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Autonomic Dysreflexia in Spinal Cord Injury Patients

North West Regional Spinal Injuries Centre
Southport & Ormskirk NHS Trust

What is autonomic dysreflexia?

Autonomic dysreflexia is a condition that develops after spinal cord injury in which episodes of potentially life-threatening hypertension may be triggered by stimulation of sensory nerves in the body below the site of injury. The clinical incident has a rapid onset; symptoms and signs are often dramatic with a huge increase in blood pressure.

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1.Why is autonomic dysreflexia so important?
2.Who are at risk of developing autonomic dysreflexia?

1.The acute increase in arterial blood pressure is associated with neurological symptoms such as blurring of vision and headache. However, more serious events including subarachnoid haemorrhage, seizures, intra-cerebral haemorrhage (Figure 1), or even death have been reported.

2. Autonomic dysreflexia occurs in patients with a spinal cord injury above the greater splanchnic outflow, usually above T6; however, autonomic dysreflexia has been reported to occur with spinal cord injuries at neurological levels as low as T8.

Predisposing Conditions?

- Autonomic dysreflexia may be triggered by an underlying disease and may also be caused by iatrogenic actions. Dyssynergic voiding, distension of the urinary bladder during cystoscopy, or because of a blocked urinary catheter is a common underlying event for the development of autonomic dysreflexia. Acute urinary tract infection and urological procedures such as cystometry, percutaneous nephrolithotomy, extracorporeal shock wave lithotripsy, urethral catheterisation or change of suprapubic catheter, electroejaculation and severe constipation may precipitate autonomic dysreflexia in a susceptible patient. Uterine contractions can also induce this syndrome.

- Profound bradycardia and cardiac arrhythmias such as premature ventricular contractions, bigeminy and second degree A-V block may occur with autonomic dysreflexia. An ischaemic pattern may be seen on ECG recording and episodes of atrial fibrillation are reported following autonomic dysreflexia in spinal cord injury subjects without other risk factors for heart disease.

What are the symptoms of autonomic dysreflexia?

The presenting symptoms of autonomic dysreflexia are diverse and include pounding headache, blurring of vision, a sensation of precordial pressure, cutis anserina (goose flesh), paraesthesia, shivering, flushing and sweating of the head, nasal obstruction, feeling of unwell and feeling sick. Severe headache, usually of occipital, bitemporal and bifrontal location is noted in more than half of the patients.

The current concepts in the neurophysiology of autonomic dysreflexia are summarised below:

- Changes in sympathetic preganglionic neurones
- Neurobiochemical changes in the spinal cord caudal to cord injury
- Remodelling of spinal cord circuits after spinal cord injury
- Hypersensitivity of vascular alpha-adrenoceptors
- Peripheral afferent stimulation causes marked noradrenaline spillover below lesion level in spinal cord injury patients

How should I treat a patient who has manifested features of autonomic dysreflexia?

- In order to prevent further blood pressure increase the patient needs to be placed in the upright sitting position.
- The symptomatic treatment of autonomic dysreflexia has a dual direction. It is directed (1) to reduce the blood pressure by sublingual administration of nifedipine (10mg) and (2) to remove the triggering factor for the dysreflexic episode. We recommend nifedipine (10mg) orally. The blood pressure is recorded every five minutes. The triggering factor for the dysreflexia is identified and steps are taken to remove the triggering factor as quickly as possible. The most common triggering factors for autonomic dysreflexia are related to the urinary tract. If a patient has a blocked catheter, it should be changed rapidly and the bladder drained forthwith. If the blood pressure is greater than 160/100mmHg five minutes after the first dose of nifedipine, another 10mg of nifedipine is given sublingually.

Is autonomic dysreflexia an advantageous reaction?

- Mild degree of autonomic dysreflexia is beneficial in the sense that it warns the patient, the carer and the health professional that something has gone amiss in the spinal cord injury subject and that urgent attention is warranted to identify the problem and to institute prompt remedial measures. For example, sweating and feeling of unwell forewarn the patient and the health professional that the urinary catheter may be partially blocked, thus allowing the bladder to get distended. A spinal cord injury subject learns to recognise these symptoms and summons for help.

