compression in the prevention of the ulceration and amputation compared with current treatments.

Design: A systematic review was performed to determine the cumulative incidence of foot ulcers and amputations with each strategy. Subsequently, a cost-effectiveness analysis was performed.

Primary and secondary outcome measures: The primary outcome was the development of ulcers and amputations with standard care versus tibial nerve decompression. The secondary outcome measures were quality adjusted life years (QALYs), incremental cost-effectiveness and net monetary benefits of the optimal strategy.

Results: Most recent data comparing decompression with standard prevention shows that surgery prevented 1447 ulcers and 409 amputations, per 10,000 over a period of 5 years. Survival was 73% for those receiving medical prevention compared with 95% for those undergoing surgery.

Conclusion: These results suggest that among patients with diabetic peripheral neuropathy and superimposed nerve compression, surgery is more effective at preventing serious comorbidities and is associated with a higher survival over time. It also generated greater long-term economic benefits. Given the high prevalence of diabetic foot syndrome in Australia, it may be worth applying this International experience to home soil.

Reference: Sarmiento, S., Pierre, J.A., Dellon, A.L., Frick, D.K. Tibial nerve decompression for the prevention of the diabetic foot: a cost–utility analysis using Markov model simulations. BMJ Open. 2019 Mar 15;9(3):e024816. doi: https://doi.org/10.1136/bmjopen-2018-024816.

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4. Post-marketing safety study to evaluate the occurrence of aseptic meningitis syndrome (AMS) in an adult population (⩾18 years) treated with doses of ⩾1 g/kg INTRAGAM10®—Arman Sabet [a], Lynette Kiers [b], Stephen Reddel [c], Philip Crispin [d], Ami Patel [e], John-Philip Lawo [f], Annmarie Pendleton [g], Ellen Bonagua [e], Paul Manwaring [h] ([a] Gold Coast University Hospital, Southport, QLD, Australia, [b] Royal Melbourne Hospital, Melbourne, VIC, Australia, [c] Concord Hospital, Concord, NSW, Australia, [d] Canberra Hospital, Canberra, ACT, Australia, [e] CSL Behring, King of Prussia, PA, USA, [f] CSL Behring, Marburg, Germany, [g] CSL Behring, Broadmeadows, Melbourne, Australia, [h] CSL Limited, Parkville, Melbourne, Australia)

On 01 Mar 2017 INTRAGAM10 (10%IVIg) replaced INTRAGAM P (6%IVIg) in Australia. As part of a post-marketing regulatory commitment, CSL-Behring conducted a study to determine incidence of AMS, migraine and severe headache with INTRAGAM10.

The prospective cohort study was conducted in adult patients treated with INTRAGAM10 at ⩾1 g/kg dose. Patients were required to report occurrence of any adverse event (AE) during and within 7 days of infusion.

From March 2017 to July 2018 39 patients were enrolled and 38 patients completed the study. One patient withdrew due to the occurrence of a rash. Clinical indications for INTRAGAM10 use were primarily neurologic and haematologic. Most patients (30/39; 76.9%) had not previously received intravenous immunoglobulin (IVIg). Median reported dose was 2g/kg with a median maximum infusion rate of 4 mL/min. Twenty-four patients (61.5%) experienced AEs within the 7-day period; 79.1% were mild/moderate, headache was the most common AE (11 patients (28.2%)). Three patients (7.7%) experienced severe headache related to the infusion that resolved 7 h, 3 days and 12 days following onset. Confounding factors were present in two of the three patients: pre-existing headache associated with a lumbar puncture undertaken prior to initiation of INTRAGAM10 and in the second patient a history of migraine. Per investigator assessments no confirmed, or probable cases of AMS were reported.

The present study complements existing registration studies and post authorisation safety data and adds to the body of data about AMS with IVIg products. INTRAGAM10 was typically well-tolerated and has a safety profile consistent with IVIg class effects.

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5. Neurophysiological characteristics in the development and recovery of peripheral neuropathy in oxaliplatin and paxlitaxel treated patients—Tiffany Li [a], Hannah C. Timmins [a], J. Matt McCrary [b], Terry Trinh [b], Lisa G. Horvath [c], Michael L. Friedlander [d], Matthew C. Kiernan [a], David Goldstein [b,d], Susanna B. Park [a] ([a] Brain and Mind Centre, University of Sydney, Australia, [b] Prince of Wales Clinical School, UNSW, Australia, [c] Chris O'Brien Lifehouse, Australia, [d] Department of Medical Oncology, Prince of Wales Hospital, Australia)

Introduction: Chemotherapy-induced peripheral neuropathy (CIPN) is a common toxicity of neurotoxic chemotherapy, presenting as numbness and tingling at the distal extremities. Despite increasing prevalence, little is known about the pattern of CIPN development and recovery. This study investigated the trajectory of CIPN from baseline to long-term follow-up using objective and subjective outcome measures, as well as trajectories for oxaliplatin and paclitaxel treated patients.

Methods: 331 patients (219 females; 55.4 ± 12.4 years) receiving various neurotoxic chemotherapies (taxanes, platinums, bortezomib, thalidomide, vincristine) were prospectively assessed at baseline, mid-treatment, end of treatment and at follow-up 3, 6, and 12 months post-treatment. At each assessment, patients underwent sural nerve conduction studies and peripheral neuropathy symptom questionnaires (questions scored 0-4). Paired sample t-tests were conducted to investigate differences across time points.

Results: Overall, there was a significant decline in sural amplitude from baseline to mid-treatment (T1) (difference 2.0 μV, $P < 0.005$) and mid-treatment to end of treatment (T2) (difference 2.9 μV, $P < 0.005$). Similarly, patients reported worsening sensory neuropathy in the lower limbs (T1 difference 0.7, $P < 0.005$, T2 0.9, $P < 0.005$). However, while symptom improvement occurred on patient report (difference 0.2, $P < 0.005$) from end of treatment to 3-months post-completion (T3), there were no improvements in sural amplitudes ($P > 0.5$).

In paclitaxel-treated patients ($N = 165$), there was decline in sural amplitude at T1 (difference 3.4 μV, $P < 0.005$), continuing at T2 (difference 2.9 μV, $P < 0.005$). Following treatment completion, sural amplitudes increased at 6-months post-treatment compared to end of treatment (difference 1.6 μV, $P < 0.05$). However at 12-months post-treatment, sural amplitudes remain reduced compared to baseline (difference 2.3 μV, $P < 0.05$). Patient reports reveal similar pattern of symptom development, with increasing symptoms at T1 (difference 0.8, $P < 0.005$), continuing at T2 (difference 0.8, $P < 0.005$). However unlike neurophysiological parameters, patients reported earlier improvement in symptoms by T3 (difference 0.5, $P < 0.005$), with further improvements from 3-months to 6-months post-treatment (T4) (difference 0.3, $P < 0.005$). At 12-months post-treatment, patients still report greater symptoms than at baseline (difference 0.8, $P < 0.005$).

In oxaliplatin-treated patients ($N = 65$), sural amplitudes did not decline from baseline until end of treatment (difference 5.5 μV, $P < 0.005$), then continued to drop at T3 (difference 3.1 μV, $P < 0.05$). At 12-months post-treatment, there was improvement in sural