

# Elderly Patient with Delirium after Myocardial Infarction

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Delirium is a transient global disorder of cognition. Almost any medical illness or medication can cause delirium. Here, we report a 71-year-old male who presented to the emergency department with a sudden change in mental status, which later resolved. An electrocardiogram was consistent with acute myocardial infarction. The patient later developed symptoms of delirium, and haloperidol was administered. The symptoms did not resolve, and risperidone was initiated instead. The patient subsequently became hypotensive, and treatment was again changed to olanzapine. He returned to full consciousness with olanzapine treatment. When the potential hypotensive effects of haloperidol and risperidone are taken into consideration, in patients with high cardiac risk, olanzapine may provide a better option for the treatment of delirium.

**Key words:** risperidone ■ hypotension ■ delirium ■ myocardial infarction ■ olanzapine ■ elderly

## INTRODUCTION

Delirium is characterized by an acute change in cognition and a disturbance of consciousness, usually resulting from an underlying medical condition or due to a medication. Delirium affects 10–30% of hospitalized patients. The associated morbidity and mortality make the diagnosis of this condition extremely important.<sup>1</sup>

Elderly patients are at high risk for developing delirium. Predisposing factors for delirium include preexisting cognitive impairment or dementia, severe illness, high number of comorbid diseases, functional impairment, advanced age, chronic renal insufficiency, polypharmacy, dehydration, malnutrition, depression, surgery, and visual or hearing impairment.<sup>2,3</sup> Myocardial infarction is one of the most important precipitating factors for delirium.<sup>3,4</sup>

The management of delirium requires diagnosis and treatment of the underlying condition, minimizing distress and—if necessary—pharmacological therapy, including antipsychotics.

## Case Report

A 71-year-old male was admitted to the emergency department with loss of consciousness. The patient had no previous a medical illness. On admission, he was confused and disoriented. He had a body temperature of 37.5°C, heart rate of 128/min (arrhythmic), respiratory rate of 18/min and blood pressure of 110/75 mmHg. Arterial oxygen saturation was 98%, glucose level was 105 mg/dl with a normal complete blood count and serum electrolyte levels. Electrocardiogram demonstrated ST-segment depression and inverted T waves in V1–V4 chest leads. Cardiac enzymes were elevated, he was diagnosed as acute myocardial infarction (AMI), and appropriate treatment was initiated. Shortly after, he entered ventricular fibrillation, which returned to sinus rhythm after direct current (DC) cardioversion. He developed pulmonary edema and severe respiratory failure and was consequently intubated.

The patient was transferred to the intensive care unit

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and extubated on the second day of admission. He became increasingly agitated; disoriented to place, person and time; confused; and experienced hallucinations and erratic behaviors, including thrashing the bed, pulling out catheters and attempting to get out of bed. A brain magnetic resonance scan ruled out any acute pathology, with mild cerebral atrophy. A diagnosis of delirium was made based on DSM-IV criteria.<sup>1</sup> Haloperidol oral solution 1 mg twice a day was initiated. Failure of delirium symptoms to resolve prompted gradual titration of the haloperidol dose up to 10 mg twice a day. The symptoms persisted for three more days. The patient was considered resistant to haloperidol, and risperidone 1 mg BID orally was started instead. The first risperidone dose was given 10 hours after the last dose of haloperidol, and its timing did not coincide with any other medication. Three hours later, the patient developed dizziness and blurred vision, and blood pressure measured at that time was 80/50 mmHg. Physical examination findings included tachycardia (104/min) and pallor. There was neither any new pathologic finding in repeat electrocardiograms nor was there cardiac enzyme elevation. This symptomatic hypotension prompted initiation of vasopressor support. The clinical picture was attributed to the cardiovascular adverse effect of risperidone, and treatment was again changed to oral olanzapine 5 mg daily; 16 hours later, the blood pressure returned to normal. The delirium eventually resolved, and he returned to full consciousness on the seventh day of olanzapine treatment. The patient underwent cardiac catheterization, which revealed a significant occlusion in the left-anterior descending coronary artery. Percutaneous transluminal coronary angioplasty was performed, and a coronary stent was placed.

He was discharged in full consciousness while using olanzapine 5 mg once daily. He visited the outpatient geriatrics clinic 15 days after discharge, and olanzapine treatment was discontinued. After a whole geriatric assessment including Mini-Mental Status Examination along with the findings on the magnetic resonance scan of the brain, he was diagnosed as having early Alzheimer's disease.

## DISCUSSION

Delirium can be the initial presentation of myocardial infarction without the expected symptoms of chest pain or dyspnea, or it can develop during its course, as in our case. A study with very old patients with myocardial infarction, delirium or agitation was detected in 28% of patients after myocardial infarction.<sup>5</sup>

Symptomatic treatment for delirium may include the use of antipsychotic drugs to control agitation and hallucinations, and to clear sensibility.<sup>3,6</sup> Haloperidol has been studied most often in the

symptomatic management of delirium.<sup>7-9</sup> Haloperidol has been known to cause ventricular arrhythmias, orthostatic hypotension, tachycardia, ECG abnormalities and even cardiac arrest.<sup>6</sup> The therapeutic effect of haloperidol should be closely monitored since some patients may be unresponsive. Cardiac side effects are less likely with atypical antipsychotics such as risperidone and olanzapine. Because of relatively high affinities for adrenoceptors, risperidone would be expected to produce orthostatic hypotension. However, by following a 3–7-day dosage escalation schedule, postural hypotension and tachycardia have been avoided in clinical trials. Risperidone has very modest effects on cardiac conduction. No significant prolongation of the QT interval was detected at dosages of 25 mg/day in early safety trials. Olanzapine is generally a well tolerated antipsychotic agent. Beside the classical problems such as weight gain and hyperlipidemia, no specific cardiac adverse effect is reported about olanzapine treatment.<sup>6</sup>

Hypotension is a common adverse effect of haloperidol; however, in our case, hypotension was seen with risperidone. Use of the Naranjo Probability Scale indicated a probable relationship between risperidone and hypotension in this patient.<sup>9</sup> The prolonged hypotension seen in our patient was attributed to the long half-life of risperidone ( $t_{1/2}$ =20 hours). The hypotensive effect of risperidone in elderly patients was also shown in previous studies.<sup>5</sup> New-generation antipsychotics with more favorable pharmacologic profiles have been introduced for the treatment of delirium in the elderly. These atypical agents cause less sedation and fewer extrapyramidal effects, and studies of neuropsychological effects in normal elderly volunteers suggest that they have other advantages. However, their potential advantages for short-term treatment, which is typical in delirium, remain unclear.

There is no doubt regarding the deteriorating effects of unfavorable hemodynamics in the setting of acute myocardial infarction. When the potential hypotensive effects of the commonly used haloperidol and risperidone are considered, it would seem more prudent to prefer olanzapine for the treatment of delirium in patients with high cardiac risk.

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