Objectives / Study

The use of terazosin, a long-acting, alpha 1-selective blocking agent, was investigated in SCI patients who developed recurrent symptoms of autonomic dysreflexia, e.g. headache, sweating, flushing of the face together with an increase in the arterial pressure.

21 Adults and 3 children with SCI were having recurring ADR without any discernible urological or other causes

Results

The dysreflexic symptoms subsided completely with the terazosin therapy in all the patients. The 21 adults and 3 children required a dose varying from 1-10mg, whereas the paediatric patients required only 1-2mg of terazosin. The side effects of postural hypotension and drowsiness were transient and mild. One tetraplegic patient developed persistent dizziness and therefore the drug therapy was discontinued.

Conclusion

In 21 adult and 3 paediatric spinal cord injury patients manifesting recurrent episodes of autonomic dysreflexia in the absence of an acute predisposing cause, the use of terazosin, a once-a-day, specific alpha-one blocker resulted in complete subsidence of the dysreflexic symptoms. However one tetraplegic patient required termination of terazosin therapy because of persistent dizziness.

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Untimely Death I

Male (11/5/1980) SCI 04/12/1998

c1430 hrs

Age at SCI 18 years

Age at death 36 years

Expected survival – up to further 30 years age up to 65-68 years

RTC

C2 fracture, passer-by resuscitation, GCS 3/15

Paramedics – intubation/ventilation – Hospital – CT brain – NAD

CT C Spine C1+C2; dislocation C6/7

M21 C/S/ Cord (5/12/98)

Contusion at C1/C2 and C6/C7 level clinically C2 complete tetraplegia (C2 AIS A) tracheostomy – ventilation fully conscious

Admission to NWRSIC

Clinical examination 13/01/99) – not weanable / APNOEIC

- Medically stable on ventilator
- Phrenic pacer implant (13/5/99)
- Phrenic pacing 24/7
- Tracheostomy tube discarded – hood stoma stent
- Discharged with full care package and competent care team

Regular follow up

Few hypertensive episodes (ADRs)

03/2001 – ADRs – BP 170/110

Reflex voiding

Cystoscopy – debris in bladder

Washed out

Spasticity +++, no responding to maximum dose of anti spastic medication

Outpatient Visit 16/05/2016

- Left Ischial Pressure sore Grade IV (18/12)
- Excessive generalised spasticity
- 18/05/16 – Left ischial pressure sore repaired
- 15/06/16 – baclofen pump implanted
- Discharged with advice to perform 4 hourly intermittent catheterisations per day
- 06/07/16 Discharged well

Private Care Agency commissioned by local CCG

New Care Manager

a) administrative , b) clinical

In house training of carers, not by spinal injury staff

Advised to perform 4 hourly intermittent catheterisation without fail. (Advised following last admission).

