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HAEMODYNAMIC EFFECTS OF ANAESTHESIA IN A PATIENT CHRONICALLY TREATED WITH ANGIOTENSIN - CONVERTING ENZYME INHIBITORS

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A 55 year old controlled hypertensive patient, on treatment with captopril, was subjected to general anaesthesia using conventional anaesthetics. The patient had two episodes of severe hypotension and bradycardia intra-operatively which responded to intravenous crystalloids and atropine. Possible mechanism of action for such haemodynamic instability during general anaesthesia and relevant literature has been reviewed. Bahrain Med Bull 1995;17(4):

Angiotensin Converting Enzyme Inhibitors (ACEI) are extensively used in the treatment of cardiovascular disease and it is well accepted that their therapeutic action is mainly mediated by inhibiting angiotensin II formation[1-3]. However the range of their therapeutic indications is increasing, whether prior plasma activity is increased or not[1,3]. Thus the number of patients chronically treated with ACEI and scheduled for surgery will likely increase[4]. Prevailing view holds that angiotensin II may contribute to haemodynamic regulation during anaesthesia[5,6]. However, some recent reports have warned of episodes of profound hypotension and bradycardia during anaesthesia in patients treated with ACEI and some authors have advocated discontinuing ACEI treatment preoperatively[7-9]. We report a case treated with ACEI, who had severe haemodynamic instability during general anaesthesia.

THE CASE

A 55 year old male, weighing 65 Kg, was admitted to the Postgraduate Institute of Medical Education and Research, Chandigarh, India for enucleation of a dentigerous cyst. His past history was unremarkable except that he had bilateral renal stones and was a known case of hypertension for 3 years. Drug treatment included Captopril 75 mg daily in divided doses.

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The patient had preoperative blood pressure of 150/90 mm Hg and pulse rate of 72 beats per minute. Preoperative serum electrolytes and haematocrit values were within normal limits. ECG and X-ray chest revealed no abnormality.

Premedication consisted of oral Diazepam 10 mg the night before and 5 mg on the morning of surgery. In addition he received intramuscular Pethidine 75 mg and Promethazine 25 mg an hour before induction of anaesthesia. Antihypertensive drug was continued till the morning of surgery. Anaesthesia was induced with Thiopentone 300 mg and Vecuronium 7 mg given intravenously over a period of approximately 90 seconds. In addition, the patient also received intravenous Lignocaine 70 mg. The trachea was intubated using 9 mm cuffed red rubber endotracheal tube. Anaesthesia was maintained with 33% O2 in N2O and Morphine 7.5 mg i.v. The lungs were ventilated using Bain Anaesthetic Breathing System with a fresh gas flow of 8 litres per minute.

Immediately after the induction of anaesthesia the heart rate suddenly dropped to 36 beats/minute and systolic arterial pressure (SAP) to 50 mm Hg. The patient was infused with 500 ml of Ringer-lactate over a period of 5 minutes and was also administered Atropine 0.6 mg iv, to which he responded and the heart rate increased to 60 beats per minute and SAP to 100 mm Hg. Fifteen minutes after induction the SAP increased to 130 mm Hg and the heart rate was maintained between 60-68 beats per minute. The patient had a second episode of bradycardia and hypotension 25 minutes after induction of anaesthesia, which again responded to i.v Atropine and rapid infusion of Ringer lactate. Subsequent course of anaesthesia was uneventful and total duration of surgery was 75 minutes. At the end of surgery the residual effect of Vecuronium was reversed with a mixture of Atropine 1.2 mg and Neostigmine 2.5 mg. Postoperatively, the patient received 40% oxygen through mask and his heart rate and arterial pressure were maintained near preoperative values. Further course of his hospital stay was uneventful.

DISCUSSION

By far the most common use of ACEI in the perioperative period comes as part of the oral antihypertensive regimen of the patients. As with other antihypertensive agents, the current consensus regarding ACEI therapy perioperatively is to continue all such drugs until surgery and to restart all such drugs as soon as possible postoperatively. Although most clinicians agree with this, there is a concern regarding the potential haemodynamic instability with perioperative administration of ACEI. Recently, prolonged hypotension has been reported in patients undergoing general anaesthesia for minor surgery in whom ACEI were given before surgery[9].

Captopril, an ACEI, reduces blood pressure by lowering total peripheral resistance, with little change in the heart rate[10]. The absence of reflex tachycardia with ACEI administration, unlike the increase in heart rate seen with other drugs that decrease afterload, has yet to be adequately explained, although enhancement of parasympathetic activity (or disinhibition) and drug induced venodilatory action have been suggested [11,12]. Captopril was preferred in our patient for control of blood pressure as this patient had bilateral renal stones and ACEI prevent deterioration of renal functions in patients with chronic renal disease[4].

This case demonstrates that hypertensive patients chronically treated with ACEI can have severe haemodynamic instability during induction of anaesthesia. Both hypotension and bradycardia however, could be easily treated with administration of intravenous crystalloids and atropine. Other authors have also reported marked decrease in blood pressure during induction of anaesthesia in hypertensive patients chronically treated with ACEI[13]. As the arterial blood pressure of ACEI treated patients is volume dependant, the decrease in arterial blood pressure could therefore be easily treated with intravenous fluid administration, as in our patient, and this confirms the role of intravascular volume dependence of arterial blood pressure in patients treated with ACEI, as reported by Mimran and Ribstein[14]. When hypotension occurred in our patient instead of tachycardia, a decrease in heart rate was observed. An increase in parasympathetic tone especially in chronic treatment with ACEI has been described[15]. The increase in heart rate after atropine possibly confirms this view.

CONCLUSION

Increased parasympathetic tone and significant dependence on intravascular volume account for the haemodynamic instability during induction of anaesthesia in hypertensive patients chronically treated with ACEI. Anaesthetists should preferably avoid the use of drugs, which themselves can cause bradycardia, and

also should give proper attention to adequate volume replacement and monitoring in such patients during the course of anaesthesia.

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