Witness Name: Prof Graham Foster
Statement No.: WITN3042001
Exhibits: WITN3042002, WITN3042003
Dated:2 April 2019

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF PROFESSOR GRAHAM FOSTER

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated 5 March 2019.

I, Professor Graham Foster, will say as follows: -

Section 1: Introduction

1. I am the Professor of Hepatology at Queen Mary University of London, Blizard Institute, 4 Newark Street, London E1 2AT. My date of birth is GRO-C 1959.
2. I am a consultant hepatologist at Barts Health where I was appointed in January 2003. I was formally a consulted Hepatologist at St Mary's Hospital, London. I qualified in 1983. My qualifications are BA (Oxon), MB BS (London), PhD (London) FRCP (London). I specialise in the management of patients with chronic hepatitis C infection and am the NHS England Clinical Lead for Viral Hepatitis, chairman of the NHS England Hepatobiliary Clinical Advisory Group and an Ex-President of The British Association for the Study of the Liver. I am the editor of The Journal of Viral Hepatitis, a trustee of The Hepatitis C Trust and I run a clinical and laboratory research program studying chronic hepatitis C infection. I manage a large cohort of patients with viral hepatitis and lead the clinical service at Barts Health where we manage large numbers of patients with decompensated cirrhosis.
3. I am the NHSE Clinical Lead for the Operational Delivery Networks for the treatment of patients with chronic hepatitis C and I am chairman of the NHSE hepatobiliary CRG

Section 2: Responses to criticism of Della Hirsch

4 I was responsible for the management of Mr Hirsch during his final illness. In order to prepare this report I have reviewed the medical records at The Royal London Hospital and relied upon my recollection of events.

5 I note from the statement from Mrs Hirsch (the patient's mother) that she has raised the following concerns:

- That because I failed to conduct a transient elastography 'Fibroscan' evaluation on Mr Hirsch, he received treatment with a combination of drugs that was inappropriate for someone with his degree of liver impairment and that a Fibroscan test would have identified liver impairment and prevented his death
- That I prevented Mr Hirsch from participating in a clinical trial.

6 In order to respond to this concern, I hope it may be helpful to provide a brief chronology of my involvement in Mr Hirsch's care.

7 Mr Hirsch was under my care with cirrhosis of the liver secondary to chronic hepatitis C infection, following administration of contaminated blood products. He had been under my care at The Royal London Hospital since February 2009. In 2011 it was my opinion, based on the results of his blood tests (chiefly the low platelet count) and the results of a fibroscan performed at The Royal Free Hospital that was communicated to me in a letter by Dr Patch (included in this report) that Mr Hirsch had cirrhosis of the liver. Since Mr Hirsch had liver cirrhosis he was at high risk of developing liver cancer and decompensated cirrhosis. In 2011-2012 new drugs for hepatitis C (protease inhibitors) became available. The drugs were newly introduced and although some side effects were recognised the full toxicity profile was not understood, as is usually the case with newly licensed drugs. In common with many liver units around the world my unit prioritised patients with cirrhosis for treatment, as such patients were at high risk of disease progression without therapy and it was judged that the risks of continued infection outweighed the risk of therapy.

8 In late 2011 I discussed the risks and benefits of therapy with Mr Hirsch and his family and he made an informed decision to undertake a course of antiviral therapy with pegylated interferon, ribavirin and telaprevir. He had a further consultation prior to initiating treatment with the viral hepatitis nursing team. He was supervised by myself and my team who had considerable experience with this drug, having participated in the clinical trials. As Mrs Hirsch indicates in her report I informed the family of the novelty of the treatment and the risks and benefits.

