U. S. DEPARTMENT OF LABOR

Employees' Compensation Appeals Board

In the Matter of DENNIS L. SAATHOFF and DEPARTMENT OF THE NAVY, PUGET SOUND NAVAL SHIPYARD, RIGGING SHOP, Bremerton, WA

Docket No. 02-1336; Submitted on the Record;
Issued May 2, 2003

DECISION and ORDER

Before COLLEEN DUFFY KIKO, DAVID S. GERSON, MICHAEL E. GROOM

The issue is whether appellant has established that he sustained renal tubular dysfunction and cadmium toxicity related to toxic chemical exposures in his federal employment.

On February 26, 2001, appellant, then a 50-year-old rigger and rigging helper, filed a notice of occupational disease alleging that he sustained renal tubular dysfunction affecting both kidneys due to exposure to toxic levels of cadmium and other substances while in the performance of duty. He explained that, from July 1999 to December 2000, he worked in areas where vessels were being dismantled, with frequent exposure to "pieces that are being cut with a torch as well as welding or carbon arching. Smoke often permeate[d] the air … even outside of barriers," particularly in the dry dock during burning operations.[1]

Appellant submitted medical evidence in support of his claim demonstrating occupational exposure to cadmium and lead and abnormally high levels of beta-2 microglobulin on July 24 and August 16, 2000 urinalyses. These results were interpreted by his physicians as indicative of renal tubular dysfunction caused by cadmium toxicity.

By decision dated May 10, 2001, the Office denied appellant's claim on the grounds that causal relationship was not established. The Office found that appellant submitted insufficient rationalized medical evidence to establish that his renal tubular dysfunction was related to any factor of his federal employment.

Appellant disagreed with this decision and, in a June 6, 2001 letter, requested an oral hearing before a representative of the Office's Branch of Hearings and Review. The hearing was held on January 10, 2002. He submitted additional evidence prior to the hearing.

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[1] In a March 28, 2001 letter, the Office of Workers' Compensation Programs advised appellant of the type of medical and factual evidence needed to establish his claim, including a complete and detailed history of cadmium exposure and rationalized statement from his treating physician explaining any relationship between these exposures and the diagnosed renal tubular dysfunction.

At the hearing appellant, through his authorized representative, asserted that the employing establishment was aware of widespread cadmium and chemical contamination but had not taken any steps to abate these hazards. Appellant alleged that, while workers were warned of lead contamination hazards, they were not notified of cadmium contamination. Appellant alleged that Dr. Dmitri Vasin, an attending Board-certified nephrologist, was pressured or influenced by Dr. Garret W. Duckworth, Jr., an employing establishment physician, to change his diagnosis from renal tubular dysfunction to a low back strain.

Appellant submitted several industrial hygiene surveys and procedure manual excerpts addressing the presence of cadmium, lead, arsenic, heavy metals and hazardous chemicals at the employing establishment, in the "cut-down" areas in which appellant worked disassembling naval vessels. A series of employing establishment industrial hygiene memoranda and safety procedures, dated from 1994 to 1996, noted the presence of Tri-n-butyl tin, organotin, anti-fouling compounds and lead vapors from cutting, grinding and sanding operations. August 22, 1996 air sampling results for dry dock 4 showed airborne cadmium and tin at zero and chromium, copper, dust, iron, lead, nickel and zinc particulates at levels from one thousandth to one tenth of permissible exposure limits.[2] A November 15, 1996 yellow alert was issued for possible exposure to arsenic and cadmium above action levels or permissible exposure limits in the burning and carbon arcing operations. Actual violations were found on May 1 and 2, 2001 inspections of the toolroom areas, classed as "serious": employees "were allowed to consume food and beverages in areas contaminated with … lead, arsenic and cadmium" and were "exposed to tools and personal protective equipment contaminated with" lead, arsenic and cadmium.

