United States Department of Labor
Employees' Compensation Appeals Board

DONNA R. EDWARDS, claiming as widow of THOMAS G. EDWARDS, Appellant	)	
and	)	**Docket No. 05-1265**
	)	**Issued: June 1, 2006**
DEPARTMENT OF ENERGY, BONNEVILLE POWER ADMINISTRATION, Hot Springs, MT, Employer	)	

Appearances:
Jeffrey P. Zeelander, Esq., for the appellant
Office of Solicitor, for the Director

Case Submitted on the Record

DECISION AND ORDER

Before:
ALEC J. KOROMILAS, Chief Judge
DAVID S. GERSON, Judge
MICHAEL E. GROOM, Alternate Judge

JURISDICTION

On September 15, 2005 appellant filed a timely appeal from the Office of Workers' Compensation Programs' merit decision dated June 2, 2004, denying her claim for compensation. Pursuant to 20 C.F.R. §§ 501.2(c) and 501.3, the Board has jurisdiction over the merits of this case.

ISSUE

The issue is whether exposure to active tuberculosis (TB) and subsequent isoniazid (INH) therapy hastened or contributed to the employee's death.

FACTUAL HISTORY

On October 20, 2003 appellant filed a claim alleging that her husband's liver failure and subsequent death was caused by his INH therapy, which he received for employment-related TB.

Appellant submitted physician's notes dated August 27, 2003 bearing an illegible signature, reflecting that the employee appeared fatigued and jaundiced. Notes from Glacier Medical Associates dated June 30, 2003 bearing an illegible signature indicated that he had been exposed to the active phase of is TB and provided a diagnosis of tuberculum converter. Appellant submitted medical notes and reports, including: laboratory reports for the period from August 27 through September 1, 2003; physician's notes dated July 16, 2002 bearing an illegible signature, reflecting the employee's complaints of right upper extremity pain; an unsigned prehospital patient form from Whitefish Fire and Ambulance dated August 29, 2003 containing illegible notes; an August 28, 2003 report of an abdominal ultrasound, reflecting enlarged liver and spleen; and reports of chest x-rays dated June 30 and August 29, 2003.

August 27, 2003 hospital notes from Dr. Jay S. Erickson, Board-certified in the area of family medicine, indicated that the employee normally drank six beers per day. Dr. Erickson stated that he suffered from acute hepatitis, likely secondary to alcohol use; hyponatremia; hypokalemia; and renal failure.

In a report dated August 28, 2003, Dr. Christopher Holdhusen, Board-certified in the area of family medicine, provided diagnoses of fulminant hepatitis, presumed due to a combination of INH and alcohol; OTOH abuse with potential for withdrawal; hyponatremia and hypokalemia; hypertension; and history of gastric bleed and now with heme-positive stool.

In an unsigned report dated August 29, 2003, Dr. Howard Tice noted that the employee was profoundly icteric, restrained and not arousable. He further noted that he had been taking INH for several months due to his exposure to TB and subsequent positive TB test. Dr. Tice provided impressions of jaundice/hepatic encephalopathy; hypoprothrombinemia and coagulopathy; hypokalemia; and hyponatremia. He opined that the employee may well have had underlying liver disease due to alcohol use, which was aggravated by drugs such as acetaminophen and INH.

In a memorandum dated August 28, 2003, the employing establishment expressed its belief that the employee contracted TB through contact with an infected employee during the course of his employment in the workplace.

A death certificate dated September 8, 2003 reflected that the cause of death was multiorgan failure/ hepatic failure.

On February 11, 2004 the Office notified appellant that the employing establishment had confirmed the "factual exposure" of her husband to TB and that the district medical adviser was reviewing the case to determine whether or not the INH therapy hastened or contributed to her husband's death.

