Watson v Lancashire TH NHST

Neutral Citation Number: *

QB 2018 000387

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice

Strand, London, WC2A 2LL

Date: 26.1.2022

Before:

MR JUSTICE RITCHIE

- -

BETWEEN

ALISON WATSON

(A protected party by her litigation friend Margaret Almond-Birtwistle)

Claimant

- and –

LANCASHIRE TEACHING HOSPITALS NHS FOUNDATION TRUST

Defendant

(William Featherby QC instructed by Addies Solicitors) for the Claimant

(Bradley Martin QC and Robert Cumming instructed by Hempsons solicitors) for the Defendant

Hearing dates: 12,13,17,18 January 2022

- -

The parties

[1]
The Claimant was aged 29 at the date of the relevant events and was a patient of the Defendant's hospital.

[2]
The Defendant runs the relevant hospital in Preston which treated the Claimant on the relevant dates in March 2015.

The case

[3]

In this case the Claimant suffered a serious stroke in May 2015. She asserts that it would have been avoided but for the Defendant's breach. The Defendant admits a breach of duty but asserts that the stroke would not have been avoided because the breach did not affect the outcome.

Bundles

[4]
For the trial I was provided with written openings by both parties and trial bundles one and two containing the core documents and core medical notes; together with trial bundle 3 split into two parts, A and B; bundle 4 split into three parts, A, B & C; and bundle 5.

Pleadings and chronology

[5]
The letter before action was dated the 7th of February 2018. In that letter the Claimant asserted that she had "full capacity". The lead up to the asserted claim was that on the 27th of March 2015:

"the Claimant got up to go to the bathroom, her vision became distorted, and she felt confused and disorientated. The left side of her face drooped and she could not speak clearly. Her left arm felt weak. She felt she was having a stroke. Her fiancée took her to the accident emergency department at the hospital".

It was asserted that (1) she was suffering from "a transient ishaemic attack" (TIA); (2) the hospital failed to diagnose TIA either as the primary or differential diagnosis and so failed to refer her to neurology experts for treatment; and (3) that the Claimant would have been prescribed Aspirin or Warfarin and would have avoided the stroke which she later suffered in May of 2015.

[6]
In the response letter dated the 7th of September 2018 the Defendant relied on the medical records. It asserted that the Claimant was suffering migraine when she attended in March 2015 but it was admitted that, given the Claimant did not have an established diagnosis of recurrent migraine and given the presence of neurological deficit, it was not appropriate for a junior doctor to reach only that diagnosis and TIA should have been the differential diagnosis. It was admitted that the Claimant's ABCD 2 score would have been four and she should have been referred within 24 hours to the Defendant's TIA service and would have been prescribed Aspirin. The Defendant's reasoning was that the symptoms displayed by the Claimant in March 2015 made it probable that she was suffering from migraine, in particular the distorted vision was more in keeping with migraine than stroke, the progression of symptoms over at least 20 minutes was likewise in keeping with migraine rather than TIA and the tempo of resolution of symptoms was suggestive of migraine rather than TIA. The attack was associated with photophobia, nausea and vomiting which would be highly atypical of TIA. The Defendant asserted that due to her young age and the fact that she was previously suffering Ankylosing Spondylitis she was at increased risk of stroke. On causation the Defendant asserted that Aspirin would not, on the balance of probabilities, have prevented the May 2015 stroke.

[7]
The Claimant issued her claim in December 2018 and in her Particulars of Claim she pleaded that:

"when the Claimant got up to go to the bathroom, her vision became disturbed and she felt confused and disorientated. The **right** side of her face drooped and she could not speak clearly. Her left arm felt weak. She felt she was having a stroke. Her husband took her to the accident and emergency

department at the hospital where she began to feel nauseated and she vomited". (My bold and underlining).

I note that the case has not been amended (save by addition of a litigation friend) and so the Claimant's case rests on her suffering those symptoms, in particular right sided facial droop.

[8]
In relation to causation the Claimant pleaded:

"if (as the Defendants admitted she should have been) the Claimant had been referred to the TIA service (or otherwise treated in accordance with the treatment she would have received there), she would have been seen within 24 hours, and she would have been treated so as to minimise the risk of stroke. In particular she would have been prescribed Aspirin or Warfarin. She would then have either avoided a stroke altogether or suffered a non or minimally injurious stroke."

[9]
In the defence dated February 2019 breach was admitted and causation was denied. It was pleaded that if the Claimant had been referred to the TIA clinic on the 27th of March she would have been started on Aspirin by the accident and emergency department at the same time and seen in 24 hours at the clinic. MRI scanning would have been carried out and the Defendant's case was that migraine would have been diagnosed so the MRI would have been normal. She would have been discharged and so no damage had been caused by the breach. In the alternative, if the Claimant had suffered a TIA, Aspirin would have been continued. The examinations and screens and tests would have been normal, (they were normal after the stroke which she suffered in May). The Claimant would not have been given Warfarin in the absence of cardiac arrhythmia. Aspirin would have been prescribed and that would not, on the balance of probabilities, have prevented the May stroke. The Defendant accepted that Aspirin mediates strokes caused by atherosclerosis but asserted that it only reduces the risk a little for cardiac generated emboli and not by more than 50%.

[10]
The Defendant made sensible part 18 requests of the Claimant to find out what the basis of the claim was on the organic cause of the asserted TIA and other matters. The answers were provided in June 2019 and were partially blocking. The Defendant asked: if the March event was a migraine whether the Claimant admitted that she would not have avoided a stroke. This was denied, however, on the second day of the trial this point was conceded by the Claimant.

[11]
The Claimant asserted in the answers that the stroke in May was caused by thromboembolic occlusion of the middle cerebral artery but the Claimant refused to plead by how much the Aspirin would have reduced the risk of stroke if she had suffered a TIA in March and left the question over until service of the expert reports. I note that the Claimant did not assert multiple TIAs which triggered migraine (the case at trial).

[12]
On the 10th of July 2019 Master Thornett ordered that a preliminary issue on causation should be tried.

[13]
The trial was adjourned from a date in 2020 and another date in 2021 partly due to the COVID pandemic and partly because the Claimant requested that she be present to give evidence live.

[14]
On the first day of the trial the Claimant asserted that she did not have capacity to give evidence and applied to rely on her witness statement in any event. The Defendant objected. I gave permission to the Claimant to rely on her witness statement. My ruling is in the transcript. I noted that the Claimant's witness statement was not signed by her but instead by the litigation friend. I also noted that the letters from the Claimant's treating neurologist were not medical reports, did not have statements of truth attached to them complying with CPR part 35 and did not go into any proper or full recitation of either the Claimant's medical notes or the criteria for deciding on lack of capacity set out in the Mental Capacity Act 2005 and the Guidance Notes thereto. However, I considered that the Claimant's voice had to be heard in these proceedings and let the evidence in, reserving my position in relation to the credibility that I would attach to the Claimant's witness statement. The Defendant was therefore deprived of the opportunity to question the Claimant. I understand that the Claimant attended all four days of the trial by cvp (video).

The Issues

[15]
On day two of the trial the parties agreed that there were four issues in the case and their wording is set out below: -

1.
"Was the facial droop that the Claimant experienced right-sided or left-sided?

2.
What was the extent and timing of onset of the left-sided weakness that the Claimant experienced?

3.
Were the symptoms with which the Claimant presented on 27 March 2015 as a result of TIAs precipitating migraine or sporadic hemiplegic migraine?

4.
If the 26/27 March 2015 event was as a result of TIAs precipitating migraine, would the stroke of 27 May 2015 have been prevented or made non- or minimally injurious as a result of the Claimant taking the Aspirin she would have been prescribed before and after she had been seen in the emergency TIA clinic on 27/28 March 2015.

The Claimant accepts that if the presentation on 27 March 2015 was not TIA, her case fails."

The evidence

[16]
I heard evidence from the Claimant's partner Mr Eastham, Dr. Osborne-Grant, Professor Brown and Dr. Sare. I take into account the medical notes and medical research papers, Nice Guidelines, Royal College Guidelines and ICHD edition 3: the classification of head disorders. I also take into account the Claimant's written witness statement.

[17]
The Claimant was born on the 16th of May 1985 and is now aged 36 years and eight months. At the time she lived in Preston with her fiancée Michael Eastham in a housing association property. They had two children: Harley, born in I think 2008 and Riley born I think in 2012. She is left handed and did not drive. As far as I know (her witness statement does not cover quantum) her hobbies involved

cooking. I do not have her work history but see from the medical notes and reports that for a while she worked in a telephone call centre and also for a while as a cook in a takeaway restaurant. She stopped working there in January 2015 for reasons which will become apparent later.

