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Letter to the Editor

Before blaming glucocorticoids for prolonged postoperative hypotension, alternative explanations must be carefully ruled out

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Quick Response Code:

Dear Editor,

We read with interest the article by Sharma and Venkatapura on a 9-year-old girl with an extra-axial mass lesion along the vermis who suffered persistent arterial hypotension intraoperatively.[3] The intraoperative hypotension was attributed to suppression of the hypothalamic-pituitary-adrenal (HPA) axis, as only hydrocortisone was effective.[3] It was concluded that resistant perioperative hypotension can occur not only with chronic steroid use but also after a single high dose of steroids and that risk factors for HPA suppression after steroid administration include cumulative duration of use, long-acting agents, and parenteral administration.[3] The study is excellent, but some points should be discussed.

The first point is that left ventricular systolic dysfunction has not been adequately ruled out as a cause of persistent arterial hypotension during surgery. Since surgery and anesthesia are stressful for any patient, it cannot be ruled out that the index patient also reacted with a cardiac stress response in the form of stress cardiomyopathy, also known as Takotsubo syndrome (TTS). Numerous cases of intraoperative TTS have been described in the literature.[1,4] Since TTS can be complicated by systolic dysfunction, heart failure, ventricular arrhythmias, and cardioembolism,[2] it would have been useful to perform bedside echocardiography to rule out this particular stress reaction.

Second, it was not considered that the mass lesion itself or the intraoperative manipulations were responsible for the persistent intraoperative arterial hypotension. Since manipulations of the brainstem were most likely also performed during the operation,[3] it cannot be excluded with certainty that a transient pressure lesion or ischemia in the brainstem at the site of the cardiorespiratory or vasoregulatory centers was responsible for the described phenomenon.

The third point is that the anesthetic drugs administered before and during surgery or their delayed metabolization and excretion were responsible for the described symptoms. We should, therefore, know what kind of analgesics, muscle relaxants, sedatives, and anesthetics were used in what dosage for this particular generalized anesthesia.

The fourth point is that there was no mention of whether pituitary hormones were measured pre- or postoperatively and whether any irregularities, particularly with regard to adrenocorticotropic hormone (ACTH), were detected. If dexamethasone were indeed responsible for the arterial hypotension, one would expect low ACTH. It is also conceivable that antidiuretic hormone production was impaired.

The fifth point is that the pathologic diagnosis of the mass lesion was not reported. Knowing the classification of the tumor could explain the blood pressure problem. What was the histopathologic diagnosis? Is it conceivable that the tumor was hormone-producing and, therefore, causing hypotension?

To summarize, this interesting study has limitations that put the results and their interpretation into perspective. Addressing these limitations could strengthen the conclusions and corroborate the study's message. Before blaming glucocorticoids and consecutive HPA suppression for persistent intraoperative hypotension, all alternative explanations must be thoroughly ruled out.

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