Health monitoring forms, dated January 6 to June 6, 2000, noted appellant's exposures to lead,[3] methylene chloride, arsenic and cadmium. In an April 3, 2001 letter, the employing establishment acknowledged that appellant had minimal exposure to cadmium and wore appropriate personal protection equipment. October 1 and 2, 2001 notification forms noted that August 27 and 29, 2001 monitoring in appellant's work area revealed 0.000017 and 0.00009 milligrams arsenic per cubic meter of air over an eight-hour period, with a permissible exposure limit of 0.010 milligrams per cubic meter of air; 0.00003 and 0.0002 milligrams cadmium with a permissible exposure limit of 0.005 milligrams; and 0.00017 and 0.00009 milligrams of lead with a permissible exposure limit of 0.050 milligrams per cubic meter.[4]

Appellant was temporarily removed from cadmium exposure effective December 18, 2000. On August 20, 2001 the employing establishment permanently removed

[2] Employing establishment reports on airborne toxins from marine coatings, dated from January to December 2000, enumerated 13 incidents of uncapped paint cans, 2 uncapped cans of paint thinner, 1 open waste drum, 3 paint spills and one incident of kitty litter, used to dry up excess paint, found on a deck. No specific toxic exposures were enumerated as related to these incidents.

[3] February 9 and August 1, 2000, serology performed for the employing establishment's lead monitoring program showed blood lead levels of 3 micrograms per deciliter, with normal levels being less than 20 micrograms and zinc protoporphyrin levels of 19 and 25 micrograms respectively per deciliter, with normal levels being less than 50.

[4] Captain G.R. Bryant, the commanding officer of the employing establishment, contended that appellant had not been exposed to cadmium, lead, arsenic and respiratory hazards beyond OSHA permissible exposure levels.

appellant from "work involving lead, cadmium and mercury … fluoride, chlorinated hydrocarbons, glycols, aminated compounds and barium."

In an October 6, 2000 report, Dr. Daniel E. Diamond, an attending urologist, diagnosed "mildly abnormal renal function testing, … perhaps related to cadmium exposure."

In a series of reports dated November 13, 2000 to April 9, 2001, Dr. Vasin noted that appellant was exposed to cadmium dust at work. Dr. Vasin noted that cadmium monitoring tests were significant for "elevated beta-2 microglobulin level of 663 [micrograms per] gram of creatinine with a reference range of 160 or less," while blood and urine cadmium levels were within normal range.[5] He obtained November 20, 2000 blood and renal function tests, which were "normal with creatinine 1.1," but showed a high risk for kidney stones.[6] Dr. Vasin diagnosed multiple abnormalities consistent with tubular dysfunction and damage, demonstrated by the presence of increased beta-2 microglobulin excretion as a sensitive, nonspecific marker, hypercalciuria, hyperuricosuria and hyperphosphaturia, in the "presence of normocalcemia, normal phosphatemia and normal level of uric acid…." Dr. Vasin stated that tubular dysfunction could be seen in cadmium and other heavy metal toxicities, "even though cadmium level in [appellant's] blood and urine did not reach the threshold level." Dr. Vasin noted that, pathenogenically, cadmium caused "early tubular dysfunction and, later, interstitial nephritis and renal insufficiency." Regarding causal relationship, Dr. Vasin stated that although it was far from proven that renal dysfunction was caused by cadmium in appellant's case (because of normal level of cadmium in blood and urine) this possibility could not be ruled out. In an April 9, 2001 report, Dr. Vasin opined that the onset of upper back pain in December 2000 was most likely due to osteoarthritis or muscle spasm, as opposed to renal complaints or cadmium exposure. Dr. Vasin permanently restricted appellant from exposure to cadmium and prescribed medication and dietary changes.

