We read the recent paper by Udriste et al. [1] with interest and noted that this patient was found to have a type 1 Brugada ECG pattern induced in the context of aluminium phosphide fatal poisoning. This is an intriguing case that provides an opportunity for further investigation regarding the effects of phosgene on myocardial sodium channel function.

We have been publishing extensively on the subject of Brugada Phenocopies (BrP) [2-6] and their clinical distinction from true congenital Brugada Syndrome (BrS) [7]. As noted by the authors [1], Brugada ECG patterns have been previously associated with toxic aluminium phosphide levels [8]; however, the exact pathophysiological mechanisms of the ECG manifestations have yet to be elucidated. To our knowledge, there have been no reports on the effects of phosphine on myocardial sodium channel function [8]; however, alterations in myocardial sodium permeability due to myocardial necrosis have been suggested [9]. Given the paucity of data, it seems more likely that toxic phosphine levels induce myocardial necrosis and associated sodium channel dysfunction thereby eliciting a BrP, rather than unmasking true congenital BrS; this however remains speculative.

This case would suggest that aluminium phosphide toxicity be considered a possible cause of BrP falling under the ‘Miscellaneous’ category in our etiological classification system [10]. This case cannot be definitely diagnosed as a BrP since the patient cannot undergo provocative testing with a sodium channel blocker. We recommend that in future cases where a patient survives aluminium phosphide poisoning and has an associated Brugada ECG pattern, the patient should undergo provocative testing with a sodium channel blocker to rule out congenital sodium channel dysfunction once clinically stable.

REFERENCES: