

**THE 1986-1990 HEPATITIS C CLASS ACTION SETTLEMENT**

IN THE MATTER OF AN APPEAL FROM THE DECISION OF THE ADMINISTRATOR  
DATED AUGUST 15, 2001, CLAIM # 1702

DATE OF HEARINGS:            November 6, 2005, Kingston  
   April 19, 2006, Ottawa  
   July 12, 2006, Kingston

REFEREE:                            Michael Mitchell

IN ATTENDANCE:                Claimant and family  
   John Callaghan and Carol Miller, for the Administration

## DECISION

1. This is an Ontario-based claimant, claim #1702 and this matter is before me in my capacity as Referee.
2. The HCV Infected Person (Mr. W.) in this case passed away on December 7, 1995 at the age of 81. Mr. W's passing came after surgery on August 24, 1995. After his passing, the family subsequently learned that, in a previous surgery, Mr. W. had received blood from a donor who subsequently tested positive for Hepatitis C. Naturally, the family was shocked by this news, and has been highly suspicious that the disease contributed to the death of Mr. W.
3. The issue in this case is whether Hepatitis C materially contributed to the death of Mr. W. While the Administrator in this case did not concede that Mr. W. actually had the disease at the time of his death, the evidence presented to me during the case was entirely centered on the issue of whether the disease materially contributed to the passing of Mr. W, in effect assuming that Mr. W had the disease. Accordingly, for the purposes of this decision, I am prepared to assume, without finding, that at the time of his death, Mr. W. was in fact infected with HCV, though there was no evidence that Mr. W. did or did not have the disease at the time of his death.
4. The hearing in this matter took place on three separate days in Kingston and Ottawa. Two physicians who were involved in Mr. W.'s surgery and care prior to his death testified. In addition, an independent expert physician, Dr. Depew, reviewed the medical file and gave his written opinion was also tendered in evidence as follows:

I have reviewed, page by page, the medical information available on [Mr. W.]. I understand from the accompanying information that it is necessary to determine whether hepatitis C infection contributed in a material way to [Mr. W.'s] death in 1995. I further understand from the enclosed information that you were notified in writing that [Mr. W.'s] received a transfusion of platelets in 1989 and that that transfusion was obtained from a patient with known chronic hepatitis C infection.

On examining the copies of all the medical records I find no evidence that [Mr. W.'s] suffered from clinically overt chronic hepatitis C. The admitting histories and physicals done during his hospital stays in 1992 and 1995 do not identify any clinical symptoms of underlying liver disease or portal hypertension and the physical exams do not identify enlargement of the liver or the spleen or the presence of any other peripheral sign of either chronic parenchymal liver disease or portal hypertension. A limited set of liver status tests obtained in 1992 are well within the normal range. The simple liver status tests obtained at his admission in 1995 were also within the normal range providing no biochemical clue that he had underlying substantial parenchymal liver damage.

[Mr. W.'s] demise in 1995 was driven by post operative complications including septicemia and accompanying acute respiratory distress syndrome. This culminated in multiple system organ failure. There were some clear cut changes in liver tests towards

the end of that admission but these changes are clearly related to the sepsis and multiple organ failure and could not be blamed on pre-existing chronic hepatitis C.

Regrettably, the record does not contain any information that would allow me to state with any measure of confidence that chronic hepatitis C contributed materially to [Mr. W.'s] death. The difficult thing with chronic hepatitis C however is that it may be present and progress silently without any overt clue. Indeed, the disease may progress to cirrhosis without demonstrating any obvious clinical signs. Cirrhosis can also be present even when the usual types of liver status tests are within the normal range. Accordingly, I could not conclude that [Mr. W.] did not have any liver damage from chronic hepatitis C. The only way to determine this would have been pathologic examination of the liver either by liver biopsy or at post mortem. I am not aware that either of these investigations were undertaken in [Mr. W.'s] case exam.

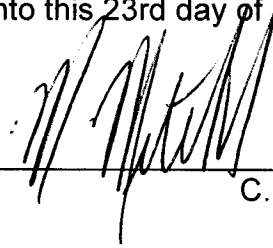
If [Mr. W.'s] acquired hepatitis C at the time of his transfusions in 1989 there would have been adequate time for the virus to have caused damage to the point of cirrhosis in the ensuing sixteen years up to his death. Unfortunately, there is absolutely nothing in the record which would enable anyone to state with any confidence that he suffered from such a process. Indeed, the bulk of the evidence suggests that progressive significant liver disease due to chronic hepatitis C infection was not present.

5. Both Dr. Rudan, the surgeon, and Dr. Prihar, the primary Resident Physician assigned to the case in the teaching hospital, gave evidence and were cross-examined. They both agreed with the opinion of Dr. Depew. They testified that essentially, during the surgery, the patient aspirated large amounts of bilious fluid and as a result, had a septicemia, with accompanying pneumonia, which resulted in "acute respiratory distress syndrome". Despite three months of intensive efforts to assist the patient, there was "multiple system organ failure" resulting in his passing. Neither of the physicians who were involved in his care and treatment are of the opinion that the events that occurred would have been any different whether or not Mr. W. had Hepatitis C. According to them, it was not a factor at all in his condition, assuming Mr. W. had the disease.
6. Based on all the evidence, there is no basis to conclude in this case that Hepatitis C contributed to the death of Mr. W. Having said that, it is easy to understand the dismay and consternation of the family on learning after his passing that he may well have contracted the disease. It is easy also to understand how the family suspects he had the disease, and that it could have contributed in a significant way to his death.
7. I have assumed for the purposes of this argument that Mr. W. did have the disease. Based on the facts in the medical file and the opinions of the physicians who treated Mr. W., and the independent assessment of Dr. Depew, there is simply no evidence upon which it could be determined that Hepatitis C contributed to the death of Mr. W., much less that it contributed in a material way. The fact is that Mr. W. needed an urgent hip replacement at the age of 81 and, as a result of post-operative complications, his condition deteriorated over

several months. There is no evidence that these complications were contributed to or worsened by Hepatitis C disease.

8. While I admire the perseverance of the family members to get to the bottom of this matter, and understand their suspicion and shock at learning that someone who had given blood to their loved one years before had tested positive for Hepatitis C, this claim must nonetheless be dismissed.

DATED at Toronto this 23rd day of November, 2006



---

C. Michael Mitchell  
Referee