In a December 2000 report, Dr. Duckworth, an employing establishment physician, noted that December 18, 2000 laboratory studies showed blood levels of cadmium at less than 0.5 micrograms per liter, below the reference range of 1.7 micrograms and also below the 30 microgram threshold for showing early signs of toxicity. Random urine studies for cadmium showed concentrations of 0.5 and 1.10 micrograms, below the applicable reference ranges of 1.2 and 3.0 micrograms respectively. Appellant's beta-2 microglobulin was 64 micrograms, with a reference range of 160 micrograms or less per gram of creatinine. Dr. Duckworth noted that appellant had possible excessive cadmium exposure but suspected that the beta-2 microglobulinuria was more likely due to underlying renal disease associated with uric acid

[5] July 24, 2000 tests performed for the employing establishment showed an elevated beta-2 microglobulin level of 310 micrograms per gram of creatinine, above the reference range of 0 to 160. Repeat urinalysis on August 16, 2000 showed 663 micrograms of beta-2 microglobulin per gram of creatinine. Random urine creatinine was .98, within the normal range of .27 and 3.00 grams per liter. November 20, 2000 laboratory tests showed hypercalciuria, hyperoxaluria, hyperuricosuria and high urinary sodium. Possible problems suggested by these results were kidney stones and a "high animal protein intake."

[6] Dr. Vasin ordered December 22, 2000 abdominal x-rays, which were negative for stones in the kidneys or ureters bilaterally. A December 22, 2000 renal sonogram showed "[p]robable mild increased renal echogenicity," indicating a "question of medical renal disease," and a "4.2 centimeter simple-appearing cyst projecting from the lower pole of the right kidney."

and oxalate metabolic factors. He also noted that beta-2 microglobulin could also be elevated due to poor renal function, lymphoma, leukemia, myeloma, nonlymphoid malignancies, sarcoidosis, mononucleosis, viral infections, chronic inflammatory disease including liver disease and autoimmune disorders.

In December 2000 and January 2001, appellant reported to the employing establishment's health clinic complaining of back pain. Based on these symptoms, Dr. Rolando P. Dulay, a clinic physician, restricted appellant from driving a forklift from December 18, 2000 to January 2, 2001. In a January 2, 2001 note, Dr. Dulay diagnosed low back pain secondary to renal tubular dysfunction. In a January 3, 2001 note, Dr. Edgar T. Briones, another clinic physician, diagnosed possible nephritis. Dr. Duckworth renewed the restriction against driving a forklift over irregular surfaces from February 1 to 20, 2001.

Appellant submitted a series of reports dated from July 5 to October 12, 2001 from Dr. David K. Bonuato, an attending physician Board-certified in occupational medicine. Dr. Bonuato noted that appellant exhibited elevated beta-2 microglobulin levels in July 2000 after working for 18 months in ship breakdown, with exposure to lead, epoxies, chromium, arsenic, cadmium and cadmium coated steel, copper, nickel, brass, copper, beryllium, zinc stellate, zinc chromate, polychlorobenzenes, cavity foams, creosote, organotin and bauxite. He reviewed laboratory reports, performed a physical examination and diagnosed elevated beta-2 microglobulin and bilateral flank pain. Dr. Bonuato commented that as appellant did not have elevated blood cadmium levels, the elevated beta-2 microglobulin levels "may or not be occupationally related." He explained that, while the specificity of the beta-2 microglobulin for cadmium exposure was relatively good, upper respiratory infection and other etiologies should be considered. He obtained a 24-hour urine test showing a cadmium level of 1.6 and elevated sodium,[7] opining that these studies showed no objective evidence of kidney damage and that elevated sodium and uric acid levels were explained by appellant's high sodium and protein intake. Dr. Bonuato diagnosed "rule out cadmium toxicity." In an October 12, 2001 letter, he stated that appellant did not have overt pathology of his kidney related to cadmium exposure. Dr. Bonuato characterized Dr. Vasin's diagnosis of renal tubular dysfunction as "debatable," and that appellant's bilateral flank pain was not related to his kidneys. He concluded that appellant should not be exposed to any potential nephrotoxic compounds, including lead and cadmium.

In a December 28, 2001 letter, Dr. Leah A. Hasley, a physician specializing in occupational medicine and an associate of Dr. Bonuato, stated that even after appellant had no occupational cadmium exposures for 10 months, he "had beta-2 microglobulinuria at the upper limit of normal," and an elevated urine pH indicating continuing renal tubular dysfunction.[8] Dr. Hasley noted that, as of October 26, 2001, appellant's beta-2 microglobulin was at zero. She opined that appellant had some type of reversible renal tubular dysfunction, although she could

[7] A July 8, 2001 urinalysis showed cadmium concentration of 1.6 micrograms, within the normal reference range of less than 3 micrograms and elevated sodium at 309 mEq per liter, above the normal reference range of 40 to 220 mEq. Serum bilirubrin was slightly elevated at 1.1 milligrams per deciliter, above the reference range of 0.1 to 1.0 milligrams.

