Autonomic Hyperreflexia

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Background

Definition

Autonomic hyperreflexia (AH) is a potentially fatal syndrome that develops in individuals with spinal cord injury at or above thoracic vertebral level T6 [1–3]. AH occurs in patients with complete and incomplete spinal cord lesions. Symptoms may occur in 85% of the cases with lesions above T5.

Symptoms

1. Symptoms include nausea, anxiety, malaise, a pricking sensation in the skull region, ringing in the head, and a characteristic throbbing headache.
2. Signs vary from light sweating, blushing, piloerection, tremor, nasal obstruction, spasticity in all extremities, twitching, hypertension, and temporary loss of consciousness to more severe signs. The latter include a state of unconsciousness, bleeding into the retina as well as subarachnoid hemorrhage and cerebrovascular stroke that can lead to death [4–7]. A characteristic feature is a reactive sinus bradycardia. Other rhythm disturbances are common.

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Pathophysiology

1. The autonomic nervous system is the division of the peripheral nervous system that carries motor information to visceral organs and glands. It is made up of the sympathetic and parasympathetic autonomic nervous systems. The sympathetic fibers are responsible for the flight-or-fight response and divert blood away from the gastrointestinal tract and skin through the process of vasoconstriction. The parasympathetic fibers typically act in opposition to the sympathetic autonomic nervous system through negative feedback control. This results in a balance of sympathetic and parasympathetic responses.
2. In those with a spinal cord injury at the level of T6 and above, a noxious or otherwise strong stimulus below the level of injury results in an unbalanced physiologic response.
3. The stimulus causes a peripheral sympathetic response through spinal reflexes, resulting in vasoconstriction below the injury. This reflex response ascends and descends the spinal cord and paraspinal sympathetic ganglia, causing both direct vasoconstriction through activation of perivascular receptors and systemic vasoconstriction through stimulation of the adrenal medulla, resulting in epinephrine and norepinephrine release into the systemic circulation. This results in hypertension, primarily through splanchnic and peripheral vasoconstriction.
4. The baroreceptors in the carotid sinus and aortic arch convey appropriate responses to hypertension through the petrosal ganglion to the nucleus ambiguus and result in strong vagal outflow, bradycardia, and vasodilatation above the level of injury. The central nervous system cannot directly detect the strong or noxious stimuli below the level of injury due to the lack of continuity of the ascending sensory fibers from the underlying spinal cord injury. Hence the descending inhibitory response travels as far as the level of neurologic injury leading to uncontrolled hypertension. As a result, there is flushing and sweating

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above the level of injury, bradycardia, pupillary constriction, and nasal congestion (unopposed parasympathetic responses). Below the level of injury, there is pale cool skin and piloerection due to sympathetic tone and lack of descending inhibitory parasympathetic modulation.

5. T6 is of particular importance in the pathogenesis as the splanchnic vascular bed is one of the body's largest reserves of circulatory volume and is controlled primarily by the great splanchnic nerve. This nerve derives its innervation from T5 to T9. Lesions to the spinal cord at or above T6 allow strong and uninhibited sympathetic tone to constrict the splanchnic vascular bed, causing systemic hypertension. Lesions below T6 generally allow enough descending inhibitory parasympathetic control to modulate the splanchnic tone and prevent hypertension [8].

Precipitating Mechanisms

Practically any stimulus, such as pain or irritation below the lesion, can lead to an episode of AH. Bladder distension or irritation is responsible for 75–85% of cases. Other causes include increased pressure in the abdominal cavity, dilatation of the viscera especially in the pelvic organs, percussion of the bladder, stimulation of the rectum, renal or biliary colic, vesicoureteral reflex, manipulations of indwelling catheters, contraction of the uterus, and introduction of a vaginal speculum [9].

Pregnancy and AH

(a) AH may occur during the peripartum period in women with spinal cord lesions at T6 or higher (as high as 85%) but has also been reported with lower level injuries (20%). The condition is precipitated by distension of the bladder or bowel, vaginal examination, pain, and uterine contractions.

(b) Parturients may experience AH during pregnancy, labor, and delivery and in the postpartum period. Sixty to eighty percent of parturients with a lesion at or above T6 reportedly experience AH during labor. However, a recent case series notes that AH occurs predominantly in women who have had a history of AH [10].