Medical records (mainly from the GP) before 27 March 2015

[18]
In relation to her health before the relevant events, which occurred when she was aged 29, she had a chequered health history. I set out only the relevant parts below.

a.
On 26th of October 2010 she reported to her GP suffering frontal headaches since Thursday which were intermittent with no nausea but with occasional blurring of vision. She also had a cough and other symptoms and she was pregnant.

b.
On the 1st of November 2010 she told her GP she continued to have headaches which came on and off, she had felt sick the day before and now described throbbing pain. The GP prescribed Co-codamol.

c.
On the 4th of November 2010 the Claimant told her GP of further headaches initially migrainous with nausea and visual symptoms but now more a tension headache. The GP discussed the difficulty of taking medication whilst pregnant and suggested exercise and scalp massage.

d.
There are many other entries between 2010 and 2011 concerning many other health issues. The Claimant was a cigarette smoker. She had previously suffered recurrent contact dermatitis.

e.
In 2011 there were various entries for other matters.

f.
On 7th August 2012 she had various tests and answered various questions. There was no family history of cardiovascular disease and no family history of stroke or TIA or diabetes.

g.
There are many other entries in August and September 2012.

h.
On the 25th of October 2012 the Claimant complained of a history of low back pain for 10 years since school. She had recently had a fall at work and since then had had back pain on and off and had fallen twice thereafter the last 1-2 months before that GP visit. She was working on a telephone enquiry job where sitting on a chair was difficult because it hurt her back.

i.
There are other entries in October and November.

j.
On the 15th of November 2012 a diagnosis was provided of sacroiliac joint arthropathy. She was referred to a rheumatologist for treatment.

k.

On the 23rd of November 2012 she complained of ongoing back and joint pain and considered Naproxen was not enough pain relief. Her shoulders and hands were affected as well. She was treated with Co-codamol.

l.
There are various other entries in November and December of 2012 concerning left shoulder pain not secondary to trauma.

m.
By February of 2013 the diagnosis provided was kyphoscoliosis and scoliosis with suspected inflammatory arthritis.

n.
By April of 2013 the GP was told by the Claimant that she was worn out and constantly tired and sleepy. She had a toddler. Blood tests were ordered and carried out.

o.
There are many entries in April of 2013 and then a few from May through to July 2013. On the 5th end 9th and 19th of July 2013 treatment for her Ankylosing Spondylosis was discussed. She had been taking Paracetemol, had stopped Tramadol due to side effects. She was put on Dihydrocodeine.

p.
By August 2013 she reported to her GP that the Dihydrocodeine made her nauseated and out of it and was looking for an alternative.

q.
On the 6th of August 2013 she complained to her GP of feeling dizzy sick and unwell. She looked tired.

r.
By the 9th of August 2013 she complained of malaise and nausea which had been slow to settle although she was feeling better. The history noted was widespread pains not just in the joints. There are many other entries in August and September.

s.
By the 25th of November 2013 the GP noted that the Claimant had been prescribed Humira for her ankle losing spondylitis.

t.
By the 13th of January 2014 the Claimant had stopped taking Humira injections because it was giving her night sweats and she was gaining weight.

u.
There are many other entries in January, February, June, August and September 2014 relating to other matters.

v.
On the 22nd of January 2015 in a telephone conversation the Claimant reported:

“sharp shooting pains frequently in neck and back of head for some time, says not a headache, no associated symptoms, using Paracetamol and Ibuprofen but no effect, pain comes and goes, wants to see GP”.

w.

On the 23rd of January 2015, at a face to face GP consultation, the Claimant:

“describes a shooting left occipital episodic pain sudden onset once or twice daily for two weeks; Left eye blurs +.”

The history taken was that she was on Humira, her vision was preserved normally, there was no vomiting and no headache, otherwise and her scalp was not tender. She walked normally and her speech was normal and the GP noted no gross motor or sensory changes. The comment provided by the GP was:

“some symptoms consistent with cluster headache”.

x.
On the 29th of January 2015 the Claimant attended the accident and emergency department of the Defendant’s hospital. Her presenting complaint was swollen knees and vision problems. The triage notes stated the patient had Ankylosing Spondylitis with cluster headaches, had been given Imigran by her GP but it made her feel worse. She was complaining of diplopia to the left eye lasting a few seconds and had fallen two days ago causing abrasions and swelling to both knees but had no active headache at present. A doctor’s clinical note at 13.10 on that day included noting Ankylosing Spondylitis that was active bilaterally in her hips and sacroiliac joints. She had stopped smoking three weeks before. She had two weeks of intermittent left eye blurring of vision with episodes lasting 2 to three seconds. There was no “recognised ? event”. (I am not sure what the word is where I placed the question mark). She was also complaining initially of shooting pains to the back of her head lasting 2 to 3 seconds occurring four or five times a day with no precipitating events. She took Imigran and the headaches changed to a dull bi-temporal headache again intermittently which had not resolved. Then she had the fall which involved a bilateral vision episode. On examination there was no gross motor loss and no ophthalmic symptoms, her pupils were equal and reactive and the diagnosis was headache with no red flags which was settling and query active arthritis secondary to trauma. The rest of the discussion relates to her knee issues.

y.
On the 10th of February 2015 the Claimant had a telephone consultation with her GP about her recent fall and her knees and pain killers.

z.
On Thursday the 26th of March 2015 the Claimant had a consultation with her GP about her knee pain and complained of ongoing low mood for the last few months, chronic pain which was getting her down, lots of anxiety, worries about the future and mentioned that she didn’t find the local Ankylosing Spondylitis support group helpful. The medication was noted as Humira and Sertraline and Ibuprofen. On examination she was tearful and would like to try antidepressant.

[19]
I note that this last entry was the day of the key symptoms which occurred either at the very end of that day or the very start of the next day.

The Claimant’s evidence

[20]
In her witness statement the Claimant impliedly asserted that her fall on the 27th of January 2015 was caused by her vision in her left eye blurring. She wondered if she had blacked out. She asserts that between 11:00 pm and 11:30 pm on the 26th of March 2015 she got up to go to the bathroom and then

went downstairs and sat on the sofa. Mick (Mr. Eastham) shouted to see if she was OK. She started to walk up the stairs and felt very confused and disorientated. She had bad pain all over her head. Her vision was disturbed and she felt very tired and weak. Her left arm felt very weak. Mick told her that the left side of her face was drooping. She could not speak clearly. She thought she was having a stroke. She asserts that 20 minutes later they got into a taxi and went to the Defendant's hospital (that would make her time of arrival approximately midnight). She had to wait some time and then saw the triage nurse. He was male. She then waited until around 6:40 am when she saw Dr. Osborne and it is her evidence that Mick explained to Dr. Osborne what had happened. She considers her symptoms improved whilst at hospital although her headache remained, but the lights were hurting her eyes and she vomited on several occasions and she told the doctor about the lights and the vomiting. She asserts that she saw another clinician prior to seeing Dr. Osborne (there is no note of this by another doctor, there was a nursing note) and that doctor informed her that she would have a scan of her head. She asserts Mick made Dr. Osborne aware of her cluster migraines and she asserts a conversation in which Dr. Osborne and she discussed stroke and Dr. Osborne dismissed stroke because the symptoms were more in keeping with migraine. Her age in addition was too young to have a stroke.

[21]
I note that the Claimant does not assert the she suffered any leg symptoms.

Michael Eastham

[22]
Mr. Eastham gave evidence in accordance with his witness statement dated October 2019. In that he stated that he heard the Claimant get up to use the bathroom and then go downstairs. She did not come straight back up to bed so he went to the stairs to ask if she was OK. She started to walk up the stairs and she started laughing hysterically. He saw that her left arm and the left side of her face were hanging down. Her arm looked like a dead weight. He came downstairs because he was worried. (I note here that he does not suggest that the Claimant fell or could not walk). He had previously seen his grandmother have TIAs so he recognised the symptoms. He specifically stated that it was the left side of her face and that the medical records were wrong in stating it was the right side of her face. (I once again note that the Claimant's pleaded case has not been amended to match Mr Eastham's evidence). In any event he called a taxi which he says took about 20 minutes and in evidence he told me it took no more than 10 minutes to get to the hospital.

[23]
He asserted that he explained matters to the triage nurse. They then had to wait until 6:50 am when they saw Dr. Osborne. He makes no mention of seeing any further clinician before Dr. Osborne. He gave evidence that the Claimant's left sided weakness was gradually resolving during the wait but she still had a headache and was being sick and was struggling with lights, saying they were too bright.

[24]
Mr Eastham asserts that when Dr. Osborne examined the Claimant she was "quite dismissive". He accepts that he explained to Dr. Osborne that the Claimant had been suffering from cluster migraines. Dr. Osborne then advised that the Claimant's symptoms were probably cluster migraines and explained that vomiting was not a symptom of stroke and that the Claimant was too young to have a stroke. Mr Eastham mentioned his granny having TIAs on three occasions but Dr. Osborne disagreed. The Claimant was later discharged without further tests or medication.

[25]

It is noteworthy, at this stage, that Mr Eastham did not assert anywhere in his witness statement that the Claimant suffered left leg weakness. I shall return to this below when considering the report of Professor Brown provided in September of 2018. In addition left leg symptoms were not pleaded. Nor were they noted in the medical records made at the hospital. However, in his evidence he once again asserted that the Claimant suffered left leg weakness. I find this to be a part of his evidence which I do not accept.

Doctor Laura Osborne

[26]
Dr. Osborne gave evidence in accordance with her witness statement dated 9th October 2019. Her married name is Osborne-Grant but I will call her doctor Osborne because that was her name at the time. She was an ST3 registrar at the time of giving evidence but at the time of the relevant events she was a trainee doctor in her second year of training. She had a vague recollection of the Claimant. She did not recall the specific details but did recall the Claimant's quite pronounced photophobia when she used a light to read and write her medical records in the consultation and she recalls the room in which the consultation took place. Basically Dr. Osborne's evidence was in accordance with the notes made in her very clear handwriting. She explained that it was her usual practise to ask what had occurred in an open question about why the Claimant had come to the hospital and she wrote down the matters that she was told chronologically. From the records Dr. Osborne thought it likely that she asked the Claimant to take her step by step through what had happened and she asserts that the history in the records is what she was told.

