cold water and dental sensitivity occurred, I would be inwardly petrified. I couldn’t focus. I developed ‘safety behaviours’ like double tapping wood or my head in order to not ‘tempt fate’. The anxiety and incessant head tapping often brought on headaches, and I’d get sudden panic attacks which would stop me in my tracks, whether I was walking in the street or in clinic, making me feel as though I was about to be violently unwell.

I felt ashamed. There were people who had gone through far worse than me – wars and assaults. And there I was, a twenty-year-old with absolutely nothing wrong with him, having these intrusive thoughts just because of a condition that most people had never heard of. I was a freak.

I went back to my GP who rolled his eyes but did agree to refer me to a therapist at Compass Wellbeing in London. After nearly a year of EMDR (Eye Movement Desensitisation and Reprocessing therapy) and CBT (Cognitive Behaviour Therapy) I came to understand it was irrational to feel this way. I was dealing with an uncontrollable stress response due to previous constant pain, fear, anxiety over what would be, or if I would stay pain-free now I was finally in remission. All these irrational thought processes had stacked up and never been dealt with. My brain had put these old memories and anxieties into boxes with open lids, so they spilled out whenever and wherever.

PTSD really impacted on my life. I wasn’t able to form meaningful relationships with the people around me as I was so scared that no one would understand me, or that I’d be labelled as ‘some crazy’ who considered himself a survivor of an imaginary disease! It wasn’t only my social life that was affected. I was anxious all the time. I jumped at loud noises or unexpected movements. I had panic attacks at the most inopportune of times.

I believe Trigeminal Neuralgia really does impact on you mentally as well as physically. There needs to be a pathway for patients to get the emotional and mental support truly required. Not everyone will need or want it, but it should nonetheless be offered. It takes a lot of courage to ask for help, even more so to ask for mental health assistance because there is a stigma attached to mental health. Sometimes, all we need is for someone to acknowledge that what we’re feeling is normal. That we’re not freaks.

We’re Survivors.

So here I am, now aged twenty-two and life is sweet: Nearly two years in remission from Trigeminal Neuralgia; recovered from PTSD; a great social life; my final year of dentistry and, possibly most important of all, now able to talk and write about my experience with Trigeminal Neuralgia and Post Traumatic Stress Disorder. Some days I may wobble, but I am not alone. I’m stronger now thanks to the cards fate dealt me and am proud of the path I walked.

A Tale of Two Taus

Review: Dr Ed Needham, Neurology Registrar, Norfolk and Norwich University Hospital.

It is always exciting when a breakthrough in fundamental science leads to a concept that may be applicable to a wide number of diseases, and such is the case in the paper by Kondo et al., which primarily investigates the role of phosphorylated tau proteins (Ptau) in neurodegeneration following traumatic brain injury (TBI).

It has long been recognised that victims of TBI can develop a tauopathy known as Chronic Traumatic Encephalopathy (CTE), which bears a pathological resemblance to Alzheimer’s disease (AD). Furthermore, TBI is a risk factor for the development of AD in its own right. Despite clear clinical association, however, the pathological bridge from TBI to CTE to AD remains elusive.

Previous work in AD has suggested that trans Ptau is a beneficial, physiological protein, whereas cis Ptau plays a pathogenic role in tauopathy (Nakamura et al., 2012). The authors of the current study developed monoclonal antibodies to both the cis and trans isomers of Ptau, and utilised them firstly to delineate the role of tau following TBI, hypoxia and metabolic stress, and secondly as therapeutic agents.

The paper starts with the identification of cis Ptau in the axons of all post-mortem brains from 16 patients with Chronic Traumatic Encephalopathy (CTE) compared with no patients in a control cohort of 8; the presence and distribution of trans Ptau was identical in both groups.

Murine models were then adopted to explore the temporal and spatial progression of tauopathy, and its subsequent effect on neurons. At 48 hours post-TBI, cis Ptau was detectable in a severity-dependent manner, and remained raised in those with both repetitive minor and single severe TBI. At this early time point the cis Ptau was restricted mainly to the cortex, but by six months the pathogenic tau isomer was detectable in other regions, including the hippocampus.

Identical production of cis Ptau was also demonstrated in two other models of brain injury: hypoxia and serum starvation.

To assess the pathogenic effect of cis Ptau on healthy neurons, the authors exposed cultured neurons to the brain lysate of TBI and healthy mice, and showed that cis (but not trans) Ptau was subsequently detectable in the neurons treated with TBI lysate, and was associated with accelerated apoptosis.

Having successfully utilised the mAbs to delineate the relative roles of tau in the above situations, attention is turned to their potential therapeutic value. Importantly, the cis Ptau mAb is highly specific in its binding, thereby avoiding unintended effects on the beneficial trans Ptau, and universally beneficial effects of its application were seen in the various murine experiments. Immunodepletion of cis Ptau prevented apoptosis in cultured neurons following the addition of brain lysate from TBI, hypoxia and serum starvation mice; stopped the spread of cis Ptau from the cortex to other structures; potential inhibited the destructive processes affecting axonal microtubules and mitochondria; halted the development of tau oligomers, aggregation and tangle epitopes, as well as cortical and white matter atrophy; and protected against risk-taking behaviour (a putative consequence of medial prefrontal cortex damage) which was widespread in untreated TBI mice.

This study provides new insights into a therapeutically modifiable pathway which may have clinically significant implications in a spectrum of neurological disease states including primary neurodegenerative disorders, cerebrovascular disease, traumatic brain injury and hypoxic-ischaemic encephalopathy.
