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13th October 2010

MEDICAL REPORT

On

The Reverend David Black (deceased)

Date of Birth: 1st May 1937

Instructed By: The Penrose Inquiry
44 Drumsheugh Gardens
Edinburgh EH3 7SW

By: Dr B. T. Colvin FRCP FRCPath

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1. INTRODUCTION

1.1 I am Dr. Brian T Colvin FRCP FRCPath, formerly consultant haematologist at Barts and The London NHS Trust with a special interest in haemophilia and allied disorders. A curriculum vitae is attached.

1.2 Material Instructions

I have been instructed by The Penrose Inquiry to give an opinion and prepare a report for the court in the case of The Reverend David Black (deceased). I have been provided with a set of papers by the Inquiry including general practice and hospital medical records from Glasgow Royal Infirmary and Edinburgh Royal Infirmary and these documents have been used by me in preparing my opinion.

1.3 Summary of the Case

1.3.1 The case concerns The Reverend David Black who was born on 1st May 1937. He suffered from moderate haemophilia A and had a resting factor VIII:C level of between 3 and 7 i.u./dl (3-7%). He was treated at the Glasgow and Edinburgh Royal Infirmaries.

1.3.2 Mr. Black must have been first treated for his haemophilia when all that was available was cryoprecipitate but he had probably received his first dose of a large pool factor VIII by 1978 or earlier, after which time infection with hepatitis C must have occurred. His blood tests showed evidence of immunity to hepatitis B.

1.3.3 By the early 1980s there was evidence that he had "non A non B" hepatitis and by 1987 there was evidence of chronic liver disease. In 1996 a liver transplant was performed but the transplanted liver soon showed evidence of recurrent hepatitis C virus (HCV) infection and in 2002 antiviral therapy was commenced. His condition continued to deteriorate and he developed hepatocellular carcinoma.

1.3.4 Mr. Black died on 31st March 2003, the cause of death being hepatitis C infection in the transplanted liver with hepatocellular carcinoma, the infection having been originally transmitted by blood products.

2. THE ISSUES TO BE ADDRESSED

2.1

I am asked to comment on:

2.1.1 The nature of the haemophilia from which Mr. Black suffered

2.2.2 The treatment he received

2.2.3 Whether or not the treatment and management of Mr. Black's haemophilia was reasonable and appropriate.

3. THE FACTS

3.1 David Black was born on 1st May 1937 and seems to have been first treated in the 1960s when cryoprecipitate became available after 1965/6. A diagnosis of haemophilia A was made although this was questioned in 1971 when the possibility of von Willebrand disease (VWD) was considered. In fact the correct diagnosis was almost certainly haemophilia A because the von Willebrand factor antigen (VWF:Ag) was recorded at 65% and the bleeding time test was normal.

3.2 Treatment with cryoprecipitate as required continued, even until 1987/8, but there is a record of factor VIII replacement for a knee haematoma on 30/06/75 and a record, dated 25/05/78, that he received 800 units of "EDIN" to cover dental extraction on that day. It is very likely that this was a large pool factor VIII concentrate prepared in Edinburgh. Even if he had not already been infected with hepatitis C by then the infection would almost certainly have been transmitted on this date.

3.3 The first abnormal liver function test that I have been able to find is dated 14/12/79 when the transaminase G.O.T. is recorded as "97" in the notes by hand. The majority of the transaminase results after this are abnormal and this is typical of hepatitis C infection. It was soon recognised that Mr. Black had "non A non B" hepatitis for which no treatment was then available. There was also evidence of immunity to hepatitis B but a sample was tested for anti HIV and found to be negative when the test became available.

3.4 By 1987 the liver and spleen were palpable and there was evidence of chronic liver disease. Portal hypertension and oesophageal varices were treated in a conventional way.

3.5 In 1994 a referral was made for consideration of interferon therapy but this was not started, partly because Mr. Black was reluctant to undergo treatment. By 1996 liver function had deteriorated to the point at which transplantation was considered. A liver transplant was performed on 21st April 1996 and I understand that hepatocellular carcinoma was found in the diseased liver. Liver function tests never returned to normal and it soon became apparent that inflammation of the transplanted liver by recurrent hepatitis C infection had taken place, followed by evidence of fibrosis.

