Pre-Existing Isolated Diastolic Heart Dysfunction in Mechanically Ventilated Patient: Obscure Panic Attacks Pre-Intubation and Unexplained Delirium Post-Intubation

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Abstract

Delirium in intensive care units (ICUs) does not always represent underlying cerebral causes. We hereby present a difficult case of ICU delirium that was secondary to undiagnosed isolated diastolic heart dysfunction (IDHD). Delirium should be considered in the larger perspective of cardio-pulmonary-cerebral etio-pathogenesis with specific therapies directed to the specific inciting factors: for instance the agitations and panic attacks with associated oxygen desaturations in non-intubated patient with pre-existing IDHD should caution the ICU team to re-focus their patient management on the undiagnosed or undertreated IDHD before jumping onto the universal sedation/analgesia protocol for delirium management in the intubated ICU patient.

Introduction

Delirium in intensive care units (ICUs) does not always represent underlying cerebral causes. We hereby present a difficult case of ICU delirium that was secondary to undiagnosed isolated diastolic heart dysfunction (IDHD).

Case Report(s)

A 57-year-old female was admitted to ICU for postoperative care after aorto-bifemoral bypass. Her past medical history was significant for obstructive sleep apnea, hypertension, chronic obstructive pulmonary disease, peripheral vascular disease, and schizo-affective disorder with alcohol abuse. On postoperative day 1, patient started showing signs of episodic agitations and oxygen desaturations (AODs). Non-invasive positive pressure ventilation (NIPPV) with high fractions of inspired oxygen (FiO2) was initiated. The response to NIPPV was less than adequate; however, two days later, unresolving episodic AODs were diagnosed as delirium tremens secondary to her history of alcohol abuse and NIPPV was converted to pressure control invasive mechanical ventilation (IPPV). Subsequently, the patient suffered from non-ST-elevation myocardial infarction (NSTEMI) with estimated ejection fraction (EF) of 35% and global hypokinesis on echocardiography. The medical management for NSTEMI was initiated; troponin levels came back to normal range. However the episodic AODs persisted and the cardiac components of hypoxemia were considered. Repeat echocardiography with bubble Doppler study within a week after NSTEMI demonstrated small intrapulmonary shunt; however global hypokinesis had completely resolved and repeat EF was 75%. Despite subsequent tracheostomy and total 10 days of IPPV for ventilator dependent respiratory failure, the patient was still requiring high amounts of sedation (lorazepam and fentanyl). During a trial of sedation vacation, one episode of AOD incidentally responded to labetolol bolus and this effect of labetalol was reproducible across multiple episodes. Hence it was inferred that the patient had IDHD and stiff hypertrophied heart secondary to long-standing hypertension wherein minimal anxiety and subsequently incited acute hypertension was precipitating episodic acute pulmonary edema and subsequent hypoxemia related worsened agitation; and the cause of delirium was vicious cardio-pulmonary-cerebral cycle of AODs. This difficult ICU delirium case was finally controlled and effectively managed by dexametomidine (for maintenance of awake anti-anxiety control) and labetalol and enalapril (for management of IDHD). The tracheostomized patient was subsequently weaned off the ventilator and transferred to the long term acute care facility on the maintenance treatment of the IDHD (alpha-beta blockers and ACE inhibitors).

Discussion

An abnormality in ventricular filling with preserved ejection fraction manifests clinically as isolated diastolic heart dysfunction. The pathophysiology
underlying this disease process is related both to the elastic properties of the myocardial tissue (passive filling), as well as the active relaxation of myocardial tissue (active filling) (1). Abnormalities in both passive and active filling of the myocardium are due to a complex interplay between increased myocardial mass and changes in the extra-myocardial collagen network (increased cross-linking of collagen as well as increased collagen deposition), which ultimately lead to a stiffened and non-compliant ventricle (2). Due to these pathophysiological features, relatively small increases in blood volume, venous tone or arterial stiffness - either alone or in combination - can result in significant increases in left atrial and pulmonary venous pressures. These increased pressures may subsequently result in acute pulmonary edema and, consequently, episodes of acute oxygen desaturation (1).

The association between heart failure and impaired cognitive function (delirium and dementia) has already been substantiated in previous studies, with impaired cerebral blood flow, neuroendocrine dysregulation and use of delirium-inciting medications for treatment of heart failure implicated as causal factors (3). These factors are especially important considerations while managing any intubated patient on mechanical ventilation as hypocapnea, which can result from hyperventilation, may lead to cerebral vasoconstriction and further reductions in cerebral blood flow. Dexmedetomidine, an alpha-2 agonist, is commonly used in the ICU setting as it is known to have a much lower risk of delirium and respiratory depression than benzodiazepines or narcotics respectively (3). In the case we have described, dexmedetomidine effectively managed the patient’s agitation without contributing to or inciting delirium and also served to mitigate neuroendocrine contributions to vasoconstriction, likely leading to reduced pulmonary venous pressures and improved cerebral perfusion.

Conclusion

In summary, the presented ICU patient elicits that delirium should be considered in the larger perspective of cardio-pulmonary-cerebral etio-pathogenesis with specific therapies directed to the specific inciting factors: for instance the agitations and panic attacks with associated oxygen desaturations in non-intubated patient with pre-existing IDHD should caution the ICU team to re-focus their patient management on the undiagnosed or undertreated IDHD before jumping onto the universal sedation/analgesia protocol for delirium management in the intubated ICU patient.

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