[27]
Dr. Osborne was told that at approximately 11.30 on the night before, the Claimant woke up to get a drink and two minutes afterwards there was onset of various symptoms. Thereafter her notes chronologically set out, with each L shaped arrow, firstly a strange feeling with unexplainable visual disturbance "- aura"; secondly the Claimant's boyfriend saw right facial droop and the Claimant struggling to talk; thirdly right sided headache; fourthly four times vomiting; fifthly photophobia; sixthly 15 to 20 minutes into the episode, the Claimant lost the use of her left arm.

[28]
On questioning about vision, Dr. Osborne wrote down "? Blurry vision. No zigzags/lines." Dr. Osborne then went on to carry out an examination of the patient under the ABCDE protocol which I don't need to go through but I should say under D that Dr. Osborne examined the Claimant's face and eyes and noticed and noted photophobia but no eye deficiency and no facial droop. She noted intact cranial nerves numbered 2 to 12. She also noted the power in the right and left arms and legs as full and the reflexes as normal except for the left arm which had reduced power. She noted her impression was migraine with resolving symptoms and her plan was to provide pills and for the Claimant to be discharged if she improved but if not she might need to be admitted.

[29]
Although she said in evidence that she planned for the Claimant to be seen by a senior colleague, Dr. Osborne did not note that. Under questioning, in her evidence, Dr. Osborne accepted that she could and probably should have written down a differential diagnosis of TIA and for referral to the TIA clinic or that the Claimant should have been seen by a more senior colleague. However she did not accept that she had been negligent.

[30]

The Claimant asserted in questioning that Dr. Osborne was negligent herself. I take into account the note made at 07.55 on 27 March 2015, probably by a nurse, that the Claimant was "awaiting senior review". I consider that this can only have been written because Dr. Osborne decided senior review was necessary and told the staff before she went off shift at 08.00. This was her verbal evidence. I find that Dr. Osborne was not herself negligent because she advised that the Claimant needed senior review. Negligence by the hospital was admitted.

Analysis

[31]
I found Dr. Osborne to be a careful, fair, logical, intelligent, straight forwards, well prepared and impressive witness. Her notes were also clear. I accept her recollection was partial in that she did recall the photophobia and did recall the room in which the examination took place (room 4) but otherwise she was relying on her notes. I find from my impression of her as a witness and from the way her notes are set out that her evidence is credible and logical. So for instance where her evidence clashes with the Claimant's written witness statement and/or the evidence of Mr Eastham, I accept the evidence of Dr. Osborne.

[32]
In relation to the Claimant's evidence set out in her witness statement there are some oddities in it. The first is the mystery doctor who had a conversation with her before the examination by Dr. Osborne. There is no note of this conversation and it is not supported by Mr Eastham. There was a nurse who took her neurological signs but I very much doubt that a nurse would advise on what medical investigations the Claimant was likely to have. Secondly of course she did not come to court to give evidence despite on two previous occasions when a trial was listed asserting through her lawyers that she wanted to come to court to give evidence. Thirdly, although she asserts left facial droop in her witness statement, her pleaded case is that she suffered right facial droop and no application was made to amend the pleaded case either before trial or at trial. The 4th point I take into account is that the evidence in relation to her lack of capacity is not full or properly laid out medical evidence with a part 35 statement. It is in very short treating neurologist letters. In addition her solicitors asserted that she had capacity in the letter before action in 2018. The 5th point is that the witness statement was signed not by the Claimant but by her litigation friend. If the Claimant was able to give this evidence carefully and slowly under careful questioning from her solicitor, then why could she not sign it at least jointly with her litigation friend? Finally, of course she has not been questioned. I do not therefore consider that her witness statement can outweigh the evidence of Dr. Osborne.

[33]
In relation to the evidence of Mr Eastham, he was clearly doing his very best and I find him to have been an honest witness. I take into account the trauma that he has been through because of the Claimant's stroke. It must have been difficult accurately to recall events when the claim was being reduced into written evidence with the solicitor both as to the left or right side of the facial symptoms that he saw before midnight and as to the chronology of the emergence of the left arm symptoms and of course the mysterious left leg symptoms not mentioned to the hospital. I gained the impression that he connected the May stroke to the March events and concluded they must have had the same cause.

[34]
Thus my findings of fact set out below will be based on the evidence of Dr. Osborne and where that conflicts with the evidence of the Claimant or Mr Eastham I prefer her evidence.

Findings of fact

[35]
I find that on the occasions set out in the GP notes and the hospital A and E notes before the 27th of March 2015 the Claimant suffered various episodes of headache. So for instance in October – November 2010 and for 2 weeks in January 2015 and that these were accompanied by visual disturbance where that is noted. I find that the January fall was associated with visual disturbance.

[36]
I find that at about 11:30pm on the 26th of March the Claimant got up either to go to the loo or to get a drink. Some minutes after waking, when she was downstairs in her own house, she suffered the onset of symptoms which consisted of a strange feeling with unexplainable visual disturbance which she herself described as "aura" and headache. I find that after those symptoms started, probably very shortly after, Mr Eastham saw her from the top of the stairs with right sided facial droop and that he then came down the stairs to help her. He noticed the Claimant struggling to talk. He did not notice her struggling with the wrong words. I find that the Claimant suffered right sided headaches and later suffered vomiting and photophobia. I find that her vision was blurry but she did not have zig zag lines or lines or a castle shape or any other unilateral shape in just one of her eyes (or both). It was blurry vision. I find that chronologically 15 to 20 minutes into the episode she started to suffer weakness of her left arm. I find that she did not suffer left leg symptoms.

[37]
I find that about 1 and a half hours or so after the onset of symptoms the Claimant and Mr Eastham travelled to the hospital, after a delay when they were arranging and waiting for a relative to arrive to take care of their children.

[38]
I find that the Claimant arrived at the hospital at or after 1am. I find that the Claimant was registered by the hospital as arriving at 1.22am on Friday 27 March 2015.

[39]
From the triage notes, which I accept are accurate and were written by Mark Pennington, I find that the Claimant was triaged at 01.50 hours. The presenting complaint was a numb face and left arm weakness, which were described as neurological deficits and that the Claimant was classified as "yellow" for triage purposes. I find that the triage notes accurately recall what was said to Mr. Pennington, save that the Claimant or Mr Eastham incorrectly recalled the onset was just after midnight. I accept Mark Pennington was told that the Claimant had suffered right sided facial droop which had resolved and also left arm heaviness. He noted a past medical history of Ankylosing Spondylitis and depression.

[40]
The Claimant was then waiting in the hospital until at 05.30 hours she was examined by a nurse who noted her eyes were open, she was orientated and she could obey commands. The nurse took her pulse and oxygen saturation and noted that her left arm had mild weakness (B2 p31).

[41]
At 06.40 the Claimant met Dr. Osborne and, as I have found above, I find that the notes Dr. Osborne made are an accurate record of what she was told by the Claimant and Mr. Eastham and what she did and of her examinations and her conclusions and findings. The presenting complaints were right sided facial droop, left arm weakness, headache and vomiting with photophobia. She was told that the first

symptoms were a strange feeling with unexplained visual disturbance – aura. The Claimant had suffered right sided headache and right sided facial droop and was struggling to talk. 15-20 minutes after the start of her symptoms she suffered loss of use of her left arm (to an extent). She had no fever, no rash, no neck stiffness, no head trauma and no seizures. Mr Eastham informed Dr. Osborne that the Claimant had cluster migraine 3-4 weeks before and was treated with Sumatriptan by her GP but had suffered severe chest pain. The previous events were described as "similar" to the March event but with no weakness or vomiting. On examination Dr. Osborne noted what she did and in particular noted that the eye examination was normal but photophobia was present. On power testing the Claimant's left arm remained weak. Her other limbs were normal. Dr. Osborne diagnosed resolving migraine symptoms.

[42]
I find as a fact that the Claimant's facial droop had been right sided and that Mr Eastham told Dr. Osborne that.

[43]
At 07.55 am the Claimant was seen again by a nurse and her "record of adult neurology" was recorded again on the relevant chart. Her left arm weakness had resolved. The nurse had spoken to Dr. Osborne and been told the Claimant was awaiting senior review. The nurse noted that advice in the medical notes. Unfortunately she was then discharged, probably at around 10.00 am. The discharge summary shows the Defendant's staff considered Humira was the possible cause of the headaches. It also recorded right sided facial droop, left arm weakness and migraine symptoms which had fully resolved.

Expert evidence

[44]
Having made the above stated findings of fact my next task is to determine whether the Claimant's symptoms on the 27th of March 2015 were caused by (1) migraine with motor aura or (2) multiple TIAs triggering migraine.

[45]
This challenge is to be fulfilled by an analysis of the expert evidence. The experts were tasked with providing a diagnosis, after the event, by analysing the symptoms suffered by the Claimant on the 27th of March in the context of her previous medical health, her post event health and by reference to their clinical experience and the many learned medical papers to which they referred.

Prof. Brown

[46]
Professor Brown was called by the Claimant. His evidence from his first report dated September 2018 on condition and prognosis can be summarised as follows. He had not seen the MRI scans when he reported. He never did on the evidence before me. When he saw the Claimant she could not remember the details and so Mr Eastham gave most of the detail. The Claimant did recall that her symptoms started with the fall in January 2015 when she injured her knees and had blurred vision and went to A&E.

