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LETTER TO THE EDITOR

Safe use of landiolol hydrochloride in a patient with marked pseudocholinesterase deficiency

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To the Editor:

Landiolol hydrochloride is an ultrashort-acting beta-blocking agent with potent beta-1 receptor selectivity, as much as 250 times greater than its beta-2 receptor selectivity. It is widely used in treating patients with supraventricular tachyarrhythmias. Because landiolol hydrochloride is metabolized in part by pseudocholinesterase, its action may be enhanced or diminished in patients with pseudocholinesterase deficiencies or variants [1]. We describe a case demonstrating the safe use of landiolol hydrochloride in a patient with marked pseudocholinesterase deficiency.

An 82-year-old man underwent ventriculoperitoneal shunt placement for normal pressure hydrocephalus. Past medical history included prolonged mechanical ventilation after general anesthesia as a consequence of pseudocholinesterase deficiency. Laboratory data showed a low level of plasma cholinesterase, at 5 IU/l (normal range, 100–240 IU/l). Other laboratory data were within normal limits, except for mild hypoalbuminemia and leukocytosis. General anesthesia was induced with fentanyl and propofol. Tracheal intubation was facilitated with vecuronium. Anesthesia was maintained with sevoflurane and fentanyl. The intraoperative course was unremarkable except for an abrupt increase in blood pressure (BP) and heart rate (HR) to 150/90 mmHg and 100 beats per minute (bpm), respectively, after the skin incision. Fentanyl 100 μg, landiolol hydrochloride 10 mg, and nicardipine 1 mg were administered, resulting in BP 110/60 mmHg and HR 70 bpm. Surgery was completed, and his vital signs remained stable. Postoperatively, there were no significant changes in the vital signs.

Landiolol hydrochloride has an ester bond in its chemical structure. It is metabolized rapidly by pseudocholinesterase in the plasma and carboxyesterase in the liver, with a half-life of 4.1 min [2]. However, pseudocholinesterase is one of the enzymes that hydrolyzes choline ester structures and is found mainly in plasma but also in the liver, smooth muscle, intestines, pancreas, heart, and brain. Pseudocholinesterase is also known to metabolize succinylcholine, mivacurium, chloroprocaine, tetracaine, cocaine, heroin, and other drugs [3].

This patient's anesthesia history suggested some deficiency in metabolism, although there was no definitive evidence of a variant pseudocholinesterase or its deficiency. Low plasma levels of pseudocholinesterase can indicate various conditions including liver cirrhosis, hepatic tumor, malnutrition, and organophosphate intoxication, but these conditions can usually be excluded by a careful medical history and review of laboratory data.

The same substrate can be hydrolyzed by different esterases [4]. As we already noted, landiolol hydrochloride is metabolized not only in the blood but also in the liver. Thus, the effect of landiolol hydrochloride in a patient with variant pseudocholinesterase may be unpredictable. It has been reported that remifentanil in a patient with pseudocholinesterase deficiency was safe [5], whereas procaine in those patients provoked seizures, although remifentanil and procaine are both metabolized by pseudocholinesterase [1]. To our knowledge, there are no clinical reports describing the use of landiolol hydrochloride in a patient with very low levels of plasma pseudocholinesterase, including pseudocholinesterase deficiencies or its variants. Although

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this case demonstrates that the use of landiolol hydrochloride did not result in significant bradycardia, caution must be exercised when landiolol hydrochloride is used in patients with variant pseudocholinesterase or its deficiency.

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