

Severe postoperative blood pressure instability in a patient with long standing Parkinson's disease

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INTRODUCTION

Parkinson's disease (PD) is a common neurological disorder. The etiology of PD is unknown but several causes are hypothesized including genetic, environmental and infectious factors. The single consistent risk factor is age (1). Although tremor and muscle rigidity are the most prominent, circulatory autonomic dysregulation is also common in PD. Known cardiovascular abnormalities include orthostatic hypotension by a decrease in baroreceptor reflex sensitivity, life-threatening cardiac arrhythmias and instability of arterial blood pressure (2, 3). We present a case of pronounced and rapid blood pressure fluctuations after spine surgery in a patient with longstanding PD and levodopa treatment. To our knowledge there are no previous reports of such unusual hemodynamic disturbances after surgery in patients with Parkinson's disease.

CASE

A 60-year-old man presented with recent neurogenic claudication and progressive paraparesis due to severe scoliosis with multiple level lumbar stenosis. The patient was scheduled for multi-level laminectomy with additional pedicle screw fixation Th12 to S2.

He had been suffering from PD for 26 years. He manifested with fluctuations of motor nerve disturbances, which progressed to severe immobility whenever the effects of levodopa wore off (on-off phenomenon). In the past he complained of collapse or near-collapse due to orthostatic hypotension. His medication consisted of the oral administration of levodopa/benserazide 100 mg/25 mg and levodopa/carbidopa/entacapone 100 mg/25 mg/200 mg six times daily. His preoperative blood pressure was 95/50 mmHg. Otherwise no

cardiopulmonary disabilities were known. He underwent an uncomplicated inguinal hernia repair under general anesthesia ten years before.

Oral anti-PD medication was continued until 2 hours preoperatively. On arrival in the operating theatre, he showed neither muscle rigidity nor resting tremor.

Anesthesia was induced at eight o'clock with fentanyl 250 µg, propofol 150 mg and rocuronium 50 mg to facilitate tracheal intubation.

Anesthesia was maintained with continuous sevoflurane (mean end tidal concentration 1.8 vol%) and remifentanyl 0.12 µg/kg/min.

The patient was placed in the prone position on a Jackson spine table. Cefazoline 1 g was given as antibiotic prophylaxis and repeated every four hours. Oral levodopa therapy was omitted during surgery.

During surgery intra-arterial blood pressure (IABP) was on average 110 mm Hg systolic and 50 mm Hg diastolic with phenylephrine on continuous infusion of 0.2 µg/kg/min. Blood loss was 2500 ml. Intravenous fluid therapy consisted of 6500 ml crystalloids, 1500 ml colloids and 1550 ml cell saver processed blood. Diuresis was 750 ml.

After seven hours of surgery arterial blood pressure suddenly dropped to systolic pressures of 50 mm Hg, not responding to extra phenylephrine 0.1 mg or ephedrine 5 mg. Assuming an anaphylactic reaction to a repeated dose of cefazoline, clemastine (antihistaminic drug) 1 mg and hydrocortisone 100 mg were administered intravenously.

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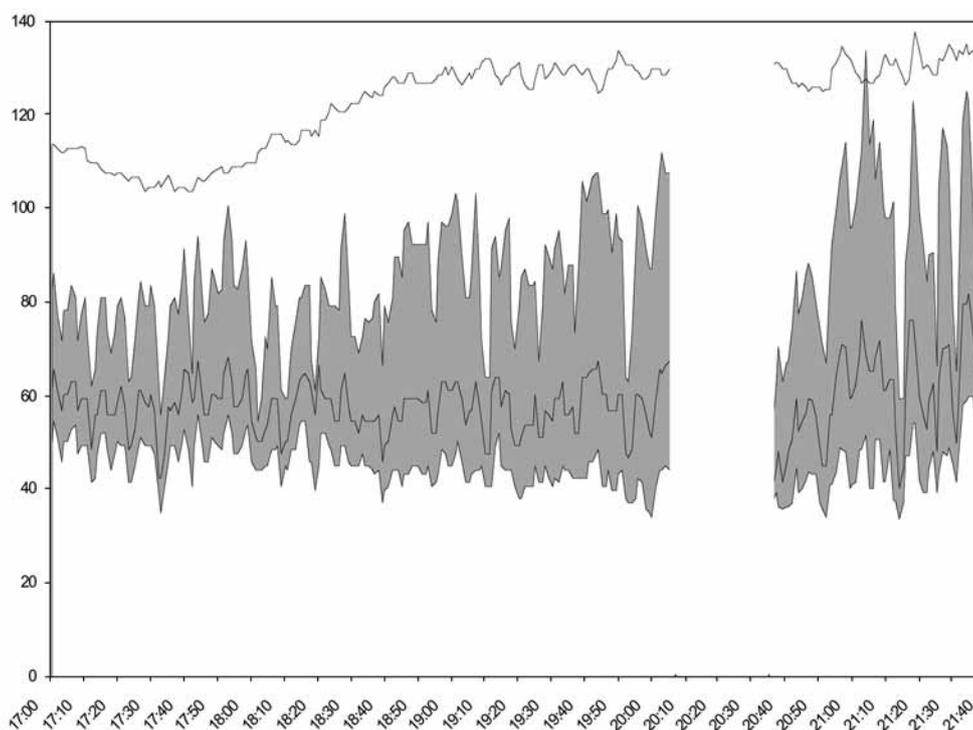