[47]
Mr Eastham told Professor Brown that on the 26th of March 2015 in the evening the Claimant became weak after getting a drink and he noted she had left sided face droop and that she was unable to lift

her left leg. Mr Eastham went on to tell Professor Brown that the "nurse" who examined the Claimant at hospital was "very dismissive" of the idea of stroke and diagnosed migraine and discharged the Claimant. This misunderstanding by Mr Eastham of the status of Dr. Osborne is another reason why I do not fully accept Mr Eastham's understanding and recollection of the events.

[48]
Professor Brown's condition and prognosis report contains very little summary of the Claimant's medical records. He stated that the records from the GP that he received were incomplete. The only past record he highlighted was that on the 26th of October 2010 when the Claimant suffered what he described as frontal headache with no nausea and occasional visual blurring.

[49]
Professor Brown opined that in May 2015 the Claimant had suffered ishaemic stroke "caused by **cerebral infarction in the territory of the right middle cerebral artery**" (B1p147 – my underlining and bold). His prognosis was that she would not recover and that she had epilepsy. He was concerned that she might not have capacity and advised the Claimant's lawyers to seek a neuropsychologist's report on capacity. No such report was put before me.

[50]
In his second report dated the 12th of January 2020, on causation, Professor Brown gave the opinion that in May 2015 the Claimant suffered a stroke "with right hemisphere **cerebral infarction in the territory of the left middle cerebral artery**" (B1p172). Comparing this with the first report he was contradicting himself and he has provided no explanation for doing so.

[51]
Professor Brown gave the opinion that the May 2015 clot was in the middle cerebral artery or formed elsewhere and travelled there as an embolism. He stated that embolism was more likely rather than in situ genesis of the blood clot. (I store that part of the report for further consideration later in this judgment). He stated that the underlying cause was not known. He advised the court that one quarter of strokes have unexplained genesis or cause. The commonest cause in young people is cardiac embolism. Large (atherosclerosis) and small vessel disease are common causes in patients over 50 but very uncommon in patients under the age of 30. (I store that part of the report for my later analysis of a medical paper by Rothwell et al).

[52]
He opined that it was likely that the Claimant had a past history of migraine and aura (B1p172). Stopping here, there was considerable cross examination by Claimant's counsel of Dr. Sare involving the suggestion that the Claimant did not have a past history of migraine and aura. That cross examination was not based on Professor Brown's report.

[53]
In relation to the Claimant's symptoms in January 2015 Professor Brown reached no conclusion in his report stating that it was possible they were caused by TIA affecting her eyes but that the symptoms were not typical of TIA. Alternatively migraine was a possible cause or there was some other cause.

[54]
In relation to March 2015 Professor Brown gave the opinion that the Claimant's symptoms had some features of migraine, specifically right sided headache, vomiting, photophobia and visual disturbance. He asserted the Claimant had some symptoms more typical of TIA namely facial weakness and limb weakness. He advised that migraine is thought to be caused by a metabolic disturbance that impairs

brain function in localised areas and that TIAs are caused by a reduction in blood flow in a localised area of the brain, as a result, in the majority of cases, of emboli temporarily blocking the artery supplying that part of the brain. He noted that the symptoms of TIA and stroke were identical and only distinguished by their duration.

[55]
Professor Brown back analysed the March 2015 symptoms suffered by the Claimant in an effort to distinguish between the two potential diagnoses. He started with the first symptoms: a strange feeling with visual disturbance "- aura". He considered Dr. Osborne's note of blurry vision with no zig zag or lines. He opined that aura is typical of migraine. He referred to the ICHD 2018 on "aura" at page 20 and gave the opinion that the Claimant's visual disturbance was not diagnostic of migraine. He advised that visual disturbance can occur in TIA as a result of ischaemia affecting the visual pathways in the brain or the ophthalmic artery. But he did not come to a conclusion on which was more likely.

[56]
In relation to facial droop Professor Brown accepted the medical notes and assumed the facial droop was on the right side (thus preferring that to the evidence of the Claimant and Mr Eastham in the report). He advised that isolated facial droop was not a feature of migraine because it was not mentioned in ICHD and therefore considered that this was more suggestive of TIA of the brain stem.

[57]
In relation to speech he advised that difficulty speaking and confusion as described by Mr Eastham were a more common feature of TIA but also were associated with migraine.

[58]
In relation to headache, on the right side, vomiting and photophobia he advised these were typical of migraine but could also be seen in TIA. He relied on a study by Lebedeva from 2018 which found that headache occurred in 30% of patients with TIA, but unilateral headache only occurred in 15%. He explained that one possibility tying these symptoms to TIA would be that that the TIA triggered migraine like symptoms in a patient who had an underlying susceptibility to migraine. His other theory was that these symptoms could have been caused by a TIA affecting the vomiting centre and sensory supply to the head which are located in the brain stem. Therefore he concluded that the symptoms of headache, vomiting and photophobia did not distinguish between migraine and TIA taken in isolation. I do not find his reasoning on this persuasive for the reasons set out below.

[59]
In relation to the left arm weakness it was Professor Brown's opinion that this was not a common feature of migraine unless the migraine was hemiplegic migraine. It was more likely TIA.

[60]
In relation to the "march of symptoms" by which Professor Brown meant their onset, their progress and their offset, he accepted that a march of symptoms over 20 minutes would be typical of migraine and stated that the migraine aura rarely lasted more than one hour. He also commented that in hemiplegic migraine, hemiparesis is usually more marked and persistent than in the Claimant's case. He pointed out there was no family history or previous hemiplegia and he suggested that that weighed against a diagnosis of hemiplegic migraine, but he could not exclude it. He advised that given the lack of family history the correct diagnosis would have to be sporadic hemiplegic migraine if the symptoms had been the result of migraine.

[61]

He advised that limb weakness was very common as a result of TIA and therefore advised that this feature was strongly in favour of TIA being the cause. The relevant TIA would need to be in the right cerebral hemisphere supplied by the anterior circulation. Then, (B1p179) he went onto opine that another TIA must have caused the right sided facial weakness and he used the words:

“being caused by separate cerebral emboli (typically from the heart) the first embolus going to the posterior circulation and the second to the anterior circulation.”

[62]
In relation to the march of the symptoms he argued that the sequence of events was more suggestive of the occurrence of two distinct TIAs. In migraine he advised that the march of symptoms from adjacent territories in the brain is generally within one hemisphere. In his opinion it would not be typical of migraine for the symptoms to jump from facial weakness on one side to arm weakness on the other side of the body.

[63]
Professor Brown then looked at the incidence of TIAs in young patients and accepted they were rare. He advised that the incidence of TIAs in patients between the ages of 15 and 44 was one in 50,000 of the population per year. This was based on a study carried out by Dennis in 1989 in Oxfordshire. In relation to hemiplegic migraine he advised based on a study from Denmark by Thomsen in 2002 that sporadic hemiplegic migraine had an incidence of one in 250,000 per annum in the population under the age of 40. He relied on this in support of TIA being more likely.

[64]
Finally Professor Brown relied on a backwards chronology. He stated that the fact that the Claimant suffered a stroke in May 2015 strongly favoured TIA in March 2015. However his evidence in support of that was from a study by Coull in 2004 which only showed that 15% of strokes were preceded by TIAs. In addition I doubt that the Coull study concentrated on patients aged 29 or thereabouts.

[65]
Professor Brown asserted that the ABCD2 and Rosier tests for stroke should be used by the court to support a finding that the most likely diagnosis for the March symptoms is multiple TIAs. Many questions were put to Dr. Sare on that basis. For the reasons given by Dr. Sare I consider that this assertion was stretching the purpose of those tests. I find that they are used in A & E and other primary clinics as a filter to identify the risk of TIA and stroke and so to trigger onwards referral to a TIA clinic or neurologist or stroke expert for diagnosis. Diagnosis is done by MRI or CT and multiple other tests and screening. I accept Dr. Sare’s evidence that these are filter tests not diagnostic tests.

[66]
It was on these grounds that Professor Brown advised the court that on balance it was more likely that the Claimant suffered TIA rather than hemiplegic migraine. He asserted that the clinical features favoured his view and that the research papers favoured TIA and that TIA was more common than hemiplegic migraine even in young adults and finally that the subsequent stroke favoured a preceding TIA.

[67]
In his report he advised the court that the Claimant suffered 2 TIAs, the first responsible for her initial symptoms affecting the left side of the brain stem and the second responsible for her left arm weakness affecting the right brain hemisphere. I note that in his report he did not conclude on

balance that the Claimant suffered TIAs causing a migraine so he provided no conclusion on the photophobia.

[68]
I was unclear about where Professor Brown placed the various clots necessary to fulfil his multiple TIAs theory so asked him (day 2) to provide a diagram marking each clot drawn on a standard medical text book showing the arteries on the brain. In the event he produced the photocopy text book diagram with no marked clots.

[69]
In his verbal evidence Professor Brown placed the following TIAs as potentially causing the following symptoms:

a.
Option one: left arm, right side of face: TIA in base of brain, not in hemispheres (day 2 p5).

b.
Option two: left arm: TIA in right hemisphere; right face: TIA in left hemisphere: (day 2 p8).

c.
Option 3: nausea and vomiting: brain stem TIA (day 2 p31).

d.
Option 4: weakness and vomiting: brain stem TIA (day 2 p 33).

e.
Option 5: disturbed speech: cerebellum TIA (day 2 p 35).

f.
Option 6: visual disturbance, right facial droop and speech problems: embolus into posterior system, vertebral artery and basal artery and posterior cerebral artery (day 2 p66).

g.
Option 7: left arm: could be caused by right hemisphere TIA or TIA going into the arm affecting the motor fibres (day 2 p68).