In a report dated February 14, 2004, the district medical adviser, Dr. Morley Slutsky, a Board-certified in the area of occupational medicine, stated that based on the information presented, he was unable to determine whether the INH therapy hastened or contributed significantly to the employee's death. He indicated that the pathologist's report would provide the best information regarding the cause of appellant's liver failure. He also stated that Dr. Tice

should be contacted for information regarding "other possible agents" which may have exacerbated the employee's chronic liver condition.

By letter dated April 16, 2004, the Office advised appellant that additional information was required in order to establish her claim, including the evidence outlined in the February 14, 2004 report of the district medical adviser. Appellant was given 30 days to submit any additional information.

In a pathology report dated September 26, 2003, Dr. Corinne Fligner, a Board-certified anatomic and clinical pathologist, stated that it was not possible to histologically confirm or deny INH toxicity. Noting that the major question at autopsy was the etiology of the employee's liver failure, she indicated that the cause of death was considered to be multiorgan failure due to underlying hepatic failure, associated with ischemic necrosis of the small bowel. She related that the etiology of the acute hepatic decompression was likely multifactoral, with possible etiologies including undiagnosed preexisting hepatic cirrhosis (likely due to chronic ethanolism); continued chronic ethanolism; and INH therapy. Dr. Fligner noted that INH therapy was known to cause serious hepatotoxicity in 1 to 2 percent of people taking it and had a case fatality rate of 10 percent. She further noted that significant risk factors for hepatotoxicity include age above 50 years and ethanol consumption. Dr. Fligner indicated that, alternatively, it was also possible that acute hepatic decompensation occurred in the setting of chronic liver dysfunction in this patient with undiagnosed cirrhosis, related to continued drinking and otherwise unknown metabolic disturbances. Dr. Fligner noted that the employee had continued to drink six to eight beers per day while on INH therapy.

By decision dated June 2, 2004, the Office denied appellant's claim, finding that the medical evidence did not demonstrate that exposure to active TB and subsequent INH therapy hastened or contributed to her husband's death.

LEGAL PRECEDENT

An award of compensation in a survivors claim may not be based on surmise, conjecture or speculation or an appellant's belief that the employee's death was caused, precipitated or aggravated by the employment.[1] Appellant has the burden of establishing by the weight of the reliable, probative and substantial medical evidence that the employee's death was causally related to an employment injury or to factors of his employment. As part of this burden, she must submit a rationalized medical opinion, based upon a complete and accurate factual and medical background, showing a causal relationship between the employee's death and an employment injury or factors of his federal employment. Appellant's unsupported belief is insufficient to establish causal relationship.[2] Causal relationship is a medical issue and can be established only by medical evidence.[3]

[1] *Sharon Yonak* (*Nicholas Yonak*), 49 ECAB 250 (1997).

[2] *See Jacqueline Brasch* (*Ronald Brasch*), 52 ECAB 252 (2001); *Leonora A. Bucco* (*Guido Bucco*), 36 ECAB 588 (1985); *see also Shirley Miles* (*William H. Miles*), Docket No. 04-670 (issued June 28, 2004).

[3] *Mary J. Briggs*, 37 ECAB 578 (1986); *Umberto Guzman*, 25 ECAB 362 (1974).

The medical evidence required to establish causal relationship is rationalized medical evidence. Rationalized medical evidence is medical evidence which includes a physician's rationalized medical opinion on the issue of whether there is a causal relationship between an employee's diagnosed conditions and the implicated employment factors. The opinion of the physician must be based on a complete factual and medical background of the employee, must be one of reasonable medical certainty and must be supported by medical rationale explaining the nature of the relationship between the employee's death and the accepted conditions or employment factors identified by the employee.[4]

ANALYSIS

The Office accepted as factual that the employee contracted TB during the course of his federal employment. The issue for determination is whether his INH therapy hastened or contributed to his death. The Board finds that the medical evidence of record is insufficient to establish appellant's claim.

