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Letter to the Editor

Management of reduced urine output in the patients with *acute* cervical spinal cord injury

Some patients with cervical spinal cord injury develop oliguria during the first few days after the injury. Silver and associates[1] stated that oliguria was not due to dehydration, failure to absorb ingested water, hypotension, or renal failure. In the first few days after trauma, oliguria is probably due to the release of an antidiuretic hormone, which represents a metabolic response to cervical spinal cord injury.

Reduced urine output is of concern to the nursing and medical staff. When a patient with cervical spinal cord injury develops reduced urine output in the first few days after trauma, the health professionals tend to administer increasing amounts of crystalloids and colloids. Indiscriminate administration of intravenous fluids in a patient with high cervical spinal injury, who has low blood pressure (systolic pressure of 80–90 mmHg), and reduced urine output due to over-secretion of antidiuretic hormone, can lead to pulmonary congestion and may further compromise the lung function.[2] Dietz and associates[3] reported a 29-year-old man who sustained C-6 tetraplegia from an automobile accident. This patient received 5 l of fluids and the pulmonary status deteriorated rapidly. In spite of artificial ventilation, the patient died of hypoxemia and hypercapnia. The authors stated that over-hydration was the initiating factor in the aetiology of adult respiratory distress syndrome (ARDS). Thus administration of large amounts of fluids may not represent a rational approach to the clinical problem of reduced urine output due to over-secretion of antidiuretic hormone.

Therefore, when other causes for oliguria eg, acute blood loss due to gastrointestinal bleeding or haemothorax, have been ruled out, an antagonist to antidiuretic hormone may be given; this may serve also as a therapeutic test. In patients with the syndrome of inappropriate antidiuresis related to central nervous system disorders, the diagnosis is made by demonstrating either nonsuppressible plasma vasopressin (AVP) levels, or effectiveness of treatments with water restriction, demeclocycline, nonpeptide V2 AVP antagonist or diphenylhydantoin.[4] The British National Formulary (BNF) lists demeclocycline as the antidiuretic hormone antagonist, which may be used in the treatment of hyponatraemia resulting from inappropriate secretion of antidiuretic hormone.[5]

Demeclocyline is thought to act by directly blocking the renal tubular effect of antidiuretic hormone. Demeclocycline has been used effectively in the patients with head injury who exhibit over-secretion of antidiuretic hormone,[6] patients manifesting the syndrome of inappropriate antidiuretic hormone secretion (SIADH) associated with vinorelbine therapy for advanced breast cancer,[7] and schizophrenic patients with psychiatric polydipsia.[8] Concomitant treatment with demeclocycline may reduce the tendency of hyponatraemia in psychiatric patients, in whom treatment with the psychotropic drugs that caused the syndrome of inappropriate antidiuretic hormone secretion must be continued.[9] Administration of 1200 mg daily of demeclocycline to patients undergoing cardiac surgery, specifically coronary artery bypass grafting (CABG), led to reliable inhibition of the effects of increased vasopressin secretion commonly seen in patients undergoing CABG procedures, who are at increased risk of complicated fluid and electrolyte problems.[10]

Analogous to the cardiac patients, tetraplegic subjects may exhibit a fragile cardiovascular status in the immediate post-injury period. Fluid overload may disrupt the delicate balance of the cardio-pulmonary system in high cervical spinal cord injury patients, and could lead to respiratory insufficiency ultimately requiring ventilatory support. Over-hydration may be the initiating factor for the aetiology of ARDS in an individual who has recently sustained tetraplegia.[3] Moderate fluid replacement with demeclocycline therapy may be a good option for management of reduced urine output during the first few days after cervical spinal cord injury. However, such an approach needs to be evidence-based. Therefore, a multi-centre study is required to investigate fluid balance in cervical spinal cord injury patients during the first few days after trauma. Blood and urine tests (osmolality, creatinine clearance, arginine vasopressin levels) must be done and clinical outcome due to therapeutic interventions should be recorded; eg, administration of large volumes of intravenous fluid (in a general hospital) resulting in precipitous respiratory embarrassment from pulmonary congestion and adult respiratory distress syndrome. Such a multi-centre study will help to develop guidelines for optimum management of reduced urine output in individuals with acute cervical spinal cord injury during the first few days after trauma.

Cervical spinal cord injury produces complex and inter-related changes in the sympathetic system and the endocrine milieu. Health professionals should be aware of this, as the alterations in fluid balance in a patient with acute cervical spinal cord injury represent only a part of the whole scenario.

S Vaidyanathan, BM Soni, G Singh, JWH Watt, T Oo, P Sett

Regional Spinal Injuries Centre, District General Hospital, Southport PR8 6PN, UK

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