[70]
Professor Brown went on to consider causation and stated that if the Claimant had suffered TIA in March 2015 an MRI scan would have shown a small area of infarction. This does not tie in with his theory that there were two TIAs. However both parties accept that if the Claimant did suffer TIAs the MRI would probably have confirmed the diagnosis. He went on to conclude that other investigations including blood tests, echocardiography, heart monitoring and angiography or CT angiography would not have shown any relevant abnormalities. He flirted with the possibility that investigations would have revealed intermittent atrial fibrillation (AF) but did not advise that AF would have been found on the balance of probabilities. If it had been found Warfarin would have been given instead of Aspirin. The Claimant did plead Warfarin should have been provided but did not pursue that case at trial.

[71]
He advised that even if the MRI scan had been normal the TIA clinic would have treated the Claimant with Aspirin. This was because TIA is a differential diagnosis sometimes not picked up on MRI and the Claimant would have gained some benefit from Aspirin just in case.

[72]
Dealing with whether Aspirin would have prevented the May 2015 stroke, Professor Brown advised that on balance Aspirin would have prevented her stroke. He relied on a 2016 paper by Rothwell, which he asserted demonstrated that Aspirin reduced the risk of recurrent stroke by 60% during the first 12 weeks after TIA. He did not split up the 12 week period despite the fact that Rothwell did. He used the general figure from Rothwell (B3p858) under the heading "result" (or the figures from the table at B3p861, lines 1 and 6 under the heading 6-12 weeks for "any ischaemic stroke").

[73]
In his verbal evidence Professor Brown drilled down into the Rothwell paper and produced an expanded version of B3p862 showing the graph for the effect of Aspirin in reducing the risk of disabling or fatal ischaemic stroke. At 8 weeks, the effect is well over a 50% reduction in the risk. It was his evidence that these figures which applied to the elderly patients in the studies equally would apply to a 29 year old woman whatever the genesis of the blot clots causing the May 2015 stroke which she suffered.

[74]
He went on to deal with the specific issue raised by Dr. Sare of whether Aspirin was effective in reducing the risk of recurrent stroke where the stroke is caused by emboli from the heart rather than emboli caused by atherosclerosis. He accepted that the Claimant's stroke in May was not caused by atherosclerosis, on the balance of probabilities. However, he advised that Aspirin was equally effective for preventing strokes caused by clots generated in other areas of the body which then travel to the brain. He stated that he would expect Aspirin, being an antiplatelet agent, would have a similar effect wherever the thrombus was formed. He accepted that the published studies suggested Aspirin was more effective in preventing large vessel atherosclerotic stroke than other types. In support of this view he performed a destructive critical analysis of a paper from 2015 by Rajkumar relied on by Dr. Sare which asserted the opposite view. He noted that at B1p188 in his report that the authors of that study concluded "that Aspirin therapy increased the proportion of patients with cardioembolic stroke". The paper which is a meta analysis of other papers does state that result (B3p632). He criticised the authors' conclusions stating they were not justified from the published data. He stated that the authors made "misleading statements" about their control population. He stated that the reasons to prescribe Aspirin prior to a stroke would be either prior cardiac disease or TIA. He criticised the authors for failing randomly to allocate patients to the two treatments. He advised that Aspirin is likely to be equally effective in the first three months whether the underlying cause of the stroke is embolism of a thrombus formed in the heart or from atherosclerosis or from the small vessels or some other cause.

[75]
Professor Brown provided a letter dated the 13th of February 2020. This letter analysed the cause of the symptoms in March on the basis of Mr Eastham's subsequent recollection that the facial droop was on the left side not the right. As a result of my findings of fact it is no longer relevant.

[76]
Professor Brown provided a CV which consists of 53 pages setting out his qualifications and published papers. I am afraid that this approach reflected his approach to his analysis of the causation of the symptoms in March. It lacked focus, was bulky and hence took up a lot of paper, whereas a short focused CV would have been more helpful.

[77]

I consider that Professor Brown's explanations became too complex; his analysis was less than adequate and that his evidence became contorted in relation to where the TIA lesions were in the brain. Whilst Professor Brown carried out a detailed analysis of the paper of Rajkumar, he failed to carry out a necessary analysis of the paper on which he relied: Rothwell. He descended into the factual evidence in a less than impartial manner by seeking to play up Mr Eastham's evidence. He relied on the Rosier test as a full diagnostic test whereas it is in fact a primary filter used for onward referral to a TIA clinic. He appeared to be grasping at straws by suggesting that multiple in situ TIAs could be caused by sticky blood - an explanation never previously put before the court in his report or the joint report. Professor Brown's evidence on day 2 in relation to the use of the term "in situ" was unimpressive. Whereas in his report and the joint report Professor Brown accepted that the most likely explanation of the genesis of multiple TIA would be from the heart, yet in his evidence he refused to stick to that opinion and instead stated that the heart was not on the balance of probabilities the most likely source of multiple TIAs. Professor Brown failed to look in depth at the Rothwell study and particularly that part upon which he relied relating to whether Aspirin had any "significant effect" on preventing strokes caused by atrial fibrillation. He had accepted the critical analysis carried out by Dr. Sare in the joint report - that a tiny cohort of only 400 patients were included by Rothwell in that sub category - and of those the results were minuscule being 2 to 8 persons either in the control group or in the group that received Aspirin and the results were of no significant statistical value, but in evidence sought to undermine that agreement.

[78]
I listened carefully to Professor Brown's evidence. He is a highly experienced and intelligent expert. He retired from NHS practice some years ago. But I found myself confused on a number of occasions during his evidence. On some occasions I felt obliged to ask for clarification so as to be able to understand what Professor Brown was saying and when explanation was provided it was, on occasion, less than clear. I find that during his evidence Professor Brown was troubled by a lack of clarity of thought. He struggled to provide a coherent theory to support his conclusion that the march of symptoms was symptomatic of multiple TIAs. For instance in his report he did not conclude that the march of symptoms was caused by TIAs which caused migraine. He theorised about that but did not so conclude. Yet in his evidence he did allege the TIAs caused migraine. The Claimant's case was not pleaded on the basis of TIAs causing migraine. His evidence on the meaning of "in situ" was contradictory. His evidence on the likely source of multiple TIAs was confusing.

Experts' joint report

[79]
I shall now turn in detail to the joint report between Professor Brown and Dr. Sare. It is 30 pages long. I will focus on those parts that relate to the findings of fact that I have made above and leave Aspirin and risk reduction to later on in this judgment.

[80]
In summary the experts agreed that the Claimant suffered a stroke in May 2015 which caused left sided neurological impairment and epilepsy. That stroke was caused by an occlusion of the right middle cerebral artery. Stopping there, it is apparent that Professor Brown abandoned the opinion that he had previously given in his causation report as set out above on the left MC artery. He gave no explanation for doing so.

[81]

The experts went on to agree that the cause of the May stroke was unknown. They agreed an embolus was possible but also that in situ thrombus formation was possible. Stopping there Professor Brown in his evidence flipped from the classic definition of the use of the words “in situ”, which I understood to mean that the blood clot formed and caused a blockage at the same site in the brain, to a wider definition during questioning by defence counsel. The wider definition appeared, if I understood it correctly, to be that a blood clot could form at any relevant site in the blood system (in situ) and then travel from that site to the brain. Subject of course to the difference between the venous system and the arterial system, I find that his evidence in relation to the terminology “in situ” was less than impressive. It is clear to me that in the joint report the experts used “in situ” blood clot to mean that it grew where it caused the blockage, not that it travelled from afar.

[82]
In the joint report the experts went on to agree that if the stroke was embolic (so that it travelled to the brain as opposed to forming “in situ”) the embolus would have formed either in the heart or the arteries supplying the brain.

[83]
Dr. Sare assisted the court with a basic anatomy lesson in her verbal evidence. There are two parts to the circulatory system that are relevant here. One side of the heart: (A) pumps oxygenated blood from the heart up the Aorta and into the brain and also to the rest of the body. Having delivered the oxygen and other nutrients, the blood returns through the venous system to the other side of the heart (B). That other side of the heart (B) pumps the blood into the lungs where the blood is oxygenated and then returns to the first side of the heart (A) previously described. If a clot forms in the venous system then it will go through the 2nd so described chamber (B) and probably end up as a pulmonary embolism. That is not relevant to this case. What is relevant to this case is the first described side of the heart (A) which pumps oxygenated blood up the Aorta and into the brain and to the rest of the body. Therefore it seems logical that the primary sources for the genesis of a travelling blood clot to the brain are the heart and the aorta. Despite this and despite what was agreed in the joint report Professor Brown expressly refused to accept that the heart or the aorta were the most likely source of embolus which could split up and cause multiple TIAs in March 2015. In his evidence he stated that the cause was unknown and that cardiac or aortic causation for the embolus that split up into multiple TIAs was not, on the balance of probabilities, likely. So that was the Claimant’s case at trial.

[84]
The experts agreed that the Claimant had no known cardiac history prior to April 2015. A cardiac event arose in April 2015 and preceded the May 2015 stroke. They agreed that the April event was diagnosed as myocarditis. Professor Brown asserted that it was possible that the Claimant had asymptomatic myocarditis after April and running into May but Dr. Sare considered that unlikely in view of the absence of symptoms. She restricted the April symptoms to an acute event. Both experts agreed that subsequent to her stroke the Claimant’s heart function results in all tests were normal. There was no evidence of arrhythmia which could cause a clot to form in the heart and then potentially travel elsewhere and her echocardiograms both in April and in May were normal. Professor Brown favoured a cardiac cause for the May stroke but advised that the cause was not identified.

