

inactivating mutations in the SMN1 gene can be partially compensated for by limited expression of SMN protein from a variable number of copies of the SMN2 gene, which provides both a molecular explanation for phenotypic severity and a target for therapy. The advent of the first tailored molecular therapy for SMA, nusinersin, based on modulating the splicing behaviour of the SMN2 gene, is the first treatment which significantly alters the natural history of motor neuron degeneration. This will change the landscape for diagnosis, clinical management and future therapeutic trials in SMA, and has implications for the molecular therapy of other neurological diseases.

PO001 SUCCESSFUL NEUROLOGY TRAINEE RESEARCH NETWORK COLLABORATIVE AUDIT

¹Eiman Abdelgadir, ²Dharmini Patel, ²Michael O’Gara, ³Oliver Leach, ¹Omar Al Masri, ¹Shakya Bhattacharjee, ¹Alexander Shah, ¹Jamie Talbot, ¹Hasan Zafar, ¹Camille Carroll. ¹Plymouth Hospital NHS Trust, UK; ²Royal Devon and Exeter Hospital, UK; ³Royal Cornwall Hospital, UK

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Introduction Successful Trainee Clinical Research Networks have been established since 2007. Our network in the peninsula, the SOUTHWEST Neurology Audit and Research Group (SONAR) is the first such Neurology trainee network in the UK. To enable development of cohesive collaborative working of the network we designed an audit which would be deliverable across three neurology centres within the peninsula.

Method We audited management of suspected acute meningitis and meningococcal sepsis against national guidelines within a 4 week period in December. A standardised anonymised data collation tool was used across the three centres and results were analysed at one centre.

Results All 9 registrars on the rotation contributed to audit methodology design and data analysis; seven contributed cases (from all three centres). Ten cases were included in the audit, 6 (Exeter), 3 (Plymouth), and 1 (Truro). Our audit highlighted deficiencies in timely senior review, delivery of antibiotics and steroids, inappropriate administration of acyclovir and delay in lumbar puncture.

Conclusion This was SONAR’s first collaborative project and demonstrated that as a group of trainees we can successfully conduct a project across multiple hospital sites. We plan to extend the scope and ambition of our future undertakings.

PO002 BACTERIAL MENINGITIS WITH MYELOPATHY AND CRANIAL NEUROPATHY

Dariush Ahrabian, Samkeliso Blundell, Krishna Chinthapalli, Gemma Cummins, Robin Howard, Edward J Wild. *Department of Neurology, National Hospital for Neurology and Neurosurgery, Queen Square, London, UK*

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Acute meningococcal meningitis in adults can be complicated by cranial neuropathies and more rarely by myelopathy. A 55-year-old woman presented with acute bacterial meningitis requiring intubation and was treated with intravenous antibiotics and dexamethasone. Cerebrospinal fluid 16S PCR was positive for *Neisseria meningitidis* and latex agglutination confirmed the W135 serotype. Ten days after presentation she

developed mild upper limb and severe lower limb weakness with hyperreflexia, despite resolution of meningism and improvement of inflammatory markers. On the next day she developed complete bilateral hearing loss and bilateral facial palsy. Magnetic resonance imaging with contrast showed bilateral enhancement of VIIth and VIIIth cranial nerves, with focal signal change within the thoracic spinal cord. Audiometry confirmed complete sensorineural deafness. She was treated with five days of intravenous methylprednisolone and continued a further nine days of intravenous antibiotics. Four weeks after onset, she remained completely deaf with mild improvement of limb and facial weakness. We report a case of acute meningococcal meningitis complicated by presumed extensive vasculitis leading to myelopathy and delayed onset of multiple cranial neuropathies. There was an unusual biphasic presentation and the vasculitis was apparently ameliorated by glucocorticoids, suggesting conventional steroid recommendations may occasionally be inadequate.

PO003 ILIOPSOAS HAEMATOMA PRESENTING AS PAINLESS WEAKNESS

Omar AlMasri, Chenchi Reddy Kankara, Stuart Weatherby. *Derriford Hospital, UK*

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Spontaneous iliopsoas haematoma is an uncommon complication of anticoagulation. Such bleeding can be life threatening and therefore early recognition is important. In the published literature the universal presenting symptom is pain. We report on the unusual case of an elderly patient who was anticoagulated with Warfarin for Atrial fibrillation, and who presented with painless weakness of the right lower limb. Anticoagulation was reversed and her condition was managed conservatively. Her case is presented in the context of the published literature.

PO004 PAINLESS AND PROGRESSIVE THINK COMPRESSIVE

Sukaina Asad, Geoffrey Shone, Dan Morris, Caroline Hayhurst, Ann Johnston. *University Hospital of Wales, Cardiff, UK*

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A 47-year-old woman was referred by her ophthalmologist with a working diagnosis of optic neuritis. She presented with a 6 week history of progressive, painless loss of vision in her left eye associated with complete loss of colour vision. She was otherwise well. On examination she was normotensive. Visual acuity on the right with pinhole was 6/9+3 and 6/0 on the left. Field testing on the right eye was full but in her left eye she was just able to appreciate grey shadows and object outlines. There was a left RAPD and bilateral disc pallor. There was no papilloedema or ophthalmoplegia. There were no other neurological signs or endocrinological abnormalities. Inflammatory markers and an autoimmune screen were normal. Neuro-imaging demonstrated a large 50 × 53 mms mass in sphenoid sinus eroding the skull base into the pituitary floor, the optic canals and displacing both cavernous sinuses. Urgent drainage of this sphenoid mucocele was performed in an attempt to preserve vision in both eyes. In conclusion painless and progressive visual loss is out of keeping for an

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