The Newcastle upon Tyne Hospitals NHS Foundation Trust

Evaluation and Management of Fever in Neurologically Injured Patients

1 Introduction

Fever (body temperature >38.3°C) occurs in up to 70% of neurologically injured patients in the ICU and has been shown to be associated with worse outcome in ischaemic brain injury, intracerebral haemorrhage and after cardiac arrest[1]. This is thought to be as a result of the exacerbation of inflammatory cascades, increasing excitotoxicity and thus worsening of neuronal damage which causes subsequent oedema and raised intracranial pressure.[2]

Elimination of fever and maintenance of normothermia has become easier in recent years following technological advances but, as yet, the benefit of fever control remains a subject of discussion.

2 Guideline scope

This guideline applies to the recognition and management of fever in neurologically injured patients managed on ward 18.

3 Evidence Review and Evaluation

A literature search was made of all relevant original research, editorials and guidelines relating to this important topic. Clinical evidence was summarised for the following conditions commonly seen on neurocritical care

- Cardiac arrest
- Spinal cord injury
- Stroke
- Subarachnoid haemorrhage
- Traumatic brain injury

4 Guideline

Initial workup

Firstly, the patient must be screened for signs of infection. This should include history if possible, clinical examination and culture of relevant areas if appropriate (eg urine, sputum, wound swab, CSF, stool, any identified collections). Routine blood tests such as FBC, CRP and blood cultures should be sent, all indwelling lines and catheters should be assessed and a recent radiology should be reviewed.

If, the clinician feels appropriate, antimicrobials should be commenced.

Control of temperature

It should be noted that fever is an adaptive response that enhances the ability to fight infection and impairment of this adaptation has been shown to prolong the course of certain types of infections (eg community acquired pneumonia, E. coli bacteraemia and Pseudomonas aeruginosa sepsis).[3]

Nevertheless, should the risk of ongoing secondary injury caused by fever be deemed to be greater than the risk of eliminating fever, a number of options are available to attain normothermia.

Pharmacological Options

Antipyretics such as paracetamol and NSAIDS are traditionally first-choice therapy for controlling fever. They block cerebral prostaglandin-E synthesis, which lowers the hypothalamic set point. This activates the body's two principal mechanisms for heat dissipation: vasodilatation and sweating.

The effectiveness of antipyretic agents is tightly linked to conditions where thermoregulation is intact. Therefore, they are likely to be less effective in brain-damaged patients with impaired thermoregulatory mechanisms.

Physical Means

External mechanisms

Surface cooling (fanning, evaporative cooling, cool cloths, sponging, ice packs, and cooling blankets) are commonly used, relatively cheap and require minimal training. Effectiveness can be variable and prolonged exposure of vasoconstricted skin to moisture can leave the patient vulnerable to pressure damage.

Invasive Temp Control

Newcastle Critical Care have access to the Alsius device (an intravascular catheter-based heat exchange system). This method results in a more precise stability at set point and has been shown to reduce the burden of fever by 64% compared to conventional treatments (paracetamol and cooling blankets). The cooling catheter-related risk was not different from a standard femoral central catheter.[4]

Shivering

In normal conditions, the hypothalamus maintains body temperature at 37ºC and initiates a shivering/ vasoconstriction response at <36ºC. Brain-injured patients have an elevated set point and so lowering the temperature, even to normothermia, has been shown to result in shivering in up to 40%. The negative sequelae of this are

- Dramatic increase in resting energy expenditure
- Increased CO_2 production
- Increased O_2 consumption

Treatment of shivering falls in to 4 categories:

1. Attempting to minimise any pyrogenic response (paracetamol)
2. Attempting to raise skin temperature by vasodilatation
    - Bair hugger at 43ºC

- Magnesium Sulphate 4g IV followed by infusion to maintain level 3-4mg/dL

3. Pethidine 50mg IV to lower shivering threshold
4. Sedation/NMBA if seizures problematic and airway secured and patient ventilated

5 Training, Implementation, Resource Implications

All clinical staff should be familiarised with these guidelines as part of continuing professional development. New staff should be introduced to them as part of induction.

6 Monitoring Section

Where appropriate, this should include what will be monitored i.e. the content of the audit, who will gather this information, which group it will be presented to and the frequency at which this will occur.

References

1. Greer DM, Funk SE, Reaven NL et al: Impact of fever on outcome in patients with stroke and neurologic injury: A comprehensive meta-analysis. Stroke 2008; 39: 3029-3035.
2. Suehiro E, Fujisawa H, Ito H, et al. Brain temperature modifies glutamate neurotoxicity in vivo. J Neurotrauma 1999; 16: 285-297.
3. Badjatia N. Hyperthermia and fever control in brain injury. Crit Care Med 2009 Vol 37;7: 250-257.
4. Diringer MN; Neurocritical Care Fever Reduction Trial Group. Treatment of fever in the neurological intensive care with a catheter-based heat exchange system. Crit Care Med 2004; 32:559-564.