CASE REPORT

AUTONOMIC DYSREFLEXIA TRIGGERED BY BREASTFEEDING IN A TETRAPLEGIC MOTHER

Fras Dakhil-Jerew, MRCS, MSc, Susan Brook, PhD and Fadel Derry, FRCS

From the National Spinal Injuries Centre, Stoke Mandeville Hospital, Aylesbury, UK

Objective: **To highlight an unusual cause of autonomic dysreflexia in tetraplegia and present a review of the literature.**
Study design: **Case report of a patient in Stoke Mandeville Hospital, UK.**
Methods: **A 33-year-old woman with C6 incomplete tetraplegia presented with signs and symptoms of autonomic dysreflexia attributed to breastfeeding.**
Results: **Cessation of breastfeeding was effective in stopping autonomic dysreflexia.**
Conclusion: **Autonomic dysreflexia is associated with a significant risk of morbidity and mortality. Identification of the triggering cause is vital in order to prevent further complications. Breastfeeding is an unusual and unexpected cause of autonomic dysreflexia.**

Key words: breastfeeding, autonomic dysreflexia, tetraplegia.

INTRODUCTION

Autonomic dysreflexia (AD) is an important medical emergency in high paraplegics and tetraplegics. It was first described by Guttmann & Whitteridge in 1947 (1) while studying the correlation between sweating and blood flow. AD represents an abnormal sympathetic response to a noxious caudal stimulus in a patient with a spinal cord lesion at or above T6 level (2). The clinical presentation of AD varies. Patients may present with mild signs and symptoms or with serious symptoms, such as severe hypertension and its consequences. These signs are reversible if the triggering cause is rectified (3).

CASE REPORT

A 33-year-old woman sustained a road traffic accident in 1981 at the age of 18 years that left her with incomplete tetraplegia (C6, American Spinal Injury Association (ASIA) Impairment Scale C). She was stable and leading a reasonably problem-free life. She occasionally experienced excessive sweating, which affected the right side of her face, neck, shoulder and arm. This was relieved instantly by lying down. Regular treatment with clonidine also prevented recurrence. She also experienced frequent episodes of fainting feelings on minimal exertion, which were attributed to a low blood pressure (average 70/40 mmHg).

At the age of 32 years, she conceived following multiple attempts at artificial insemination. Her pregnancy was uneventful and ended at term (40 weeks gestation). She was admitted to the National Spinal Injuries Centre 4 days before her expected date of delivery for close monitoring of her blood pressure. Her blood pressure was stable up until she started labour; however, the progress of labour was halted as her blood pressure was increasing rapidly to dangerous levels (170/130 mmHg). An emergency caesarean section (under epidural analgesia) was chosen as the best option for safe delivery.

She was happy with her first female baby and enjoyed breastfeeding her. The puerperal period was unremarkable. However, on the seventh day, the patient developed a sustained rise in blood pressure of 170/130 mmHg for 30 min with severe headache; her pulse rate was 82 beats/min. An electrocardiogram (ECG) was normal. The diagnosis of AD was made. Her blood pressure dropped to 130/95 mmHg within 10 min following the administration of 10 mg of sublingual nifedipine. A full obstetric check was carried out and eclampsia was excluded. An examination of the breasts did not reveal any signs of lactating mastitis or nipple cracks. Her abdominal wound was healing and healthy looking. No cause was identified for her AD.

On the tenth day, the patient was found to be unresponsive following a fit whilst breastfeeding her daughter. Further examination revealed cyanosis, irregular inspiratory efforts and a blood pressure of 120/85 mmHg. Her pupils were equal and reacting to light. Diazepam was given rectally and the patient responded within 8 min. She was confused and agitated. Liver function tests and electrolytes were normal. She was normoglycaemic. An ECG was normal. Arterial blood gas analysis was unremarkable. A few hours later, the patient continued to have a headache and her blood pressure was raised, ranging between 140/95 mmHg and 150/100 mmHg with a pulse rate of 76–82 beats/min.

It was observed that breastfeeding was clearly related to her episodes of AD and thus she was advised to stop. All symptoms and episodes of AD disappeared after she stopped breastfeeding and her blood pressure returned to its usual range of 90/60 mmHg to 80/50mmHg. She was able to go home.

DISCUSSION

In 1947, Ludwig Guttmann & David Whitteridge (1), working at Stoke Mandeville Hospital, described how distension of the viscera in a series of spinal patients had produced an autonomic response inducing profound effects on cardiovascular activity (4). This is now termed AD.

