and hypercapnia. However, glibenclamide could close the K_{ATP} channels after binding to SURs.[4] Thereby, it prevents the exit of potassium from intracellular to extracellular. Based on the aforementioned mechanisms, one may speculate that glibenclamide may be an alternative for those with refectory hyperkalemia.

When no other alternatives are available, naturally one may try to use a well-established and time honored therapeutic molecule glibenclamide. However, more studies/reports are warranted to establish the usefulness of glibenclamide in refractory hyperkalemia. Another aspect is that cardioversion may not be effective in the presence of hyperkalemia. Despite all, the issues related to refractory hyperkalemia are

less discussed and rarely considered in education and practice. Therefore, practitioners and emergency physicians shall be made aware of off label use of glibenclamide, while handling a case of refectory refractory hyperkalemia.

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References

1. Sodhi K, Garg S, Singh B, Shrivastava A, Singla MK. Refractory hyperkalemia related to heparin abuse. Indian J Crit Care Med 2013;17:388-91.
2. Senthilkumaran S, Balamurgan N, Karthikeyan V, Azhagunatesan K. Heparin induced hyperkalemia-forgotten focus. J Gen Med India 2009;21:14-6.
3. Singer M, Coluzzi F, O'Brien A, Clapp LH. Reversal of life-threatening, drug-related potassium-channel syndrome by glibenclamide. Lancet 2005;365:1873-5.
4. Quayle JM, Nelson MT, Standen NB. ATP-sensitive and inwardly rectifying potassium channels in smooth muscle. Physiol Rev 1997;77:1165-232.

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