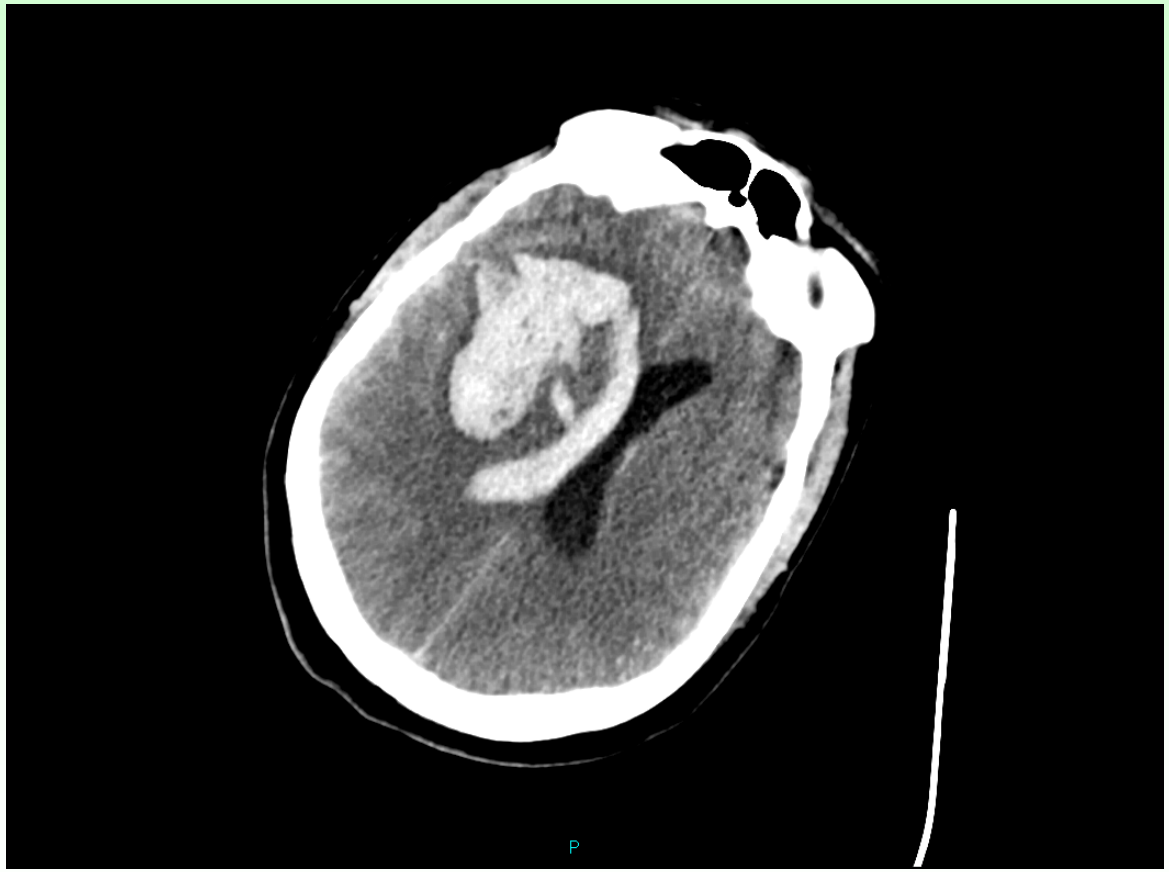
14/07/2016

- Urinary tract infection
- Recurring retention
- Symptoms of headache, confusion, agitation
- 3 BP recordings performed only
- Paracetamol given for headaches (!)
- Patient described in and out of consciousness
- 19/07/16 0500 hours – deeply unconscious following bout of severe headache, confusion and agitation
- I/C tried and failed. Nifedipine not given (PANIC ++)

