

Additive effect of propofol for attenuation of hypertension in a patient with undiagnosed pheochromocytoma

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Pheochromocytomas are rare, mostly benign catecholamine-producing tumours of chromaffin cells of the adrenal medulla or of a paraganglion associated with a high cardiovascular morbidity and mortality if left untreated. During surgery, massive release of catecholamines can result in potentially fatal hypertensive crises and cardiac arrhythmias [1]. Management of patients with pheochromocytoma remains a challenge for the anaesthesiologist with regard to the maintenance of anaesthesia and control of hypertensive crises during surgery [2]. We describe the role of propofol in the treatment of a patient with severe hypertension who was clinically diagnosed during the operation.

A 53-yr-old female (153 cm, 70 kg, ASA II) presented for the removal of a mass located in the retroperitoneal region between the aorta and inferior vena cava. Her past medical history revealed well-regulated hypertension for about 14 yr. After standard non-invasive monitoring, general anaesthesia consisted of induction with propofol 2.5 mg kg^{-1} , fentanyl $2 \mu\text{g kg}^{-1}$ and rocuronium 0.6 mg kg^{-1} followed by intubation. Fifteen minutes after incision, the blood pressure (BP) started to rise (180/110 mmHg). The concentration of isoflurane was increased and additional fentanyl (100 μg) and nitroglycerine (100 μg) were administered. After monitoring the radial artery and central venous pressure (CVP), a nitroglycerine infusion was started. Infusion rates rapidly increased and hence diltiazem 25 mg (with 5 mg increments), metoprolol 5 mg (with 1 mg increments) and esmolol 40 mg were given over 10 min. During the surgical procedure, a serious hypertension attack (240/150 mmHg) developed in the course of the palpation of the mass and a nitroprusside infusion was promptly started as the mass was considered to be an atypically located pheochromocytoma. Surgical manipulation was discontinued.

Despite all medications including nitroglycerine ($4 \mu\text{g kg}^{-1} \text{ min}^{-1}$), nitroprusside ($2 \mu\text{g kg}^{-1} \text{ min}^{-1}$)

infusions and increased isoflurane concentration (3%), satisfactory control of BP was not obtained. Intravenous (i.v.) propofol administration was started at a dose of 1 mg kg^{-1} bolus and $6 \text{ mg kg}^{-1} \text{ h}^{-1}$ infusion. After 5 min, BP markedly decreased within normal ranges and the doses of propofol, nitroglycerine and nitroprusside were reduced to $3 \text{ mg kg}^{-1} \text{ h}^{-1}$, $2 \mu\text{g kg}^{-1} \text{ min}^{-1}$ and $0.5 \mu\text{g kg}^{-1} \text{ min}^{-1}$, respectively. Surgery was allowed to continue and the mass was totally removed. After removal of the mass, the vasodilators were discontinued. During the administration of propofol anaesthesia, fluid infusion was increased (3000 mL crystalloid, 1000 mL colloid) using the CVP as a guide. Subsequently, dopamine in a dose of $5 \mu\text{g kg}^{-1} \text{ min}^{-1}$ was started and continued for 2 h in the postoperative period.

At the end of the operation, the patient was extubated without any problem. She was monitored in the ICU overnight and discharged from the hospital on day 6. Examination of the mass confirmed it to be a pheochromocytoma.

There have been numerous case reports of anaesthetic management in patients with a pheochromocytoma but only a few were undiagnosed cases before operation [2,3]. Because the location of these tumours may be in extra-adrenal regions including sympathetic ganglia, carotid body and aortic chemoreceptors, diagnosis in the preoperative period is not always possible. The most commonly used anaesthetic for planned surgical procedures in cases with pheochromocytoma is usually propofol [4,5]. Inhalational-based anaesthesia was planned in our case. Vasoactive drugs such as non-selective α - and β -antagonists, calcium-channel-blocking agents, nitroglycerine and nitroprusside can be used for the control of hypertension in these patients [1]. In our patient, although we administered high doses of vasodilators and inhalational agents, it was difficult to control hypertensive crises. The vasodilator property of inhalation agent, during hypertension was also considered to be hazardous in terms of intracranial hypertension. Therefore, anaesthesia was changed to propofol infusion in terms of its useful effect on systemic arterial pressure and indirectly on intracranial pressure control. Propofol causes a decrease in arterial pressure owing to a drop in systemic vascular resistance (inhibition of

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sympathetic activity), cardiac contractility and preload [6]. Immediately after propofol infusion with the combination of the vasodilatory drugs, satisfactory control of arterial tension was provided for the remaining period of operation. In conclusion, this case suggests that the administration of propofol should be kept in mind for the control of serious intraoperative hypertensive attacks when vasodilator treatment under inhalation-based anaesthesia is inadequate.

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