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Letter to the Editor

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A fatal adverse event upon adenotonsillectomy in a child. Are Brugada syndrome and propofol real accomplices?

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Dear Editor,

We read with great interest the article with the title "Post-operative Brugada electrocardiographic pattern, polymorphic ventricular tachycardia, and sudden death in a child after administration of propofol anaesthesia".[1] They alluded that a fatal ventricular arrhythmia was induced by propofol in a patient with Brugada syndrome. Imputability analysis can be done using different methods, including expert judgement, the World Health Organisation, or another algorithmic method.[2,3] Concerning the expert judgement, it is crucial to understand what the actual cause of unresponsiveness was. Hypoxia, due to obstructive airway, respiratory depression, or both, rather than a primary arrhythmia, could be a culprit. Naloxone administration implies opioid overdose with symptoms of respiratory and consciousness deterioration. Due to its shorter duration of action than morphine, reoccurrence of the initial symptoms is a caveat. Accordingly, the American Academy of Otolaryngology – Head and Neck Surgery Foundation strongly recommends against administering or prescribing codeine after tonsillectomy in children younger than 12 years.[4] This child received morphine. Besides, post-adenotonsillectomy haemorrhage is a well-known severe complication that might initially have caused or contributed to an obstructive airway[5] but may have played a role when disseminated intravascular coagulopathy was presumed.

Although the report states that at least 12 minutes elapsed before resuscitation, the total time of unresponsiveness and hypoxia remain unclear. The electrocardiographic pattern, as already described in cases of Brugada syndrome phenocopy,[6] could be related to myocardial ischaemia, which was supported by the echocardiographic findings. Another potential trigger not excluded is electrolyte imbalance.

If the electrocardiogram was a diagnostic finding of an unidentified Brugada syndrome, one might wonder why a single propofol dose induced a malignant arrhythmia only almost 6 hours later. From a pharmacokinetic point of view, this seems very unlikely.

Though afebrile during hospitalisation, it is plausible that post-operative fever was developing when discharged. Fever, an altered autonomic state (abolishment of the adrenergic drive post-operatively, or by falling asleep in the car) in patients with Brugada syndrome, has been reported to trigger malignant arrhythmias.[7]

Causality assessment for propofol according to the structured expert judgement method is considered as "*Unlikely*" because of the time to drug intake makes a relationship improbable and disease or other drugs provide plausible explanations. When using Kramer's algorithm – one of the best-performing methods in the literature – causality is also categorised as "*Unlikely*" because of no dose-dependent relationship and other conditions potentially clarifying the event. Finally, we did not include propofol infusion syndrome in the structured imputability analysis, since one dose of propofol was administered.[8,9]

Concluding, we also support a 12-lead preoperative screening, in a subset of children. Nevertheless, the conclusion of "a small risk of fatal ventricular arrhythmias in the post-operative period in patients who receive propofol anaesthesia" is not confirmed by prospective clinical trials.[10,11]

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Conflicts of Interest. None.

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