[85]
The experts agreed that inflammatory conditions like Ankolysing Spondylitis are associated with an increased risk of stroke.

[86]
The experts agreed that TIA is a mild form of ischaemic stroke where the symptoms last less than 24 hours.

[87]
They also agreed that particularly in younger women migraine was well documented as giving rise to a risk of subsequent stroke. The experts agreed that simultaneous TIA and migraine is uncommon. The experts agreed an increase in the risk of subsequent stroke arises in patients with migraine with aura.

[88]
In the joint report the experts were then tasked with going through the March 2015 symptoms, one by one, to advise the court on which symptoms were more characteristic of migraine and which were more characteristic of TIA. Importantly at paragraph 20(a) (the first one on the Claimant's agenda, not the second one on the Defendant's agenda) the experts agreed:

"…that migraine may occur with an aura. For the ease of the court we consider headache, photophobia, nausea etc to be features of headache, and aura refers to focal neurological symptoms which occur in association with migraine. These include the visual changes, speech disturbance, unilateral sensory loss, and unilateral weakness. We agree that weakness is a motor aura. We agree that ICHD has multiple different classifications of aura and that they classify migraine with a motor aura as "hemiplegic migraine" which they refer to as "sporadic" if there is no family history. We will refer to migraine with motor aura as "hemiplegic migraine" from now on in this statement."

"We agree that stroke and migraine with aura can have similar characteristics. These include visual changes, speech disturbance, unilateral sensory loss, and unilateral weakness. We agree that headache can occur in both conditions and is not strongly discriminatory. These symptoms taken on their own are therefore unhelpful in distinguishing the two conditions."

"We agree that photophobia is not a described feature of TIA or ischaemic stroke".

"We agree it is the pattern of symptoms is helpful (sic) in distinguishing the two conditions for example migrainous symptoms show a progression through different symptoms and evolve with time. ICHD notes:

" when aura symptoms are multiple, they usually follow one another in succession, beginning with visual, then sensory, then a phasic; But the reverse and other orders have been noted. The accepted duration for most aura symptoms is one hour, but motor symptoms are often longer lasting."

"we agree that TIA, when singular, will usually show simultaneous onset of multiple symptoms (eg speech and weakness). Rarely single TIA or stroke will cause staggered symptoms. GS's view was that in this case the first symptom is usually the last to resolve as this is the core of the stroke where blood flow is lowest. MB's view is that when more than one TIA follows another in quick succession (crescendo TIAs), it is the last symptom that is the last to resolve because it is almost always more severe than the preceding symptoms."

[89]
The experts also agreed that the Claimant suffered migraine with visual aura in 2010. Also at paragraph 22 (on the Claimant's agenda) the experts agreed that the Claimant had a history of migraine. This is an important agreement in my judgment. It did not support the line of questioning

which the Claimant pursued at trial of Dr. Sare, seeking to undermine that agreement, unless of course Professor Brown was resiling from his agreement.

[90]
They agreed that migraine is the most common cause of headache especially in women. That comment appeared to relate to the January 2015 headaches and visual disturbance which the Claimant suffered. Dr. Sare advised that the January 2015 headaches and visual disturbance were consistent with migraine. Professor Brown advised that the symptoms were atypical for migraine. I accept Dr. Sare's evidence on this point.

[91]
The answers in paras. 26 and 28 of the joint report (on the Claimant's agenda) go to the root of the issue. Dr. Sare diagnosed migraine as the cause of the Claimant's symptoms in March 2015.

[92]
At paragraph 28 of the joint report Dr. Sare advised that the symptoms and in particular their onset and offset were consistent with the march of symptoms classically described for migraine with motor aura. Further the photophobia is characteristic of migraine not TIA.

[93]
Both experts agreed that if the events in March 2015 were a migraine then they are properly described as a hemiplegic migraine sporadic in nature.

[94]
The experts agreed that both hemiplegic migraine and TIAs are rare disorders in 29 year old women without vascular risk factors. Dr. Sare advised that hemiplegic migraine was far more common in young women and that TIA was less common in young women.

[95]
Professor Brown advised in the joint report that:

"the history was not sufficient. The patient required further investigations to distinguish between hemiplegic migraine and TIA / minor stroke."

Yet he also wrote in the joint report:

"on the balance of probability, the focal neurological symptoms were the result of TIAs."

He then went on to say that the TIAs "probably precipitated migraine."

Dr. Sare

[96]
Doctor Sare is a busy and experienced neurologist working at Queens Medical Centre in Nottingham. She has been a consultant since 2011. She deals with stroke patients very regularly. She gave evidence in accordance with her report dated December 2019. In her opinion the Claimant suffered headaches with visual disturbance between January and March 2015. She had no neurological symptoms before the 26th of March 2015. She had visual disturbance on one occasion associated with a fall on the 27th of January 2015. She considered the symptoms before 26/27 March 2015 to be in keeping with migraine but not in keeping with TIA.

[97]

On the 27th of March 2015, on the basis of the medical records, Dr. Sare pointed out that the Claimant developed visual disturbance namely distorted vision, right sided facial droop and speech difficulty and then 20 minutes afterwards suffered left arm weakness. She also suffered headache and photophobia, nausea and vomiting. The facial droop resolved before 01:50 am and all of the symptoms except for the neurological left arm weakness resolved by 06:50 in the morning on the 27th of March.

[98]
Dr. Sare accepted that there was a differential diagnosis of TIAs causing migraine soon thereafter but advised the court that the symptom range and more importantly the march of the symptoms, namely their onset, progress and offset and the chronology therein was more in keeping with migraine with aura including motor weakness.

[99]
She considered that the distorted vision strongly favoured migraine instead of stroke. In TIA the visual loss would usually be unilateral. She opined that the progress over the first 20 minutes of the symptoms was more in keeping with migraine. She also advised that the tempo of resolution namely the visual disturbance, the speech difficulties and the face difficulties evolving first and resolving first and the arm weakness evolving second and resolving second strongly favoured migraine rather than TIA.

[100]
Finally she opined that photophobia, nausea and vomiting were typical of migraine unless it was a particular rare type of stroke, therefore she advised the court that it was more likely that the March 2015 symptoms were migraine.

[101]
She advised getting a report from a cardiologist on the April 2015 cardiac symptoms. I have seen no such report. She noted all the heart investigations were normal thereafter.

[102]
In relation to the May stroke Dr. Sare advised that it was large. She noted that the treating doctors concluded that the Humira pills which the Claimant was taking were the cause, but Dr. Sare did not agree. She noted the suspicion of Lupus but that the secondary testing excluded Lupus. She noted the echocardiograms during and after the May 2015 event showed that the heart was structurally normal and that tracing showed no arrhythmia and no palpitations. She advised that atrial fibrillation rarely occurs in structurally normal hearts. She noted that the Claimant’s heart was monitored after the stroke and even the stress of being in ITU and of having a cranial operation did not cause any heart arrhythmia. Echocardiography after the stroke showed no clots in the heart or reason for heart clots and therefore she concluded that atrial fibrillation was unlikely to be the cause of the stroke.

[103]
Dr. Sare applied clearly explained logic in a way that impressed me in the report and in her evidence. She considered whether the myocarditis, which was diagnosed in April, could have been present in March 2015 and hence could have been relevant to the March symptoms. But she advised that there was no headache or vomiting in April when it emerged with symptomatic chest pain and it had not caused chest pain at any time before April and so she did not see the logic of how myocarditis could have caused a blood clot which caused a TIA in March when it was completely asymptomatic until April. In addition she noted the steep rise in Troponin (a marker of cardiac dysfunction) which suggested an acute myocarditis in April not a chronic condition.

[104]
Dr. Sare accepted that if the Defendant had performed an ABCD2 test the hospital would have started the Claimant on Aspirin in the accident and emergency department in March 2015 because the Claimant's score would have been four and therefore "high risk" for TIA or stroke. However, she also advised that it would have been likely after MRI that the TIA clinic would have diagnosed migraine and therefore they would have stopped Aspirin. Therefore the breach by failure to refer the Claimant to the TIA clinic was and is irrelevant to the outcome.

[105]
Dr. Sare advised that it is unlikely at the TIA clinic, if the Claimant had suffered a TIA, that atrial fibrillation would have been found. Therefore the Claimant would not have been prescribed Warfarin. She would have been prescribed Aspirin (if she had suffered a TIA) and Dr. Sare accepted that Aspirin would have reduced the risk of stroke if the cause of the May stroke was not cardiological. However in her report she separated out, as Rothwell did, the various periods post TIA. Rothwell showed Aspirin was effective in reducing the risk of all recurrent strokes (large and small) by 58% in the first six weeks and 40% between weeks 6 and 12. I note that B3p861 and the table there in at line 1 sets out those figures. The Claimant's big stroke in May occurred just over eight weeks after the March events and therefore on Dr. Sare's evidence and reading of Rothwell she would have fallen into the 40% reduced risk band. So Dr. Sare advised that the stroke would not have been avoided on the balance of probabilities. Finally Dr. Sare advised that Aspirin was minimally effective where the stroke was generated by an embolus arising in the heart.