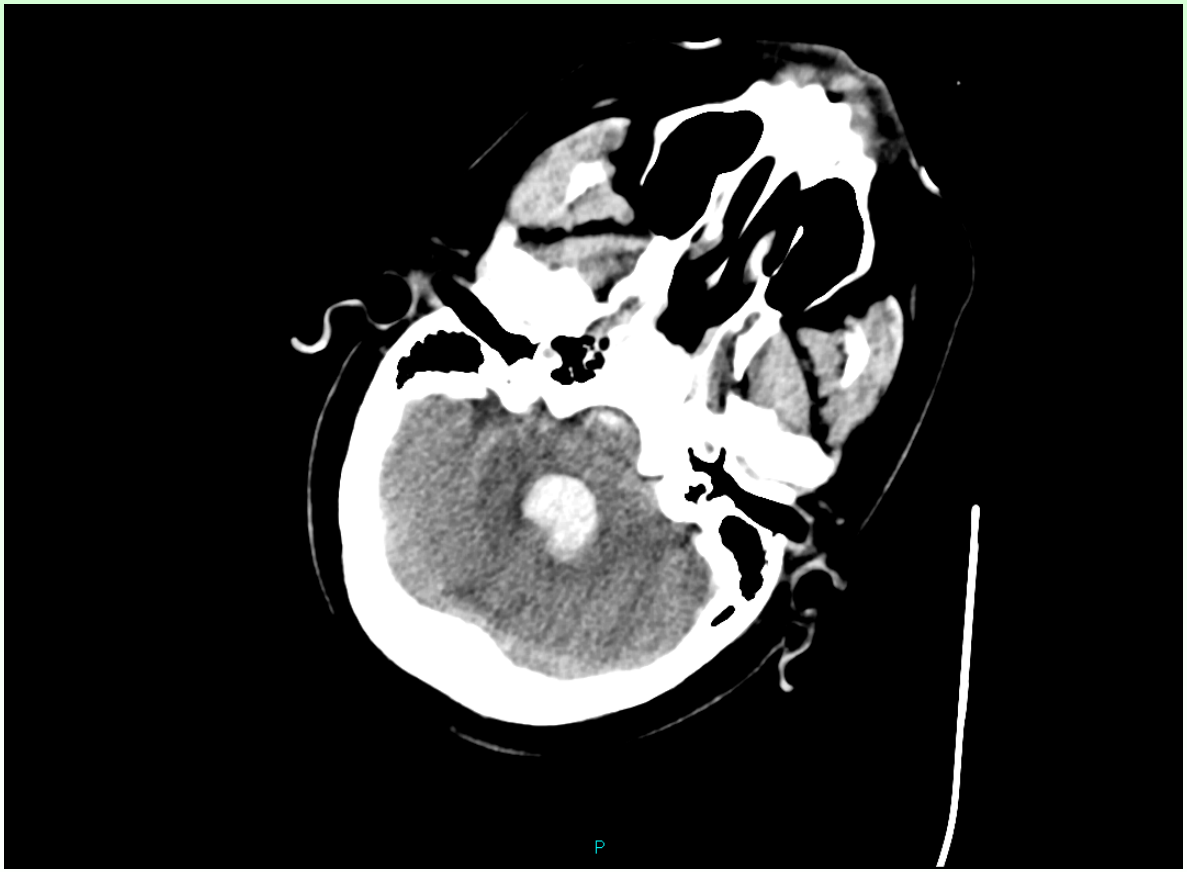
999

- 999 called – taken to local hospital, GCS 3/15
- CT scan
- Cerebral haemorrhage
- 21/07/17 – death pronounced
- Coroners inquest 17/02/17 cause of death
  a) cerebral haemorrhage due to ADR's
  b) tetraplegia

