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LETTER TO THE EDITOR

Risk of endotoxemia associated with clinical incidence of contaminated propofol

Dear Editor,

I read the interesting short communication of Shy-Hong and colleagues, in which they confirmed, in a different way than usual, the infectious risks for endotoxemia associated with propofol contamination. They did this through the report of four consecutive patients.[1] I would like to add further clinical information and epidemiological outcomes, which the authors did not consider, but which I consider are important to aid the discussion about this topic that probably represents a public health problem in many countries.

Although there are multiple preservative substances that are added to propofol with the objective of reducing infectious risks and reducing probiotic characteristics, it is important to have careful management and to use the manipulation guidance given by most manufacturers. Since the introduction of propofol by the US Food and Drug Administration in 1989, it has been implicated in several outbreaks of postoperative sepsis, even including many fatalities. Furthermore, many studies have confirmed the involvement of propofol contamination in bacterial growth and its support of endotoxin production. There is clinical evidence of fungal infections originating after propofol infusions (relative risk = 8.8, $p = 0.048$), and also by bad conditions during its administration.[2] In 1999, Henry et al conducted a case–control study, in which they concluded that the infusion of propofol is an important risk factor for infections (odds ratio = 22, 95% confidence interval = 2.1–550).[3] Other studies have mentioned the incidence of ampules with contaminated propofol. Webb et al retrospectively obtained a value of 5.6% (18/302) infected ampules in the intensive care unit.[4] However, the results exposed by Shy-Hong and colleagues have already been mentioned in other *in vitro* studies. Arduino and colleagues measured endotoxin concentration of four Gram-negative bacteria (isolated from outbreak cases and laboratory stock cultures) incubated at 30 °C using the *Limulus* amebocyte gel-clot assay method. They concluded that there was strong endotoxin production postinoculation in propofol, increasing the concentration from 0 ng/mL to 14.72 ng/mL after 72 hours in the most extreme case of *Enterobacter cloacae*.[5]

Finally, I would like to congratulate the effort of the authors on this line of investigation and I would also like to encourage the development of new studies *in vivo* to analyze the association of propofol in postoperative infections.

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