Appellant submitted numerous treatment notes bearing illegible signatures. These forms, lacking proper identification, cannot be considered as probative evidence.[5] An August 28, 2003 report of an abdominal ultrasound reflected enlarged liver and spleen. However, the report is unsigned and offers no opinion on the cause of the condition. The report is of no probative value on the issue of causal relationship.[6]

In hospital notes dated August 27, 2003, Dr. Erickson indicated that the employee experienced acute hepatitis, likely secondary to alcohol use; hyponatremia; hypokalemia; and renal failure. Dr. Erickson's opinion does not support appellant's contention that her husband's liver condition was causally related to his INH treatment.

In an August 28, 2003 report, Dr. Holdhusen noted that appellant had habitually consumed six or more alcoholic drinks per day for a long period of time. He diagnosed fulminant hepatitis, presumably due to a combination of INH treatment and alcohol; OTOH abuse, with potential for withdrawal; hyponatremia and hypokalemia; hypertension and history of gastric bleed. Dr. Holdhusen's opinion as to a causal relationship between appellant's hepatitis, INH treatment and alcohol abuse is speculative and equivocal. He did not provide adequate explanation as to how a combination of INH and alcohol could have caused or contributed to the employee's hepatitis. Therefore, his opinion lacks probative value.

In his August 29, 2003 report, Dr. Tice noted that the employee had been taking INH for several months due to his exposure to TB and subsequent positive TB test. Dr. Tice opined that he may well have had underlying liver disease due to alcohol abuse, which was aggravated by drugs such as acetaminophen and INH. Dr. Tice's opinion is also equivocal and based upon an indefinite diagnosis. He failed to provide a rationalized explanation as to how INH treatment

[4] *Donna L. Mims*, 53 ECAB 730 (2002).

[5] *Vickey C. Randall*, 51 ECAB 357 (2000); *Merton J. Sills*, 39 ECAB 572 (1988).

[6] *Id. See also Michael E. Smith*, 50 ECAB 313 (1999).

caused or contributed to the employee's liver condition. Therefore, his opinion is of diminished probative value.

Based on the information presented to him, the district medical adviser was unable to determine whether the INH therapy hastened or contributed to the employee's hepatic failure. He recommended obtaining a copy of the pathologist's report. Accordingly, his report offers no support for appellant's claim.

Dr. Fligner's pathology report failed to establish that the employee's INH therapy hastened or contributed to his death. The report did not provide an unequivocal opinion regarding the cause of his liver failure, the stated cause of death. Rather, Dr. Fligner provided detailed alternative theories, stating that it was not possible to histologically confirm or deny INH toxicity. She indicated that, on the one hand, the liver failure might be due to a combination of undiagnosed cirrhosis (likely due to ethanolism) and INH therapy. On the other hand, she stated that it was also possible that acute hepatic decomposition occurred "in the setting of chronic liver dysfunction in this patient with undiagnosed cirrhosis, related to continued drinking and otherwise unknown metabolic disturbances." Although Dr. Fligner noted that the employee's alcohol abuse and age put him at risk for hepatotoxicity, in light of his INH therapy, she was unable to conclude or state within a reasonable degree of medical certainty that the INH therapy hastened or contributed to his death. For these reasons, the Board finds that Dr. Fligner's report is of diminished probative value.

There is no medical evidence of record which provides a well-rationalized opinion that the accepted job-related TB or subsequent INH therapy hastened or contributed to the employee's death. Therefore, the Board finds that appellant has failed to establish her claim.

CONCLUSION

The Board finds that appellant has not met her burden of proof to establish that the employee's death was causally related to factors of employment. She failed to establish that exposure to active TB and subsequent INH therapy hastened or contributed to her husband's death.

ORDER

IT IS HEREBY ORDERED THAT the decision of the Office of Workers' Compensation Programs dated June 2, 2004 is affirmed.

Issued: June 1, 2006
Washington, DC

Alec J. Koromilas, Chief Judge
Employees' Compensation Appeals Board

David S. Gerson, Judge
Employees' Compensation Appeals Board

Michael E. Groom, Alternate Judge
Employees' Compensation Appeals Board