3.6 In 2002 an attempt was made to treat the infection with pegylated interferon and ribavirin but Mr. Black's condition continued to deteriorate and hepatocellular carcinoma developed.

3.7 Mr. Black died on 31st October 2003.

4. OPINION

4.1 Mr. Black had moderate haemophilia A which was correctly treated with blood products, including cryoprecipitate in the first instance. He would not have been suitable for treatment with desmopressin (DDAVP) and this approach was only described in 1977. On a balance of probabilities he was first treated with a large pool factor VIII concentrate by 25th May 1978 to cover dental extraction and it is now known that virtually all those treated with large pool factor VIII concentrates before 1985/6 became infected with hepatitis C. Even if all his treatment had been with cryoprecipitate and red blood cell transfusion until as late as the mid 1980s it is still likely that he would have contracted hepatitis C.

4.2 It is perhaps surprising that Mr Black was still being given cryoprecipitate in 1987/8 but, since his liver function tests were already abnormal and the anti HIV test remained negative, the use of cryoprecipitate after the introduction of virally inactivated large pool concentrates in the mid 1980s is clinically irrelevant.

4.3 The course of HCV infection was relatively rapid in his case but it took nearly 20 years to progress from infection to transplantation. Treatment with interferon was largely ineffective in the early 1990s and in my opinion nothing was lost by any delay in referral to a hepatologist until 1994, especially since there was good evidence of chronic liver disease by that date and it seems that Mr. Black was not keen on treatment. (Strictly speaking this is a matter for a hepatologist to comment on but I can speak from personal experience of managing HCV infection with hepatologist support at this time.)

4.4 The procedure of liver transplantation would have "cured" Mr. Black's haemophilia because of factor VIII production by the transplanted liver but reinfection of the liver was inevitable and it is not surprising to me that the modern antiviral treatment with pegylated interferon and ribavirin was ineffective at this stage.

4.5 The final development of hepatocellular carcinoma was a direct result of HCV infection, whether it occurred in the original or the transplanted liver.

4.6 Mr. Black's death on 31st March 2003 was due to hepatitis C infection in the transplanted liver together with hepatocellular carcinoma, the infection having been originally transmitted by blood products.

4.7 I have been unable to find that Mr. Black's treatment was unreasonable or inappropriate at any stage of his illness and care, save that it was unusual to have continued to use cryoprecipitate as late as 1987/8. In my opinion this decision had no effect whatever on the outcome of the case.

5. CONCLUSION

5.1 The Reverend David Black had moderate haemophilia A which was appropriately treated with cryoprecipitate and large pool factor VIII concentrates prior to the development of viral inactivation for factor VIII concentrates in the mid 1980s.

5.2 Infection with hepatitis C in Mr. Black's case was not avoidable and, before the introduction of pegylated interferon and ribavirin, specific antiviral therapy for hepatitis C was generally ineffective.

5.3 The course of hepatitis C infection is very variable but the need for liver transplantation nearly 20 years after infection is not exceptional in my experience.

5.4 It is not surprising that the transplanted liver became infected and fibrotic or that a hepatocellular carcinoma developed.

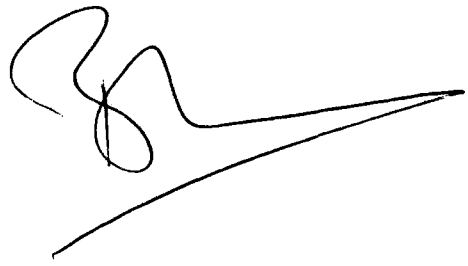
5.5 I have found no evidence of unreasonable or inappropriate treatment in this case but I am not an expert in the field of hepatology and HCV infection, except for my personal experience of its diagnosis and management in the field of haemophilia care at the relevant times.

6. STATEMENT OF COMPLIANCE

6.1 I understand my duty as an expert witness is to the Inquiry. I have complied with that duty. This report includes all matters relevant to the issues on which my expert evidence is given. I have given details in this report of any matters which might affect the validity of this report. I have addressed this report to the Inquiry.

8. STATEMENT OF TRUTH

8.1 I confirm that insofar as the facts stated in my report are within my knowledge, I have made clear which they are and believe them to be true, and that the opinions I have expressed represent my true and complete professional opinion.



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