Sepsis is ‘A life-threatening organ dysfunction caused by a dysregulated host response to infection’ This definition is perhaps the start of the problem. The exact incidence of sepsis is unknown, an apparent increased incidence may just be due to better recording of cases. Cases of missed sepsis have recently been highlighted in the press, been the subject of the Health Services Ombudsman website ‘Time to Act’ and even resulted in criminal prosecution for doctors mismanaging sepsis. The Department of Health has made the recognition and early management of sepsis the target of a major national campaign.

Recognition of sepsis involves measuring basic clinical data and simple laboratory tests. It is not complex but it remains an area fraught with difficulty for clinicians.

There are a myriad of guidelines both long (NICE sepsis) and short (sepsis 6) which are not complex, so why is it still so hard?

Human factors result in both diagnostic errors and failure to adhere to protocols. This talk will focus on the cognitive biases which impede sepsis recognition and contribute to clinical errors. Improving sepsis recognition may take more than more protocols.

**Background** Guillain-Barré syndrome (GBS) can be associated with ‘swine ’flu’ vaccination, so in 2009 surveillance for GBS and Fisher syndrome (FS) were established via the British Paediatric Surveillance Unit (BPSU) before UK immunisation against pandemic H1N1 influenza commenced. Then in 2010 there were reports from Scandinavia of an association between narcolepsy and Pandemrix (a monovalent pandemic strain ‘flu vaccine containing the oil-in-water adjuvant AS03). To investigate this in the UK it was necessary to identify retrospectively children who had been diagnosed with narcolepsy from before the 2009 pandemic: the BPSU system was not suitable for this.

**Results** BPSU GBS study: 112 children with GBS (66 boys and 46 girls) and 3 boys with FS were identified between September 2009 and September 2011 inclusive. There was an infection in the 3 months preceding onset in 92/112 GBS and 3/3 FS cases. In England, 7 had received pandemic A/H1N1 seasonal influenza vaccine before GBS onset (3/7 within 6 months, including 1 within 3 months). The number of GBS cases who had been vaccinated was no greater than expected by chance.

**Narcolepsy study:** 16 sleep centres and paediatric neurology centres in England identified 75 cases aged 4–18 years with narcolepsy onset after January 1st 2008. Eleven were vaccinated before onset; seven within 6 months. The odds ratio for vaccination at any time prior to onset in cases diagnosed by July 2011 was 14.4 (95% confidence intervals 4.3 to 48.5) and 16.2 (3.1 to 84.5) for vaccination within 6 months of onset. The relative incidence in a self-controlled case series analysis with onset from October 2008 to December 2010 was 9.9 (2.1 to 47.9). The attributable risk was between 1 in 57 500 and 1 in 52 000 doses.

**Conclusion** The BPSU GBS study found no increased risk of GBS in those children living in England after pandemic A/H1N1 2009 influenza vaccine or 2010/2011 seasonal influenza vaccine. In contrast, the narcolepsy study indicated that there was a causal association between vaccination with AS03 adjuvanted pandemic A/H1N1 vaccine and narcolepsy, confirming reports from Finland and Sweden.

**Aims** To investigate a possible association between fever admissions and 4 component Meningococcal B (4CMenB).

**Methods** 4CMenB is given at 8 and 16 weeks in the first year of life. Children included in this study were aged under 1 year in Scotland pre- and post-introduction of 4CMenB vaccine (pre—September 2014 to August 2015) and post—September 2015 to June 2016). The main outcome measure was hospitalisation for fever attributable to 4CMenB vaccine. Analysis was by self-controlled case series using linked routinely collected healthcare data, where the risk period was the 3 days immediately following receipt of a vaccine dose.

**Results** The post-introduction model showed an increased risk in the 3 days after dose 1 (relative incidence (RI), 10.78; 95% CI: 8.31 to 14.00) and dose 3 (RI, 9.80; 95% CI: 7.10 to 13.62), with a smaller increased risk after dose 2 (RI, 2.20; 95% CI: 1.27 to 3.82). The magnitude of these effects was greater than in the pre-introduction model. The attributable fractions were 90.7%, 54.8% and 89.7%, equating to 162, 14 and 84 vaccine attributable cases per 1 000 000 doses, respectively. This is equivalent to 102 extra hospitalisations in Scotland annually, based on a birth cohort of 55 100 and extrapolated to 1430 across the UK based on a birth cohort of 77 165.

**Conclusion** There is an increased risk of hospital admission with fever within 3 days of the routine childhood immunisations at 8 and 16 weeks following introduction of 4CMenB vaccine. The results indicate that further understanding of the current use of prophylactic paracetamol is needed. Communication to parents and health professionals has been re-examined, and guidance on the use of prophylactic paracetamol reinforced.

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