

## Letters to Editor

### Thoracic epidural analgesia for type B aortic dissection

Sir,

Acute type B aortic dissection (ABAD) is a life-threatening vascular emergency with an incidence estimated at 5-30 per 1 million people per year.<sup>[1]</sup> Available evidence support endovascular and surgical treatment for ABAD complicated by shock or organ damage. In uncomplicated ABAD, benefits of endovascular treatment are not well established and the preferred management mandates close clinical and imaging monitoring and treatment of elevated arterial blood pressure (ABP) and pain.<sup>[1,2]</sup> We report on a patient with ABAD in whom, in contrast to standard therapy, epidural clonidine was highly effective in controlling elevated ABP and pain. Although epidural clonidine has been employed in different clinical settings, this is, to our knowledge, the first report on its use in ABAD.

A 48-year-old male patient with unremarkable medical history apart from cigarette smoking and mild hypertension arrived at the emergency department because of intense back and abdominal pain. A computed tomographic angiogram showed an ABAD extending from the subclavian to the iliac vessels with the sparing of the coeliac trunk and the renal arteries perfused from the false lumen. At arrival in the intensive care unit, physical and neurological examinations were normal; the patient was mildly sedated but easily arousable and all arterial pulses were present without murmur; blood pressure and heart rate (HR) were elevated (ABP 190-200/100 mmHg; HR 90-100 bpm) despite intravenous (iv) infusion of labetalol (40 mg/h), nitroglycerin (2 mcg/kg/min) and morphine (20 mg/die). A continuous monitoring (i.e., SpO<sub>2</sub>, electrocardiography, radial invasive blood pressure) was initiated, and a right jugular vein catheter was positioned. Because of a headache at 12 h after back pain onset, nitroglycerin infusion was discontinued, and iv fenoldopam (0.5 mcg/kg/min) was initiated. In the first 24 h, despite the increasing to maximal dosages of both iv (labetalol 60 mg/h, fenoldopam 1.5 mcg/kg/min) and oral antihypertensives (enalapril, amlodipine), ABP and pain remained poorly controlled (ABP >150/>95 mmHg; pain Numeric Rating Scale (NRS) best 3-5, pain NRS worst 7-8). The patient underwent a second computed tomographic angiogram that was unchanged. The vascular surgeon decided no active intervention. Then, an epidural catheter was positioned at T10-11 intervertebral space. After a lidocaine test dose, 15 ml of ropivacaine 0.1% were slowly given, followed by a continuous epidural infusion of 0.075% ropivacaine

and clonidine 1.25 mcg/ml at 10 ml/h. ABP and HR (110-120/70-80 mmHg, 55-65 bpm) normalized rapidly, and the pain abated within 2 h (NRS = 2-3). Infusion rates of labetalol and fenoldopam were tapered off and discontinued 12 h later. On day 2 the patient was hemodynamically and neurologically stable, and almost free of pain (NRS = 1-2). On day 3, because of dry mouth and blurred vision, a third computed tomographic angiogram was performed and was unchanged from two previous. Epidural clonidine was withdrawn. On day 5, ropivacaine was discontinued and the patient remained on oral antihypertensives. The patient was then discharged to a medical ward on day 7 and home on day 14. No renal insufficiency, intestinal ischemia or other organ failure occurred, no surgical or endovascular treatment were needed.

When surgery is not recommended, the patient with ABAD needs to be closely monitored, and hypertension and pain treated aggressively.<sup>[1,2]</sup> In retrospective studies, refractory hypertension (i.e., arterial hypertension uncontrolled despite already being on three antihypertensive drugs at maximal dose) increased mortality rates from aortic rupture.<sup>[1,2]</sup> In our patient, different antihypertensive drugs were either not tolerated or ineffective in controlling ABP, and morphine was only partially effective in controlling pain, a factor well known to increase ABP.

For long time, analgesia has been feared to mask symptoms of ABAD progression thus delaying appropriate treatment. Recent reports, however, show that opioid analgesia does not increase wrong treatment decisions. Serial clinical examinations and imaging rather than refraining from pain control are crucial to monitor the evolution of ABAD.<sup>[1-3]</sup> The low doses of epidural ropivacaine and epidural clonidine normalized ABP and decreased pain intensity without preventing neurological monitoring and without causing hemodynamic side effects. Clonidine provided excellent analgesia and should be considered whenever opioids are not effective or contraindicated. Clonidine and ropivacaine as well, can be used as a sole drug for epidural analgesia. However, in previous trials and in our own experience, the combination of clonidine and ropivacaine

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improves analgesia and side-effects.<sup>[4]</sup> In our patient, the primary interest was to control elevated ABP and pain without causing neurological symptoms from higher doses of ropivacaine. On the other hand, arterial hypotension is a dose-dependent effect, which occurs seldom when epidural clonidine is infused at low rates (i.e., <20 µg/h) in hypertensive patients and which is promptly amenable with fluid replacement. Finally, epidural clonidine has preconditioning and protective properties against ischemic injury of brain, heart and kidneys, which are potentially of interest for ABAD patients.<sup>[5]</sup>

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### Conflict of interest

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