AUTONOMIC DYSREFLEXIA

Information for HealthCare Professionals

Autonomic dysreflexia is the term used to describe the autonomic response to painful (noxious) stimuli perceived below the level of lesion. This is a potential complication for all patients with spinal cord lesions above the level of T6. The most common stimulus is a blocked catheter. This problem manifests itself as acute hypertension. Systolic blood pressure can easily exceed 200mmHg. Unresolved it can cause significant complications including stoke, seizures, severe myocardial ischemia and death.

This reflex response is usually suppressed during the period of spinal shock however should still be considered.

The main presenting features of Autonomic Dysreflexia are:

- Severe (pounding) headache
- Profound vaso-dilation (flushing) above the level of cord lesion and vaso-constriction (pallor) below this 'line of demarcation' – visible even in different skin types
- Profuse sweating above the level of cord lesion

In the presence of visible primary symptoms, it is recommended that the initial investigation and treatment of cause should not be delayed through a poorly prioritised need to take and document a patient's blood pressure and pulse.

Mechanism

Body functions involve central and reflex control systems within the brain, spinal cord and nerve structures. The autonomic nervous system is a regulatory branch of the central nervous system that helps people adapt to changes in their environment acting though its two branches the parasympathetic nervous system and the sympathetic nervous system. The branches work complementary to each other usually with one activating and the other inhibiting the actions of internal organs. Connections to different parts of the body are organised segmentally from the spinal cord. The sympathetic nervous system connections come from T1 to L2 segments those to the major blood vessels in the trunk, abdomen and legs are from T5 to T12/L2 spinal cord levels.

Autonomic dysreflexia results from widespread reflex activity of the sympathetic nervous system below the level of injury, triggered by an ascending sensory (usually noxious) stimulus. Following stimulation, over activity of the sympathetic ganglia remains uncontrolled due to isolation of the spinal cord below the injury from normal

regulation by vasomotor centers in the brainstem. Release of substances, such as noradrenaline, cause severe vasoconstriction with skin pallor, pilo-erection and a sudden rise in blood pressure (BP), which is usually accompanied by a pounding headache.

When the body's baroreceptors in the aortic arch and carotid bodies sense the increase in blood pressure they increase the parasympathetic signaling as a compensatory mechanism resulting in bradycardia (via the vagus nerve) and flushing (focal peripheral vasodilation); probably also responsible for headache and profuse sweating above the level of injury (via sympathetic inhibitory outflow from vasomotor centers). However, both these mechanisms are insufficient to satisfactorily control paroxysmal hypertension due to massive sympathetically mediated vasoconstriction of the splanchnic bed.

The commonest presenting symptoms of Autonomic Dysreflexia are:

- Flushed appearance of skin above the level of lesion
- Profuse sweating above the level of lesion
- Pallor below the level of lesion
- 'Pounding' headache
- Non-drainage of urine (urinary obstruction being commonest cause)
- Severe hypertension
- Nasal congestion
- Pilo erection

The commonest causes of Autonomic Dysreflexia are:

- Distended bladder (usually due to catheter blockage or some other form of bladder outlet obstruction)
- Distended bowel (usually due to constipation or impaction)
- Ingrown toenail
- Trauma below level of lesion
- Pressure sore / burns/ sunburn
- Urinary tract infection / bladder spasms
- Renal calculi / bladder calculi
- Deep vein thrombosis / pulmonary embolism

Actions in the event of Autonomic Dysreflexia should be prioritised as follows:

1. Identify or eliminate the most common (most potentially lethal) cause of Autonomic Dysreflexia which is non-drainage of urine. If this is not the cause, then proceed to investigate alternative causes according to the list provided above. Reassure your patient throughout as anxiety increases problem.

2. Identify and remove the noxious stimulus e.g. re-catheterise immediately in the event of a blocked catheter (do not attempt a bladder washout as there is no guarantee that the fluid will be returned). If no alternative catheter is available it

may be appropriate to remove the blocked catheter completely to allow for possibility of urethral drainage until re-catheterisation is possible.

3. If appropriate, sit the patient up, or tilt the bed head-up, to induce some element of postural hypotension. Do this gradually, as it may actually worsen symptoms and hinder investigation where distended bladder or bowel is the cause.

4. If symptoms remain unresolved after removal of noxious stimulus or if noxious stimulus cannot be identified then administer prescribed proprietary chemical vaso-dilator such as sublingual glyceryl trinitrate (GTN) or sublingual captopril (25mg).

   NB: Nifedipine capsules, which were previously recommended for use in treating Autonomic Dysreflexia have been withdrawn from routine use in UK due to being linked with post-incident hypotensive crises.

5. Record blood pressure and give further reassurance. Monitor patient's condition.

References

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