[8] November 8, 2001 urinalysis showed beta-2 microglobulin at 227, within the normal range of 300 micrograms per liter, cadmium at less than 0.5 micrograms per liter and creatinine within normal limits.

not say for certain whether cadmium exposure was the sole cause. She recommended that appellant refrain from working in an environment that involves exposure to heavy metals.

By decision dated and finalized March 18, 2002, the Office hearing representative affirmed the May 10, 2001 decision. The hearing representative found that appellant submitted insufficient evidence to establish the diagnosis of cadmium toxicity or other condition related to occupational chemical exposures.

The Board finds that appellant has failed to meet his burden of proof in establishing that he sustained renal tubular dysfunction, cadmium toxicity, or other illness related to metal or chemical exposure in the performance of duty.

To establish that an injury was sustained in the performance of duty in an occupational disease claim, a claimant must submit the following: (1) medical evidence establishing the presence or existence of the disease or condition for which compensation is claimed; (2) a factual statement identifying employment factors alleged to have caused or contributed to the presence or occurrence of the disease or condition; and (3) medical evidence establishing that the diagnosed condition is causally related to the employment factors identified by the claimant. The medical opinion must be one of reasonable medical certainty and must be supported by medical rationale explaining the nature of the relationship between the diagnosed condition and the specific employment factors identified by the claimant.[9]

Appellant has not submitted sufficient medical evidence to establish he sustained renal tubular dysfunction or to substantiate that any occupational exposure to cadmium, lead, arsenic or other nephrotoxins caused renal tubular dysfunction.

In a December 13, 2000 report, Dr. Vasin, an attending Board-certified nephrologist, diagnosed renal tubular dysfunction based on the nonspecific marker of increased beta-2 microglobulinuria, hypercalciuria, hyperuricosuria and hyperphosphaturia, in the "presence of normocalcemia, normal phosphatemia and normal level of uric acid." Dr. Keith A. Schulze, an attending internist, diagnosed renal tubular dysfunction in a January 5, 2001 report. In a December 28, 2001 letter, Dr. Hasley, an attending physician specializing in occupational medicine, stated that appellant's chronically elevated beta-2 microglobulin 10 months after all occupational exposures were to have ceased indicated the presence of "some type of reversible renal tubular dysfunction."

However, two physicians opined that elevated beta-2 microglobulin was not diagnostic of renal tubular dysfunction. Dr. Duckworth, an employing establishment physician, opined that the elevated beta-2 microglobulin could be indicative of malignancies, auto-immune disorders, viral infections, sarcoidosis, or liver disease. Dr. Duckworth did not diagnose renal tubular dysfunction. Similarly, Dr. Bonuato, an attending physician Board-certified in occupational medicine, stated that appellant's elevated beta-2 microglobulin level was not suggestive of renal tubular dysfunction as his blood and urine cadmium levels were normal. He noted that beta-2 microglobulinuria could also be caused by common illnesses such as upper respiratory infection. Dr. Bonuato characterized Dr. Vasin's diagnosis of renal tubular dysfunction as "debatable."

[9] *Charles E. Burke*, 47 ECAB 185 (1995).

Appellant has submitted evidence to establish that he was exposed to cadmium, lead, arsenic, methylene chloride, organotin, anti-fouling compounds, chromium, copper, iron, nickel and zinc in the performance of duty. The industrial hygiene surveys, official correspondence and safety manual excerpts document the presence of these compounds at concentrations below permissible exposure limits. Although the November 13, 2001 OSHA safety violation notices indicate that appellant may have been exposed to high levels of lead, arsenic and cadmium in the tool and break areas, he has not submitted evidence on the amount of time he was present in those areas, for what duration, or the quantities and concentrations of any substances found. The November 13, 2001 OSHA notices do not establish that appellant was exposed to cadmium, lead or arsenic at levels above the permissible exposure limits.