Fig. 1. — Blood pressure curve. Note the marked and rapid fluctuations in blood pressure

The arterial blood pressure responded eventually to an intravenous injection of 50 μg epinephrine. Surgery was completed, the emergence from anesthesia was uneventful and the patient was extubated in the operating theatre. The patient was transferred awake to the Intensive Care Unit. In the ICU the patient was hypotensive and tachycardic as shown in figure 1.

Besides this hypotension he had recurrent episodes of even deeper hypotension, with systolic blood pressures of 55 mmHg, which lasted every time for three to four minutes and always ended spontaneously. The patient remained conscious during all episodes of hypotension. Respiration was adequate. His hands showed only a slight tremor. A state of volume depletion was assumed and the patient was treated by intravenous volume replacement with 9 liters of crystalloids over the next 12 hours and infusion of low dose norepinephrine. His oral medication for PD was restarted three hours postoperatively. He received his regular dose of levodopa/benserazide 100 mg/25 mg and levodopa/carbidopa/entacapone 100 mg/25 mg/200 mg orally. While the hypotension wearied of slowly the blood pressure oscillations kept occurring until the next morning. The patient could then be transferred to the ward in a normal cardiovascular state.

Four weeks later, the patient developed pain in both arms and legs with progressive tetraparesis. Magnetic Resonance Imaging of the cervical spine showed signs of spondylodiscitis C6 to C7 with epidural abscess and spinal cord compression. The patient underwent an anterior cervical decompression with evacuation of the abscess under general anesthesia. This procedure lasted four hours and blood loss was minimal. PD medication was continued sharply and no blood pressure fluctuations were noticed. Nevertheless the patient's general and neurological condition worsened over the next week and the patient died in the hospital five weeks after the first operation.

DISCUSSION

This patient suffered from PD with severe motor symptoms, on-off phenomenon and orthostatic hypotension. During a period of hypovolemic hypotension after long lasting and complex spinal surgery a remarkable circulatory instability with rapid fluctuation of blood pressure manifested. This kind of circulatory instability has not been described before. The situation suggested an autonomous switch that was turned on and off

every few minutes. Several possible explanations to elucidate the hypotension and remarkable spontaneous recovery were analyzed.

First, as mentioned, we thought of an anaphylactic reaction to cefazoline. Although the patient received cefazoline without untoward effect earlier during the procedure, it is well known that anaphylaxis can occur. Moreover, the blood pressure restored at first after antihistaminic medication, steroids and epinephrine. However, the recurrence of the episodes of hypotension postoperatively not responding to norepinephrine brought us to reject this explanation.

Second, in perioperative care tachycardia and hypotension point at hypovolemia until the opposite has been proven. Although his fluid balance was largely positive at the end of the surgical procedure as well as during the stay in the ICU, hypovolemia must be considered as a trigger for his adrenergic system to be active at a maximum.

This brings us to the third and speculative explanation. There are only a few reports available on hemodynamic instability in Parkinson's disease. The best documented abnormality is orthostatic hypotension (3, 4). Orthostatic hypotension is a common finding in the later stages of Parkinson's disease (3). Also a lowered cardiac sympathetic nerve performance has been found in patients with Parkinson's disease. This was shown in the study of Nakamura et al who describe a significant lower response during exercise (5).

The abrupt onset and oscillating character of the instability almost instinctively made us assume an "end of levodopa dose" phenomenon. After all, we had not been able to continue oral or intravenous levodopa therapy during surgery. Although end of dose symptoms are described especially for the motoric manifestations of PD, levodopamine is known to have an effect on the autonomous nervous system as well. This effect of levodopamine on the blood pressure is far from elucidated and strongly depends on the dose and the period that the patient is on levodopa therapy. The most commonly

mentioned cardiovascular side effect is orthostatic hypotension, which can be a manifestation of PD itself as well. It is however quite conceivable that levodopamine as precursor of several catecholamines is necessary for the compensation of hypovolemic hypotension. Intrinsic catecholamines tried to compensate the hypotension a long time but became depleted. Storages had to be built up every time like an accumulator and then for a short time there were enough catecholamines to bring blood pressure to higher values. After all, the patient recovered after fluid therapy and the next dose of levodopa therapy orally, suggesting that a combination of factors have played a role in the remarkable episodes of instability in arterial blood pressure.

When available, levodopamine therapy should be continued intravenously during surgery. In the Netherlands no intravenous preparation is available. We recommend to administer levodopamine therapy by nasogastric tube during the operation. For short procedures levodopamine therapy can be postponed until the patient is recovered from anesthesia. We recommend that the time elapsed without levodopamine therapy should be as short as possible.

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