[106]
In her evidence in court Dr. Sare focused on the symptoms and the pattern of symptoms in particular their onset, their chronology, their progression and their offset. Taking each symptom in turn.

[107]
Visual disturbance. Generally this can be caused either by migraine or by TIA. A TIA would cause visual disturbance either by the blockage being in the ophthalmic artery which would cause visual disturbance only in one eye or the clot being in the occipital lobe to cause disturbances in both. (Prof. Brown advised blurring could only be caused by a clot in the brain stem). The Claimant did not have a visual field defect in the sense of a blocked out shape in her vision and she did not have a unilateral visual field defect. It would be typical of TIA in one ophthalmic artery for the symptoms to be unilateral.

[108]
The Claimant's complaints of bilateral visual disturbance were of blurring and were "difficult to describe". Therefore on balance Dr. Sare favoured migraine for the visual symptoms. The Claimant had also suffered previous blurred vision on many occasions before March 2015.

[109]
Facial droop. I have already found that the facial droop was unilateral and on the right. Dr. Sare accepted that facial droop could occur either as a result of TIA or migraine. She advised that the right facial droop would need a clot to be in a specific part of the brain controlling the right side of the face if it were TIA.

[110]
Speech. Dr. Sare advised that is important to distinguish between:

(A)

dysarthria which would be more characteristic of migraine; that could be described as slurring or struggling to talk due to physical effects on the face; and

(B)
dysphasia which is more typical of TIA affecting a particular centre in the brain which would more likely cause confused speech itself or a primary language disorder.

Dr. Sare considered that the reports of the Claimant struggling to talk in the Claimant's evidence and that of Mr Eastham did not suggest that she was having difficulty finding the right words or was confused and so the symptoms were dysarthria and were indicative of slurring and hence leaned towards migraine.

[111]
Right sided headache. Professor Brown said he was "struck" by how the triage notes focussed on neurological deficit. Dr. Sare explained why the triage notes listed the presenting complaint as "neurological" rather than headache. This Claimant had suffered headaches through January 2015, so headaches were not new to her. I note that in her witness statement the Claimant asserts that headache was present at onset of the 26/7 March 2015 symptoms. Dr. Sare explained that the reason why the Claimant went to A & E was likely because she had developed a weak left arm and face droop which she had never before suffered. Dr. Sare advised that headaches are absolutely typical of migraine but only arise in a small percentage of TIA cases. The evidence was that headache arose in only about 30% of TIA cases, although Professor Brown stated it occurred in 20% of TIA cases (Day 2 p20). Therefore Dr. Sare advised that this factor favoured migraine.

[112]
Nausea and vomiting. Both experts agreed that nausea and vomiting are characteristic of migraine and not characteristic of TIA. A paper by Venkat (2018) provided by the experts showed that nausea and vomiting only occurred in 6% of TIAs. If it was caused by TIA then a stroke in the relevant region of the brain would be necessary to cause it. Professor Brown gave verbal evidence that these features together with photophobia were non specific and do not help distinguish (day 2 p27-31) but in the same breath he accepted they were common features of migraine and stated only that they "can occur in TIA". This was one of his less acceptable approaches to his evidence. If the feature was more likely in migraine, he would on occasion say it did not help distinguish. If was more common in TIA, he would say it did help distinguish.

[113]
Photophobia. Both experts agreed that photophobia was typical of migraine and that it was not typical of TIA.

[114]
Left arm weakness. Both experts agreed that left arm neurology was a common feature of TIA and a less common feature of migraine unless the diagnosis was hemiplegic migraine.

[115]
The march of symptoms: onset, progress, chronology and offset. Dr. Sare's main rationale for advising the court that the march of symptoms were hemiplegic migraine rather than multiple TIAs related to the pattern of the onset, development and offset of symptoms. In addition she relied upon the duration. The pattern in migraine, she advised, was that reduced cell activity started in one area of the brain and then spread out across the brain tissue so that symptoms developed and progressed for over 15 to 20 minutes. So the symptoms starting in the eyes and face and then 20 minutes later

moving on to the left arm fitted this pattern. Dr. Sare also advised that the ICHD summary confirmed the normal order for migraine was for symptoms to emerge in the same order that they emerged in the Claimant. In addition for migraine it is usual for the symptoms to turn off in the same order. So if A emerged then B, they would resolve A then B. That is what happened in the Claimant's case in March 2015. The arm symptoms came on after the face, speech, headache and visual symptoms and the arm symptoms lasted longer so ended after the face and visual symptoms. Dr. Sare advised that it was characteristic of migraine that the visual symptoms and the speech would start first and motor symptoms would come later. In contrast Dr. Sare advised that in TIA the symptoms very commonly all come on at the same time when a single clot gets stuck in the brain.

[116]
Organic Pattern. In relation to the pattern Dr. Sare explained that for the symptoms that I have found the Claimant to have suffered to be caused by TIA the Claimant would need to prove multiple TIAs arose and then caused migraine. This explanation was not Professor Brown's conclusion in his reports but was adopted by him in the joint report after discussing matters with Dr. Sare. The visual problems would be caused by a clot in the optical arteries (pleural) if they were in both eyes or in the occipital lobe. The facial droop would be another clot in a different position. The language dysphasia would be another clot in a different position, and the left arm symptoms would be another clot. Dr. Sare was very sceptical of a single clot in the brain stem causing the Claimant's pattern of symptoms. It was her evidence that the brain stem is like a junction box with the fibres very close together and that a clot there would likely cause left and right symptoms, not unilateral symptoms, and it would also cause other symptoms. Finally Dr. Sare relied on the photophobia which is characteristic of migraine not TIA so the TIAs would have to have caused migraine as well.

[117]
Dr. Sare analysed the cause of the multiple TIAs. For the Claimant to succeed in proving the multiple TIA explanation Dr. Sare advised that on the agreed evidence the cause of the Claimants TIA/s could either be "in situ or cardiac".

[118]
For the symptoms pattern I have found on the facts, it was Dr. Sare's evidence that the necessary multiple TIAs are unlikely to have arisen "in situ". This would require them to develop in 2 or 3 different arteries in the brain within 20 minutes of each other. Multiple TIAs developing independently in situ and coincidentally would more likely occur with a generalised vascular disorder or inflammation of the brain blood vessels. There was no such disease in this case in this Claimant.

[119]
Moving on to look at cardiac generation of multiple TIAs, Dr. Sare accepted that cardiac thrombus could explain multiple TIAs emerging at the same time, a blood clot created in the heart would move into the aorta and then the brain arteries and break up at the relevant junctions to spread smaller clots around the brain. However, I must take into account that it is the Claimant's case, on Professor Brown's evidence in court, that on the balance of probabilities, any multiple TIAs were not caused or developed in the heart. The experts agree that the cause is unknown. In addition Dr. Sare pointed out that there was no cardiac defect shown in any test in April or May 2015 and cardiac thrombus in the absence of any atrial fibrillation or structural heart defect or disease or inflammation is very unlikely. As for the Aorta causing the clot – Dr. Sare pointed to the angiography on 23.4.2015 (B4p1205) which showed that the Claimant's aorta was unobstructed. There was only minor plaque in the right coronary artery. So overall no source for multiple TIAs could be identified and the most likely source

(the heart) was not proposed on the balance of probabilities by Professor Brown as the likely source in his evidence.

[120]
Finally, in relation to the theory propounded by Professor Brown that because of the Claimant's stroke in May, that fact alone makes it more likely that she suffered a mini stroke (TIA) in March, Dr. Sare advised the court that only in a small percentage of serious stroke cases does a TIA precede the stroke. That was agreed in the joint report. So Professor Brown's theory on this is not a persuasive factor.

Findings on the March 2015 diagnosis

[121]
For the reasons set out above I prefer and accept Dr. Sare's evidence on the most probable diagnosis for the cause of the symptoms which the Claimant suffered on 27 March 2015. I reject Professor Brown's evidence on the diagnosis save where on day 2 (p56) he accepted that if the court finds the symptoms were caused by sporadic hemiplegic migraine that diagnosis would explain all of the symptoms which the Claimant suffered.

[122]
I find on the balance of probabilities for the reasons provided by Dr. Sare that the cause of the Claimant's symptoms on the 26th and 27th of March 2015 was migraine with motor aura (or hemiplegic migraine which is not familial). I consider that diagnosis to be more likely than multiple TIAs which triggered coincidental migraine symptoms.

Causation

[123]
As a result of the findings I have made above the last question about Aspirin is no longer relevant. However, in case it becomes necessary I shall set out my conclusions and findings on Aspirin and TIAs in relation to this case on the evidence I have heard and read.

[124]
I should first make clear that I have heard no evidence from a cardiologist or a haematologist. I think that I would have been assisted by such expertise.

[125]
It is agreed between the neurologists who gave evidence that Aspirin does not provide sufficient risk reduction against primary strokes to satisfy the threshold for the but for test of causation. The Rothwell paper confirms that. It is perhaps counter intuitive (as was stated in the discussion in the Rothwell paper without using the same word) that this drug would provide high short term protection against secondary (second stroke) clotting but would not provide high protection against primary (first stroke) clotting, but this is wholly a matter for medical science. That is the evidence before me.

[126]
In the joint expert report the experts set out the following evidence of their areas of agreement and disagreement.

[127]
The experts agreed that Aspirin is used to try to prevent stroke in patients with a high risk of stroke for instance with previous heart disease or previous strokes who are in sinus rhythm. However

Warfarin is used where there is demonstrated cardiac arrhythmia and where the patient has hypercoagulability (sticky blood).