AD can present as an emergency. The incidence ranges from 48% to 83% in patients with complete or incomplete injuries (2, 4, 5). AD is characterized by episodic hypertension, often combined with bradycardia, sweating, chills, nausea, flushing and severe headache (3, 5, 6). The symptoms and signs are diverse, but for clinical use, a definition of increase in systolic blood pressure by at least 20%, combined with at least one of the above symptoms has been suggested (5). The increase in blood pressure can be mild or severe enough to cause blurring of vision, cerebral and subarachnoid haemorrhage, seizures, neurogenic pulmonary oedema, coma or death (7–9). Systolic blood pressures of 250–300 mmHg and diastolic blood pressures of 200–220 mmHg have been reported (6, 10).

The onset of AD can be triggered by several factors. Stimulation of the lower urinary tract accounts for most episodes of AD, and bladder distension appears to be the precipitant in 75–85% of cases (6). Other urinary tract triggers include infection, urethral distension, instrumentation, stones and testicular torsion. The second most common precipitant is bowel distension due to faecal impaction, accounting for 13–19% of cases (6). Numerous less common causes have been cited, including skin irritations, wounds, sores and ingrown toenails (6), pulmonary emboli (11), acute surgical abdomen (12), gastro-oesophageal reflux (13), skeletal fractures (14) and intramuscular injections (6). In women, triggers can be uterine contractions during menstruation or labour, with a risk of AD of 85–90% during labour and delivery (15), ovarian cysts (7) and breastfeeding (16).

Understanding the pathophysiology of AD is important for proper treatment planning. The patient described here has spinal cord injury at the C6 level with problems of excessive sweating above the level of injury. She generally has low blood pressure, but with noxious stimuli (breastfeeding in this case) it increased rapidly to high levels. Injuries above the splanchnic sympathetic outflow (T5–T6) result in loss of the descending central sympathetic inhibitory activity from the hypothalamus and brain-stem. Due to the loss of supraspinal sympathetic control, during distension of the bladder or bowel, the consequent afferent barrage into the dorsal horn of the lumbosacral spinal cord results in massive sympathetic discharge below the level of the injury. This produces vasoconstriction of the muscular, splanchnic and cutaneous vessels (17). Normally, such hypertension would also be counteracted by a baro-receptor induced generalized vasodilation, but with no connection between the baro-receptor and the body, the only remaining way centrally to counteract the hypertension is vasodilation above the lesion level, which produces headaches, sweating and skin flushing. Usually the resulting hypertension produces a reflex bradycardia (parasympathetic fibres of the vagus nerve run outside the spinal cord and are therefore undamaged by

spinal cord injury). However, bradycardia has not been observed in our patient. The mechanism is unknown, but our observation was supported by 4 other cases from the literature. Krassioukov et al. (18) reported an increase in heart rate of 30 beats/min above the resting heart beat in 2 patients with early AD following acute traumatic C3 complete tetraplegia. Silver (19) described 2 C6 complete tetraplegic patients with no significant change in heart rate during AD episodes shortly after their injuries.

The treatment of AD is well documented in the clinical practice guidelines published by the Paralyzed Veterans of America (20). Once elevation of blood pressure, along with the typical signs and symptoms of AD, has been confirmed, the first step in management is to sit the patient upright. Unlike the management of most hypertensive crises, treatment in this situation is based on the blood pressure itself, and not on its effects on the end organs. The next step is a rapid survey of possible triggering factors, such as an obstruction of urinary outlet, a faecal mass or skin lesions. If the elevated blood pressure does not start to decline or the cause cannot be determined, then medical treatment is essential. Our patient responded satisfactorily to withdrawal of breastfeeding and administration of nifedipine. In general, the best antihypertensive medications to use in this situation should have a rapid onset and short duration of action. Nifedipine and nitrates appear to be the most commonly used medications (5, 20, 21), although no randomized controlled trials have been undertaken and much of the evidence available comes from case reports or small series of patients. Other antihypertensive agents reported to be effective in this setting include hydralazine, mecamylamine, diazoxide, prazosin and clonidine (5, 21).

In conclusion, AD is a preventable medical emergency in tetraplegic patients. A high index of suspicion is required to establish the diagnosis. Triggering factor(s) should be reversed immediately. Breastfeeding is a rare cause of AD. In this case, cessation of breastfeeding was effective to stop AD.

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