Severe autonomic dysreflexia induced cardiac arrest under isoflurane anesthesia in a patient with lower thoracic spine injury

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Abstract
We present a case of severe autonomic dysreflexia (AD) progressing to cardiac arrest and death under isoflurane anesthesia. Though AD in chronic cervical spine injury is a common entity, occurrence of such an event in the stage of flaccid paralysis in lower dorsal spinal cord injury is rare, especially under general anesthesia. Manipulation of urinary bladder catheter under light plane of isoflurane anesthesia might be the precipitating factor. Increasing concentration of isoflurane failed to abort the episode or might have aggravated it. High level of suspicion and vigilance is necessary to prevent, diagnose and treat such a condition.

Key words: Autonomic dysreflexia, cardiac arrest, spinal cord injury

Introduction
Autonomic dysreflexia (AD) is a potentially life-threatening condition in which noxious visceral or cutaneous stimuli cause a sudden, massive, uninhibited reflex sympathetic discharge in individuals with high-level spinal cord injury (SCI). While AD is well recognized in chronic stage of SCI, it has only been rarely documented in the acute phase (1 month) after SCI, especially below T6. If not recognized as a medical emergency and promptly treated, acute AD may result in devastating complications. We present a rare case of severe intractable early onset AD, in a patient with complete SCI below T9, progressing to asystole under isoflurane anesthesia.

Case Report
A 25-year-old man sustained fracture dislocation at T7-T8 vertebrae following a road traffic accident. He was scheduled for pedicle screw fixation of T7-T8 vertebrae two weeks after injury. On pre-anesthetic evaluation, patient presented with complaints of inability to move his lower limbs and numbness in lower half of body. Clinical examination revealed a conscious, well oriented, afebrile patient with pulse rate of 76 beats per minute and blood pressure of 110/70 mmHg. Central nervous examination revealed complete bilateral flaccid paralysis (paraplegia) and loss of all sensations below T9 dermatome with areflexia. There was no voluntary contraction of the anal sphincter with absence of sensation around the anus. Magnetic resonance imaging showed complete transaction of spinal cord at T10 level. All routine investigations were within normal limits.

While applying standard monitoring in the operating room, it was found that patient’s urinary bladder was distended up to the umbilicus though a bladder was in situ. It was decided to induce anesthesia and thereafter change of blocked urinary catheter and place central venous pressure and intra-arterial pressure monitoring lines. After adequate preloading, general anesthesia was induced with fentanyl 100 mcg and thiopentone 250 mg intravenous (IV). Tracheal intubation was done after adequate relaxation with 6 mg vecuronium. Anesthesia was maintained with isoflurane in mixture of oxygen and nitrous oxide. Post-induction vitals were stable with 100%
saturation and end-tidal carbon dioxide of 36 mmHg. Blocked urinary catheter was removed and re-catheterization done. 1500 ml of urine was drained. Subsequent to bladder catheterization and decompression, patient developed polymorphic premature ventricular contractions with hypertension. Isoflurane concentration was increased, but it was followed by severe bradycardia (38 beats per minute) with malignant hypertension (220 mmHg/100 mmHg). Sodium nitroprusside was started at a rate of 1 mcg/kg/min. As bradycardia was refractory, atropine 1 mg IV was administered. Despite this, patient developed asystole. Cardiopulmonary resuscitation (CPR) was started with 100% oxygen. Sodium nitroprusside was stopped immediately and repeat dose of atropine 1 mg IV was given. Advanced cardiac life support was continued for 30 min with no return of spontaneous circulation. Autopsy revealed mildly dilated heart with a weight of 350 g. All three major coronary arteries were normal and histological examination showed no infarction.