[128]
In relation to the Rothwell paper (2016) the experts agreed this was a retrospective analysis of multiple other trials of Aspirin which themselves looked at stroke prevention long term after previous stroke. The authors in Rothwell however focused on looking at the effects of Aspirin in the first three months after TIA. The experts agreed it was the most useful paper providing evidence about Aspirin use in the first three months after TIA.

[129]
The experts agreed that Rothwell's general overall conclusion showed that Aspirin reduces the risk of stroke after TIA in the patients included in the study. They agreed that that reduction was 60% overall (for all strokes, large and small) as stated in the paper. They agreed the beneficial effects of Aspirin occurred in the first 12 weeks and not thereafter.

[130]
The experts did not agree on two key matters: (1) the effectiveness of Aspirin on the risk of strokes formed by different specific organic processes; (2) whether the results for strokes suffered (mainly) by men between the ages of 51 and up to very old age were relevant to a woman aged 29. These two issues are of course interlinked because in old age vascular and other diseases are more prevalent and the circulatory system is less smooth, clean and robust. They disagreed on the issue of whether Aspirin was more effective in relation to strokes generated by atherosclerosis rather than the heart. They disagreed on whether Aspirin would have prevented a secondary stroke in a 29 year old woman with no atherosclerosis like the Claimant.

Age

[131]
Professor Brown in the joint report accepted that probably only a few patients aged 29 (if any) would have been included in the Rothwell trials because it is very rare for TIA to occur in that age group. Dr. Sare advised that Rothwell's results were relevant only to the cohort studied in those papers. She pointed out that the average age of the cohort was 58 and above and 84% of patients included in the Rothwell studies were older than 51. Most were male and most had the traditional risk factors for ischaemic stroke. Therefore Dr. Sare advised that the Rothwell paper was of little relevance for a 29 year old woman with no traditional risk factors for stroke and in particular where the stroke was not caused by atherosclerosis.

[132]
Professor Brown was prepared to make the jump to say that Aspirin would be as effective in young women as it is in older men. This approach overlooks age and the cause of the clot, for instance: (1) in situ or (2) atherosclerotic or (3) cardiac or (4) aortic or (5) idiopathic. It was agreed that the Claimant did not have atherosclerosis (bunged up arteries).

[133]
Dr Sare's evidence was that the Rothwell paper only produced a 40% reduction in risk between weeks 6 and 12 in the cohort. Having carefully read Rothwell and in particular the table at page 861, that 40% figure comes from lines 1 and 6 which relate to "any ischaemic stroke".

[134]

I do accept as a fact, because both experts so advise, that Aspirin may be effective in producing some reduction in recurrent stroke arising from all causes in the first 12 weeks after TIA. The question is: by how much in this case for this Claimant? To answer that I need to take into account not only her gender, but her lack of atherosclerosis and her lack of any proved heart condition in March 2015, her previous Ankolysing Spondylitis and her lack of any systemic or acute vascular disease and her age.

[135]
The Claimant is a young woman of 29 and the issue in this case is: "how do the Rothwell results relate to the Claimant?". The supplementary appendix to then paper by Rothwell considered the effects of Aspirin and the patient's age, but the youngest category therein was "under 65" and both other categories were over 65. There was no sub category for under 50, let alone under 40 or under 30. Professor Brown also relied on the practice of neurologists prescribing Aspirin to children "because it is generally believed that it is effective" (day 2 p49) and in his experience it worked equally well. I did not find that evidence sufficient.

AF

[136]
In relation to the evidence of the effectiveness of Aspirin in cases involving stroke caused by atrial fibrillation, in the joint report both experts noted that Dr. Sare had carried out a critical analysis of the base data in Rothwell and counted that less than 400 patients in the Rothwell studies (including the control groups) had atrial fibrillation. The experts agreed that Rothwell had not published the precise figures for patients in the study with AF so Dr. Sare did the detailed analysis. The paper itself shows that the confidence intervals for the figures for the effect of Aspirin on patients with AF produced results which are not significant. Dr. Sare explained that the sample size was far too small. Having accepted Dr. Sare's figure of 400 in the joint report, in his evidence Professor Brown abandoned his joint report position and sought to undermine the critical analysis done by Dr. Sare. This was unimpressive in my judgment.

[137]
I accept Dr. Sare's evidence on the atrial fibrillation issue. It was she who carried out a critical analysis of the base data in relation to the cohort included in the Rothwell papers. She counted around 400 atrial fibrillation patients and showed the data behind Rothwell's conclusion (that Aspirin had a "similar effect" – p863) was insufficient to justify the conclusion. I find that no significant conclusion could be drawn in relation to the effect of Aspirin on recurrent stroke in patients with atrial fibrillation. More so those who are young women. I accept her criticism. To be clear, Dr. Sare said in her evidence this defect does not undermine the importance of Rothwell's findings for the large older cohort that were included in the study.

[138]
Dr. Sare relied on the Nice Guidelines CG180 at page 8. She also relied on the Cochrane reviews by Aguila in 2005 and 2007, which showed that Aspirin did not have a significant effect on preventing stroke in those with atrial fibrillation but Warfarin did. She made the powerful point that it is for those reasons that the Nice Guidelines and Royal College Guidelines were not changed after Rothwell and Warfarin is still the treatment of choice in patients with atrial fibrillation. Whereas, if Professor Brown's evidence in relation to Rothwell had been accepted generally within the medical community, Aspirin would be advised and provided as another treatment of choice where the patient had atrial fibrillation. I found Dr. Sare's evidence persuasive on this. In addition, no evidence was put before the court about whether there were any women under 30 in the Rothwell study at all.

[139]
On balance I accept that Rothwell shows that Aspirin is likely to be effective at preventing substantially more than 50% of recurrent serious or fatal strokes for those within the Rothwell study age group with the organic characteristics that come with ageing in the first 12 weeks after TIA (Bundle 3 p 862A) and so also at week 8.

Cardiac clots

[140]
I do not accept that it is proven on balance that the same percentage: (60% or indeed a higher reduction in risk for very serious stroke [B4p862A]), applies to a 29 year old woman for multiple TIAs most probably generated by her heart in weeks 6-12 after TIA. If the March 2015 symptoms were caused by multiple TIAs I find that Dr. Sare's evidence is more likely correct and that they would have been caused by the heart. If they were caused by the heart the most likely cause was atrial fibrillation. I find on the evidence before me that the claimant's case on the effect of Aspirin in preventing secondary cardiac clots by over 50% in women aged 29-30 is not proven.

Atherosclerotic clots

[141]
Dr. Sare advised that Aspirin was more effective in preventing recurrent stroke where the cause of the stroke was atherosclerosis. Professor Brown agreed it was effective in preventing recurrent strokes generated from that source. I accept that evidence. However the Claimant did not have atherosclerosis so it does not resolve the issue.

Idiopathic clots

[142]
The Claimant's case was advanced on the basis of Professor Brown's theory that if the court did find that the March symptoms were caused by TIAs then there were multiple TIAs and these were idiopathic (of unknown origin and not from the heart on the balance of probabilities). On that basis he asserted that Rothwell applied to this Claimant and Aspirin would have prevented her May stroke (because the reduction of the risk would have been over 50%).

[143]
For the reasons given by Dr. Sare I do not accept on the balance of probabilities, that the Rothwell figures for (mainly) men, 84% of whom were over 51, are proven to apply to the Claimant. For example looking closely at the data on p861 in weeks 6-12 post TIA there is only a 40% reduction achieved in all ischaemic strokes (line 1, "any Aspirin v control", the figure is 0.60); and the same for "Aspirin v control" (line 6). Using these figures the case is unproven on causation.

[144]
The figures for "disabling or fatal stroke" show a 52% reduction (lines 2 and 7, the figures are both 0.48) which would be enough but it only applies on my findings to the population cohort in the study (much older and mainly men). Only a minor variation in the studied cohort or in the organic causes of the strokes would make a substantial difference to causation. The Claimant was a 30 year old woman at the time of the May stroke.

[145]

I find that the Claimant has not discharged the burden of proof in relation to Aspirin. If taken as prescribed from 27th March 2015 whether Aspirin would have prevented the Claimant suffering the May 2015 stroke is not proven on the balance of probabilities on the evidence before me.

Conclusions

[146]
As to each issue:

1.
"Was the facial droop that the Claimant experienced right-sided or left-sided?" I find right sided.

2.
"What was the extent and timing of onset of the left-sided weakness that the Claimant experienced?" I find 15-20 minutes after the onset of the other symptoms.

3.
"Were the symptoms with which the Claimant presented on 27 March 2015 as a result of TIAs precipitating migraine or sporadic hemiplegic migraine?" I find sporadic hemiplegic migraine.

4.
"If the 26/27 March 2015 event was as a result of TIAs precipitating migraine, would the stroke of 27 May 2015 have been prevented or made non- or minimally injurious as a result of the Claimant taking the Aspirin she would have been prescribed before and after she had been seen in the emergency TIA clinic on 27/28 March 2015." I find that causation is unproven on the balance of probabilities.

[147]
For the reasons set out above I dismiss the claim and enter judgment for the Defendant.

[148]
I award the Defendant its costs to be paid by the Claimant on the standard basis to be assessed if not agreed. This costs order is subject to any further submissions to be made to me via my clerk by the parties in writing by 4pm on Tuesday 25th January 2022.

Ritchie J