

Witness Name: Dermot Gleeson  
Statement No: WITN7084001  
Exhibits: WITN7084002-018  
Dated: November 2022

## **INFECTED BLOOD INQUIRY**

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### **WRITTEN STATEMENT OF DERMOT GLEESON**

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I provide this statement in response to the request under Rule 9 of the Inquiry Rules 2006 dated 27th June 2022

I, Dermot Gleeson, will say as follows: -

#### **Section 1: Introduction**

1. My DOB is GRO-C 1953. Since 2018 I have been a Locum Consultant Hepatologist at Sheffield Teaching Hospitals (STH) NHS Foundation Trust, prior to which (from 1993-2018) I was a Full time Consultant Hepatologist at STH. My work address is: Liver Unit, AO Floor Robert Hadfield Building, Northern General Hospital, Sheffield S5 7AU.

#### **Section 2: Responses to criticisms by W2700**

2. May I first apologise for the long delay in replying to the request and making this statement. The request was received in December 2020. We attempted to find Mrs Lowe's hospital notes to address the questions raised but were unable to do so. It would appear they have been destroyed. We were therefore directed to the NHS Digital Facility which contains some GP records of deceased patients. We contacted them in April 2021, and they replied quickly seeking further information. Unfortunately I missed this email and it was drawn to my attention in January 2022. I then provided the extra information and on 21 January 2022, I received Mrs

Lowe's primary care records (relevant records are exhibited) which include several letters sent from our department and also from the department of Gastroenterology in Rotherham. I believe that these accurately summarise the essential details of Mrs Lowe's liver condition. My initial report was submitted to the Directorate Governance Dept on 22 February 2022 further enquiries for medical records were subsequently made on my behalf by the Trust, this unfortunately added additional delay in finalising this response.

3. In his statement, Mr Lowe says that his mother Barbara Lowe was infected by blood transfusion during a mitral valve replacement operation on 13 February 1988. He believed that she had contracted hepatitis C. Some years later Mrs Lowe (because of deteriorating health) saw her doctor and was referred to the Royal Hallamshire Hospital where she saw me in October 2003 (Exhibit WITN7084008). She was accompanied by her daughter Rachel.
4. Mr Lowe stated that I "explained in a very blunt and unsympathetic manner that my mum had hepatitis C and that she had been infected from the blood transfusion in 1988". Mrs Lowe and Rachel were not given any information about hepatitis C. It is stated, in the witness statement provided by Mr Lowe, that Mrs Lowe was given Interferon for hepatitis C, but she received no advice at any stage in regard to accessing the financial support that patients who contract hepatitis C from blood products are entitled to.

My summary of GP records, including correspondence from Royal Hallamshire and Rotherham Hospitals is as follows:

5. In early 2003, Mrs Lowe was referred to Dr Prasad, Acute Medicine Consultant, in Rotherham because of fatigue and abnormal liver tests. Dr Prasad saw her on 15 January 2003 and organised an ultrasound scan and blood tests to establish the cause of the abnormal liver tests (Exhibit WITN7084002). On 3 February 2003 she was referred to Dr Basumani, a Gastroenterologist, with experience of treating liver disease. Dr Prasad's referral letter states that Mrs Lowe had tested negative for hepatitis C antibody and hepatitis B surface antigen (Exhibit WITN7084003). However, she had another antibody called the anti-nuclear antibody and her serum immunoglobulin G was elevated. This combination of tests suggested a very different form of hepatitis called auto immune hepatitis.

6. On 24 February 2003 Dr Basumani wrote to me about Mrs Lowe. He mentioned her fatigue and abnormal liver tests and also that she had significant memory problems, possibly dating from a stroke. His letter mentions again that her "hepatitis B and C serology is negative" but, she had an anti-nuclear antibody and a raised serum immunoglobulin G. He thought that she had autoimmune hepatitis and requested that I organise a transjugular liver biopsy to make a final diagnosis of her liver condition (Exhibit WITN7084004).
7. According to my clinic letter, I saw Mrs Lowe on the 17 June 2003. I agreed that she needed a liver biopsy to confirm a diagnosis of auto immune hepatitis. Again, I noted the negative hepatitis B and C tests from Rotherham (Exhibit WITN7084005). A transjugular liver biopsy was performed on 18 August. Examination of the biopsy showed portal inflammation and interface hepatitis with plasma cells, consistent with moderately active auto immune hepatitis.
8. Mrs Lowe was commenced on corticosteroid drug treatment on 18 September 2003 (Exhibit WITN7084006). This is the standard treatment for auto immune hepatitis. The dose was reduced a week later on 25 September 2003 (Exhibit WITN7084007) as she was not sleeping well, and I reviewed her again on 9 October 2003 (Exhibit WITN7084008). She felt better, and her liver blood test, the so-called ALT enzyme (which when raised, indicates liver inflammation) had returned to normal. I recommended that she start a second drug Azathioprine for the autoimmune hepatitis. I asked that this be done in Rotherham and that she continue to be monitored there. Mrs Lowe was then followed up in Rotherham (Exhibit WITN7084009). Because she developed a rash thought to be related to Azathioprine this was stopped in early 2004 however, she continued on a reduced dose of Prednisolone (steroid).
9. Mrs Lowe was referred back to our unit in December 2005 after over 2 years treatment with Prednisolone (Exhibit WITN7084010). A transjugular liver biopsy was repeated and this showed no scarring of the liver (improved from stage 3 scarring on the original biopsy). In addition, there was minimal chronic hepatitis, which means that her auto immune hepatitis had gone into remission. This is normally regarded as a good response to steroids (normal serum ALT enzyme, resolution of hepatitis on liver biopsy).