**Discussion**

AD is a life-threatening complication of SCI that results in an uncontrolled sympathetic response secondary to a precipitant. Suspicion of AD is high with SCI at T6 or above. They usually have normal systolic blood pressure of 90-110 mmHg. A sudden 20 to 40 mmHg increase of systolic blood pressure over baseline may indicate AD. Tachycardia and reflex bradycardia are most commonly observed in this setting, and are attributed to heightened noradrenergic sympathetic activity and reflex vagal responses, respectively. Other electrocardiographic abnormalities that have been observed during an attack of autonomic hyperreflexia include premature atrial and ventricular contractions, alterations in T-wave and U-wave amplitudes, atrial fibrillation, bigeminy and conduction block. However, the arrhythmias associated with AD have seldom been recognized as a cause of cardiac arrest in individuals with traumatic spinal cord injury. A report of AD-induced recurrent ventricular fibrillation in a patient with C6 tetraplegia by Cholachis et al. was one of such a rare description available in the literature. Contrary to the successful outcome of CPR observed by these authors, no return of spontaneous circulation was obtained in our patient. This could possibly be due to inherently poor survival rate associated with asystole (about 7% to 24%) as compared to a shockable rhythm (25% to 34%). Moreover, there is possibility of delayed diagnosis and treatment of AD as blood pressure monitoring was non-invasive and continual, but not continuous; thus, no real-time blood pressure was available at the time of removal of the blocked urinary catheter. Hypertension was evident during the period of bladder decompression. The use of atropine for asystole was futile in the present case.

Classical signs and symptoms of AD include pounding headache, profuse sweating above the level of the lesion, piloerection or goose bumps, flushing of the skin, blurred vision, spots in the visual field, nasal congestion and anxiety. AD patients may have one or more of these symptoms, which may be minimal. Most of these diagnostic features can be appreciated only in conscious patients and might have been obscured by general anesthesia in our patient. This possibly resulted in further delay in diagnosis. Some of the serious consequences of AD include retinal, cerebral or subarachnoid hemorrhage.

Several factors that trigger AD have been described in the literature. Irritation of the urinary bladder and gastrointestinal tract are among the most common causes of this condition. Catheterization and manipulation of an indwelling catheter, urinary tract infection, detrusor sphincter dyssynergia and bladder percussion are all well-known precipitating factors. The onset of AD in a tetraplegic patient following the removal of blocked urinary catheter and while the external urethral meatus was being cleaned with chlorhexidine prior to catheterization has been described. Afferent impulses are transmitted through pelvic, pudendal and hypogastric nerves to the isolated spinal cord and cause massive sympathetic response from adrenal medulla and sympathetic nervous system which is no longer under central hypothalamic control. Vasocostriction occurs below the level of the lesion causing hypertension. This may stimulate baroreceptor reflex producing bradycardia, heart block, ventricular arrhythmias and even cardiac arrest. Compensatory vasodilatation above the level of the injury results in headache, flushing and nasal congestion.

Researchers believed that orthostatic hypotension was confined to the acute phase of SCI and AD to the chronic phase of SCI. However, the presence of AD in the early phases of SCI has been reported recently. A retrospective analysis demonstrated 5.7% (3 of 58) incidence of early AD in patients with acute SCI above T6. All the three individuals who developed evidence of early AD had complete cervical tetraplegia. Though, the occurrence of severe AD is rare in early stages, especially in patients with low SCI, our patient developed severe intractable AD leading to cardiac arrest following sudden decompression of bladder. It could possibly be due to a lighter plane of anesthesia during the removal of blocked catheter as well as during re-catheterization of bladder. The occurrence of such a severe AD in the phase of flaccid paralysis, especially in SCI below T6, has not been reported in the literature. Our patient, with complete spinal cord lesion at T10, developed severe AD under general anesthesia with...
isoflurane that was refractory to the vasodilator therapy, and ultimately led to cardiac arrest and death.

When AD occurs during surgery, if possible, management should begin with the removal of precipitant stimulus. Most episodes appear to be brief and self-limited and in most cases no specific treatment is required. Blocked urinary catheter or impacted fecal matter must be attended to. Dysreflexia occurring under general anesthesia is best managed by increasing anesthetic depth in the first instance. Nonetheless, high incidence of AD and associated dysrhythmias has been reported during halothane anesthesia and the use of a myocardial depressant in patients with increased systemic vascular resistance has been questioned. Upright positioning may produce a desirable fall in blood pressure. Further, a wide range of vasodilators has been used to abort the episodes of AD.

Conclusion

We present a rare case of severe AD resulting in asystole and death in a patient with SCI below T6 segment, in the absence of prior symptoms. The precipitating event was sudden decompression of bladder under general anesthesia. Such an event during isoflurane anaesthesia has not been reported previously.

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