척수재활

게시일시 및 장소 : 10 월 18 일(금) 13:15-18:00 Room G(3F)

질의응답 일시 및 장소 : 10 월 18 일(금) 16:21-16:25 Room G(3F)

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Case report: Tamsulosin Induced Life-Threatening Hypotension in Spinal Cord Injury Patient

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Introduction

Tamsulosin is a selective α_1-adrenergic receptor(AR) antagonist, which has been widely used for benign prostatic hypertrophy(BPH). Moreover, it is effective on neurogenic bladder and commonly used for neurogenic bladder in patients with spinal cord injury. Its common adverse events are dizziness, abnormal ejaculation. Hypotension may be another adverse event(AE), nevertheless, only few cases of tamsulosin induced severe hypotension were reported.

Case presentation

A 59-year-old woman visited ER after slipped down. She had surgery because of fracture at C6 vertebrae and symptoms of myelopathy. Then, she was classified as C3 AIS D(American Spinal Injury Association Impairment Scale) and transferred to Rehabilitation medicine for further rehabilitation. We prescribed tamsulosin because she suffered from voiding difficulty. Her blood pressure(BP) remained stable , but occasionally she felt dizzy and BP fell down probably due to orthostatic hypotension. And another problem was difficulty in defecation accompanied by nausea and lightheadedness. This could have been vasovagal symptoms. At night after applying tamsulosin for 9 days, her BP suddenly fell down to 70/40mmHg. Before the event, she had difficulty in defecation and felt dizzy. Heart rate was 50beats/min(bpm), body temperature was 36.3ºC, and O2 saturation was 82%. Initially, she was treated with normal-saline hydration, O2 supplement and norephinephrine(NE). Despite the treatment, her BP decreased to 50/40mmHg and we thought hypovolemia was not the underlying cause. Atropine was applied and her BP raised to 120/80mmHg. We stopped tamsulosin because of its possible hypotensive effect. Next day, BP fell down again to 60/40mmHg after defecation. Atropine and NE were administered with hydration, and BP raised up to 100/60mmHg. Heart rate was ranged from 90bpm to 110bpm. NE was tapered for 3 days and her laboratory test, vital signs all remained stable. She met criteria of Systemic Inflammatory Response Syndrome. Therefore we suspected septic shock may be the cause, but no evidences of infection were found. Then, we assumed cardiogenic shock may be the reason. However, her electrocardiogram showed sinus rhythm and no evidence of cardiogenic shock was found.

After stopping tamsulosin, her symptoms all recovered soon and no more events of difficulty in defecation were found. Except for all other causes, tamsulosin and vasovagal syncope were left.

To our knowledge, the possible cause for this case could be tamsulosin itself or malignant vasovagal syncope associated with tamsulosin. There have been several cases of tamsulosin induced AE in old-age men, but no reports were found of life-threatening AE of tamsulosin in SCI patient.

Conclusion

We concluded this case showed an SCI patient with vasovagal symptoms fell into shock status associated with tamsulosin. Therefore, physicians should consider that tamsulosin could cause severe hypotension in patients with SCI having vasovagal symptoms.