10. Mrs Lowe continued to be followed up thereafter by Dr Basumani in Rotherham. At one stage she stopped the Prednisolone. Her serum globulins became abnormal in 2007 (Exhibit WITN7084011). Although the serum ALT enzyme remained normal there was concern about the autoimmune hepatitis reactivating. Thus, she restarted Prednisolone shortly thereafter and (from the outpatient letters) remained on a small dose of Prednisolone for several more years (Exhibit WITN7084012).
11. She was unfortunately diagnosed with leukaemia in 2011. This was managed by the Haematology Department in Rotherham (Exhibit WITN7084013). She continued to see Dr Basumani for monitoring of the auto immune hepatitis. This however, appeared to be in remission in 2012 when she was taking Prednisolone 5 mg od (Exhibit WITN7084014). In 2013, the leukaemia was unfortunately progressing and there was a plan to use stronger drugs to try and control it. Routine screening prior to commencing these drugs tested for hepatitis B and C markers and these were absent, indicating a negative result (Exhibit WITN7084015). . She was tested again in January 2014 for hepatitis B and C and again these were negative (Exhibit WITN7084016). From the results I have seen, between 2004 and 2014 her ALT enzyme levels were consistently normal (Exhibit WITN7084017).
12. Unfortunately, Mrs Lowe developed chronic kidney disease in 2009 and aortic stenosis in 2014, the year she died. I understand the causes of death were cardiogenic shock, calcific aortic stenosis, and chronic lymphatic leukaemia. Her liver condition was not mentioned as a cause of death.

### **Conclusion**

13. I can confirm that I saw Mrs Lowe in October 2003 and again in 2005, although I do not remember the consultations. Having reviewed the medical records I cannot accept that I told her she had hepatitis C. Mrs Lowe did not have hepatitis C, either when I saw her in 2003 or subsequently. She had a very different form of hepatitis, called auto immune hepatitis.
14. The term hepatitis simply means inflammation of the liver. There are many causes, and they include viral infections (such as hepatitis B and hepatitis C). However, hepatitis is often due to non-infective causes; these include alcohol and reactions

to drugs. In addition, there is a non-infective condition which is what Mrs Lowe had, called auto immune hepatitis where the body's immune system no longer tolerates a body organ and causes inflammation of that organ. Examples of auto immune diseases include rheumatoid arthritis, and psoriasis. Auto immune hepatitis is a typical auto immune disease. It is not thought to be related to infection. Our unit looks after hundreds of patients with auto immune hepatitis. They are routinely checked for hepatitis C and we would not make a diagnosis of auto immune hepatitis if testing for hepatitis C was positive. Mrs Lowe was negative for hepatitis C in 2003 and she had blood tests that suggested auto immune hepatitis. The liver biopsy supported this diagnosis. She had a good response to steroid treatment. Her liver tests returned to normal, and a repeat biopsy two years later showed much improved appearances. As far as I can tell from the records, she remained in remission for the final years of her life.

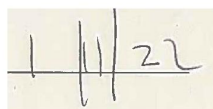
15. Mrs Lowe was subsequently looked after by Dr Basumani, who is an experienced Gastroenterologist and Hepatologist. Again, I find it inconceivable that Dr Basumani would have told Mrs Lowe that she had hepatitis C. All his letters indicate (as do mine) that she had auto immune hepatitis.
16. I have been through the GP's other records and can find no reference to hepatitis C nor can I find any reference either in the GP records or in hospital notes to the effect that Mrs Lowe received Pegylated Interferon.
17. In regard to the other points raised, I do not believe that Mrs Lowe was entitled to the financial assistance referred to in his letter, as her liver condition, autoimmune hepatitis, was not due to a blood borne virus (hepatitis B or C) and was not caused by the blood transfusion.
18. I apologise if my manner caused offence to Mrs Lowe or her family and if I did not make the issues raised within this statement adequately clear to Mrs Lowe and her daughter when I saw them in October 2003.
19. Subsequently, I have been sent the clinical records, covering the time when Mrs Lowe was seen by me in 2003 and 2005 and had two liver biopsies. Essentially, they corroborate what I stated above. In my written clinic record, I note the

Rotherham tests, including that Hepatitis B and C markers were negative and the diagnosis was "probable autoimmune hepatitis". I did not record what I said at the time to Mrs Lowe and her daughter or if they asked me anything about hepatitis C following her operation in 1988. If such a conversation had occurred, I think I would have recorded it, although I cannot be sure.

### **Statement of Truth**

I believe that the facts stated in this witness statement are true.

Signed  GRO-C

Dated 

### **Table of exhibits:**

<b>Date</b>	<b>Notes/ Description</b>	<b>Exhibit number</b>
2003.01.15	Letter from Dr Prasad to Dr Venkataram	WITN7084002
2003.02.03	Letter from Dr Prasad to Dr Basumani	WITN7084003
2003.02.24	Letter from Dr Basumani to Dr Gleeson	WITN7084004
2003.06.17	Letter from Dr Gleeson to Dr Basumani	WITN7084005
2003.09.18	Letter from Dr Gleeson to Dr Basumani	WITN7084006
2003.09.25	Letter from Dr Gleeson to Dr Venkataram	WITN7084007
2003.10.09	Dr Gleeson letter to Dr Basumani	WITN7084008

2004.01.03	Rotherham follow up letter	WITN7084009
2005.12	Referral to STH	WITN7084010
2006.07.19	Letter to Dr Venkataram	WITN7084011
2007.08.15	Letter to Dr Venkataraman	WITN7084012
2011.08.02	Referral from Dr Venkataraman	WITN7084013
2012.02.23	Letter to Dr Venkataraman	WITN7084014
2013.01-10	Letter to Dr Venkataraman	WITN7084015
2013.05.20 / 2014.01.30	Rotherham HCV results	WITN7084016
2004-2014	Medical results	WITN7084017