(c) The hypertension associated with AH can be extreme and must be differentiated from preeclampsia and pheochromocytoma. Hypertension may resolve between contractions in AH but persist in preeclampsia and pheochromocytoma. Other signs and symptoms of AH are mentioned above. The severity of symptoms can vary from mild to hypertensive crisis.

(d) AH can result in acute uteroplacental insufficiency, fetal hypoxemia, and fetal bradycardia.

(e) The perception of labor is possible with lesions above T10. Some patients are insensate, while others may experience contractions as strong abdominal spasms. Thus, labor may serve a trigger for AH in those who perceive pain as well as in those who do not. However, AH is more likely in higher lesions with complete transection [9–11].

(f) Parturients with a potential for AHR likely have an increased cesarean delivery rate [10].

Management

1. An increasing number of women with spinal cord injuries are becoming pregnant. Recognition and prevention of AH is critical in managing these patients. These patients must be seen early to assess risk, to obtain a history, and to devise a plan for management in the peripartum period.
2. The results of numerous observational studies and case reports indicate the importance of adequate analgesia and anesthesia during pregnancy, pregnancy-related procedures, labor and delivery, and in the postpartum period despite a parturient's apparent lack of sensation [10].
3. Labor analgesia
   (a) Epidural analgesia has been reported as the best option during labor for the prevention of AH. Inhibition of afferent impulses through neuraxial blockade has long been recommended. Previous case reports and series recommend the use of epidural, combined spinal-epidural, and even intrathecal catheter placement for the prevention of AH [10–13]. This is achieved with local anesthetics; narcotics have not been effective in preventing noxious stimuli from reaching the spinal cord [10].
   (b) Combined spinal-epidural labor analgesia may provide improved analgesia, particularly in the second stage, and should be considered.
   (c) In parturients who have had a spinal fusion, which could affect the spread of analgesia or present a challenge for neuraxial placement, a spinal catheter could be entertained. However the risk of headache must be considered.
   (d) Assessing a level in these parturients may be difficult; Sharpe et al. recommend following hemodynamics as well as deep tendon reflexes in the lower extremities and abdominal wall [10].

4. Cesarean section

   When cesarean delivery or instrumental delivery is indicated, spinal or epidural is of critical importance in preventing AH.

   (a) Some parturients may have had spinal fusions making for an impossible epidural placement. In these cases, some authors recommend general anesthesia in order to prevent an episode of AH [10].

   (b) Another option is the placement of a spinal catheter though at the risk of subsequent headache.

5. Given the risk of AH, parturients with spinal cord injury should deliver in a unit capable of invasive hemodynamic monitoring.

   Many anesthesiologists advise continuous arterial line monitoring in parturients at risk for AH [10].

6. Management of acute autonomic hyperreflexia includes antihypertensives and providing additional local anesthetic if an epidural is in place.

   (a) If hypertension is present, sitting and removal of any constrictive devices are advised. Theoretically, sitting allows pooling of abdominal and lower extremity blood and thereby reduces blood pressure [9]. Constrictive devices may serve as a trigger for AH and loosening allows pooling in vessel beds [9].

   (b) Elimination of any precipitating factors is most important. For example, the bladder should be drained, and indwelling urinary catheters should be evaluated for malfunction [9].

   (c) The use of antihypertensive drugs may be necessary if systolic blood pressure remains above 150 mmHg. The antihypertensive medication should have a rapid onset and short duration of action. Some options for blood pressure control include nifedipine, nitrates, and prostaglandin E2. Hydralazine and magnesium have also been used in pregnancy, but the benefit is not yet known [9].

   (d) AH may resolve because of medication, not because of correction of the underlying cause. Unless the etiology is identified and addressed, recurrence should be expected [14–17].

7. Patients commonly experience AH in the postpartum period, perhaps due to pain bladder distension and uterine contractions. For this reason, maintenance of neuraxial analgesia for several days should be considered in parturients with a history of AH. Observation in an intensive care unit for high-risk parturients may be beneficial [10].

8. Multidisciplinary management is critically important to patients at risk for AH. This should be done early to improve information, management, and outcome [18].

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