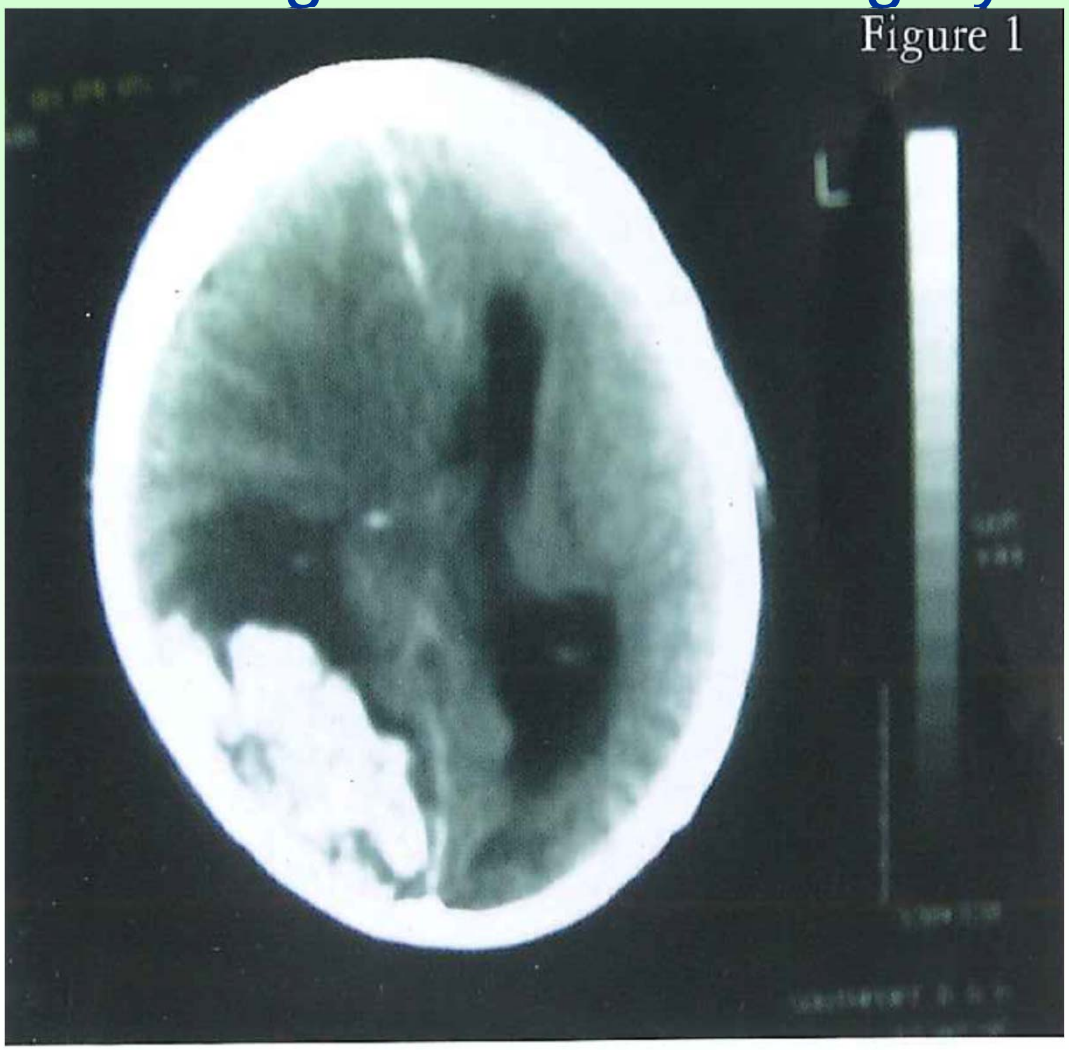
CT scan



CT scan



Cerebral haemorrhage in a tetraplegic patient following renal stone surgery



Untimely Death II

- Male aged 49 – motorcycle accident 1992
- Complete C6 SCI (C6 ASIA A)
- Fiercely independent
  - Active participation in Games
  - Self caring

2009 (chronic phase SCI)

- Manual Evacuation (ME) for bowel management
- Developed grand mal seizure (daughter found him in wet room)
- Diagnosis – epilepsy due to oral baclofen (5mgm once a day) ?
- Baclofen stopped

Reported excessive abdominal muscle spasms

- 24 hour ECG
  - Sinus rhythm
  - Max 97 bpm
  - Min 39 bpm

Collapses were concluded as autonomic dysreflexia caused by manual evacuation

Baclofen for spasms recommenced (10mgm BD)

No further seizure or collapses

2015

- Daughter finds him again unconscious in the toilet
- Left sided facial droop
- Expressive dysphasia
- BP 164/84; T 36.4^{o}C
- GC Score 11/15
- Full recovery within 40 minutes
- CT brain – no pathology

The act of manual evacuation of bowel precipitated autonomic dysreflexia - leading to collapse

Patient insisted on ME as bowel management

September 2016

- Daughter found him unresponsive in the toilet sitting in his wheelchair – blood and faeces on the floor. He had latex glove on the left hand and holding toilet paper.
- Paramedics – confirmed dead 30/09/2016
- Medico-legal post mortem examination carried out
- No cerebral haemorrhage
- All system examination – normal, except hard faeces found in the pelvic colon and rectum.

Cause of death

- Autonomic Dysreflexia
- Tetraplegia

Autonomic dysreflexia – hypertension – coronary artery spasm – myocardia ischaemia – fatal arrhythmia

(excessive sympathetic stimulation)

Learning Points

- Autonomic Dysreflexia occurs in 90% of SCI (T5 and above)
- Usually from lower urinary tract dysfunction
- Rarely fatal
- Awareness and prompt treatment to prevent death
- All patients given ADR information and card
- Advised to carry Nifedipine at all times

Learning Points

- NHS England mandates review of case records of patient who die to identify areas of improvement in care for other patients.
- ME is a recognised method of managing neuropathic bowels. This can cause ADR's which can be asymptomatic and very rarely causes death

- Use of 2% lidocaine gel in ano rectal region prior to ME
- Consider alternative bowel management options eg; TAI, colostomy, ACE or ileostomy in patient where all above measures have failed to control recurring ADRS

Any Questions

Thank you