9 In January 2012 Mr Hirsch developed a rash during treatment with telaprevir – this was a recognised complication but the rash was unusual and, as Mrs Hirsch indicates, given the recent introduction of these drugs there was much discussion at liver conferences about the side effect profile and much sharing of information to

assist clinicians' management. As Mrs Hirsch makes clear I discussed the case with internationally experienced colleagues, who supported the approach that we were taking. These discussions were shared with the patient and his family who continued to play an active role in the shared decision-making. An immediate dermatology review was arranged when Mr Hirsch developed skin lesions and my recollection is that he was seen on the same day of the referral

10 During therapy Mr Hirsch sadly developed sepsis. He was brought immediately to The Royal London Hospital where he received treatment from our intensive care team. Sadly he did not respond to therapy and died. I chose to visit the Intensive Care Unit on the day after Mr Hirsch was admitted (Saturday) and I spent some time discussing the case with the lead ITU consultant and we agreed that antibiotic therapy and full supportive care should continue. I took the opportunity to speak to the family and to comfort them at this difficult time. The medical records document a discussion with myself, Mr Hirsch's family and the ITU consultant and note that 'the family thanked us for our care'.

11 I hope I can now respond to the concerns raised.

12 In response to the concern that because I failed to conduct a transient elastography – Fibroscan - evaluation on Mr Hirsch, he received treatment with a combination of drugs that was inappropriate for someone with his degree of liver impairment. I would like to explain that at that time Fibroscan testing was in its infancy and its value was the subject of debate. Some believed it reliable but others were not convinced that it was sufficiently accurate for clinical use. At that time the NHS had not reached a decision on its value and Fibroscan was approved for use in the USA only in 2013. It is therefore correct to say that I, and a large number of hepatology colleagues, was sceptical about the value of the Fibroscan at that time. Further research has now concluded that Fibroscan is helpful in excluding liver cirrhosis, but it is much less accurate in determining other stages of fibrosis. Mr Hirsch had undergone a Fibroscan test at the Royal Free Hospital in July 2007 and the results, a score of 10.5, was known to me. A Fibroscan score of 10.5 is relatively high and indicates significant scarring. Therefore prior to antiviral therapy my clinical assessment, based chiefly on his blood tests and supported by the Fibroscan in 2007 was that Mr Hirsch had cirrhosis of the liver and a further Fibroscan would have added no additional diagnostic information. There was no evidence that a Fibroscan score could be used to determine liver prognosis and no evidence that a Fibroscan could be used to stratify patients who were more or less likely to develop complications with antiviral therapy. This remains the case today.

13 I would like to explain that, although it is now recognised that telaprevir is associated with life threatening septic events, this had not been noted during the clinical trials and was only identified after licensing (after Mr Hirsch had died). However there is no data to support the suggestion that a Fibroscan would have been useful to stratify patients into those at risk of infection.

14 When telaprevir was introduced in 2011 it was seen as a major advance in the treatment of hepatitis C. My unit was one of the first to offer this therapy to NHS patients and a number of patients with cirrhosis were successfully treated. I appreciate that it is no comfort to the family to learn that all of the other patients so treated survived and continue to attend my clinic. By contrast patients with viral strains that were not responsive to telaprevir were not offered antiviral therapy until some years later and, sadly, many of those who were seen in my clinic in 2011 have subsequently died from complications of cirrhosis – chiefly liver cancer. In 2011 we believed that the risks of death from complications of cirrhosis greatly outweighed the risks from telaprevir-based treatment and subsequent events have confirmed this.

15 In response to the concern that I did not allow Mr Hirsch to participate in a clinical trial. I have no recollection of this and the notes do not record any evidence that access to a clinical trial was requested or denied. The Royal London Liver Unit has a very active clinical trial programme and we routinely offer all patients with viral hepatitis enrolment in clinical trials if they meet the entry criteria. Patients who wish to transfer to other units to participate in trials that are not taking place in our hospital are free to do so and as a matter of policy we support such transfers and we encourage patients to access novel treatments wherever it can be provided. It is noteworthy that many of the early clinical trials in hepatitis C excluded patients with clotting disorders.

16 I have reflected extensively on the decisions made in 2011 based on the information available to us at the time, and with the knowledge that we have now, and still consider them to have been correct.

Section 3: Other Issues

I do not believe there are other issues relating to this case

Statement of Truth

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

Signed GRO-C

Dated 2 APRIL 2019

Table of exhibits:

Date	Notes	Exhibit number
19/11/2007	Letter from Dr David Patch (Royal Free Hospital) to Dr D Twena regarding 'fibroscan' performed on Nicholas Hirsch	WITN3042002
17/03/2012	Patient notes	WITN3042003