LETTER TO THE EDITOR

4-Aminopyridine as a life-saving treatment in calcium channel antagonist intoxication

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To the Editor,

With interest we read the article written by Rietjens et al. concerning practical recommendations for calcium channel antagonist poisoning.[1] The authors give a nice overview of the pathophysiology and the treatment options. However, one potential life-saving option was not mentioned. In 2007 we published a case report on the use of 4-aminopyridine (fampridine) treatment in case of calcium channel poisoning.[2] We described a case of amlodipine intoxication but this substance has also been used in verapamil intoxication.[3-5] 4-Aminopyridine inhibits different types of potassium channels (G-protein coupled potassium channels, ATP-sensitive potassium channel, Na+ -activated potassium channel[6]). This blocking action causes a slight depolarisation, thereby opening Na+ and subsequently calcium channels. In particular, the Na+ influx can elicit a rise in cytosolic Ca2+ concentration by inhibiting the Na+/Ca2+ exchanger, which under physiological conditions removes Ca2+ out of the cell driven by the Na+ gradient. 4-Aminopyridine-mediated Na+ influx will decrease the Na+ gradient and thereby decrease the driving force for this Ca2+-extruding Na+/Ca2+ exchanger. Therefore, in calcium entry blocker overdose, 4-aminopyridine can increase the cytosolic Ca2+ concentration very efficiently independent of the calcium channels.

In addition, variability exists between calcium entry blockers as intoxication with diltiazem can usually be treated with calcium infusion and we do not advise 4-aminopyridine in this type of intoxication. Any differentiation in the type of calcium entry blocker is lacking in the paper by Rietjens et al.

In conclusion, we think that the 4-aminopyridine-treatment option deserves a place in a review concerning this topic.

DISCLOSURES

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