Appellant submitted several medical reports addressing a possible causal relationship between occupational cadmium exposure and renal tubular dysfunction. However, these reports are speculative on causal relationship.

Dr. Vasin explained that cadmium caused a predictable pattern of renal pathology, beginning with renal tubular dysfunction and progressing to interstitial nephritis and renal insufficiency. He opined that appellant's renal tubular dysfunction could be caused by cadmium or other heavy metal toxicity, even though his serum and urine cadmium levels were below the level generally considered to be toxic. He commented that although it was "far from proven that the dysfunction is caused by cadmium … this possibility cannot be ruled out." Dr. Hasley stated that she could not state "for certain whether cadmium exposure was the sole cause" of appellant's renal tubular dysfunction.

These opinions are equivocal on the issue of causal relationship and insufficient to meet appellant's burden of proof. Dr. Vasin stated that the relationship between appellant's cadmium exposure and renal tubular dysfunction was "far from proven." Dr. Hasley noted that she could not be certain whether "cadmium exposure was the sole cause" of appellant's renal tubular dysfunction. The opinions of Drs. Vasin and Hasley are too speculative and equivocal on the issue of causal relationship to meet appellant's burden of proof.[10]

Dr. Bonuato opined that appellant did "not have overt pathology of his kidney related to cadmium exposure." Dr. Bonuato explained that beta-2 microglobulinuria caused by cadmium toxicity would be accompanied by elevated urine cadmium levels stating: "In [appellant's] case cadmium has never been demonstrated to be elevated in his urine suggesting that he does not have cadmium toxicity." Dr. Duckworth noted that the "work relatedness" of appellant's kidney abnormalities and cadmium exposure was "preliminary and provisional," as elevated beta-2 microglobulin could be caused by a variety of inflammatory and disease processes unrelated to any heavy metal exposures.

While appellant alleged that Dr. Duckworth improperly communicated with and influenced the opinion rendered by Dr. Vasin, the evidence does not support this contention. The

[10] *See William S. Wright*, 45 ECAB 498 (1994) (a physician's statement that appellant's medication "could very well have been" the cause of his condition was equivocal and speculative); s*ee Leonard J. O'Keefe*, 14 ECAB 42, 48 (1962) (where the Board held that medical opinions based upon an incomplete history or which are speculative or equivocal in character have little probative value).

record does contain a December 18, 2000 chart note in which a clerk at Dr. Vasin's office noted sending appellant's laboratory results to Dr. Duckworth. The Board notes that there is no provision which would preclude Dr. Duckworth from communicating with Dr. Vasin. Office procedures provide that Office medical advisers should refrain from oral communication with a physician selected as an impartial medical specialist to resolve a conflict in medical opinion.[11] There is no appearance that the courtesy of exchanging appellant's laboratory test results in any way influenced the opinion provided by Dr. Vasin. The Board finds that appellant's contention is not substantiated.[12]

Appellant has failed to establish that he sustained renal tubular dysfunction due to any cadmium exposure in the performance of duty.[13]

The decision of the Office of Workers' Compensation Programs dated and finalized March 18, 2002 is hereby affirmed.

Dated, Washington, DC
May 2, 2003

Colleen Duffy Kiko
Member

David S. Gerson
Alternate Member

Michael E. Groom
Alternate Member

[11] *See* Federal (FECA) Procedure Manual, Part 3 -- Medical, *Medical Examinations,* Chapter 3.500.6 (September 1995).

[12] Appellant submitted medical literature regarding the toxicity and carcinogenic properties of cadmium. However, these articles do not refer directly to the employee or to the employing establishment. The Board has held that excerpts from publications and medical literature are not of probative value in establishing causal relationship as they do not specifically address the individual claimant's medical situation and work factors. Therefore, such materials do not aid in determining causal relationship as they are of general application. *Gloria J. McPherson,* 51 ECAB 441 (2000).

[13] On appeal, appellant submitted new factual evidence; however, this evidence was not before the Office at the time it issued the final decision of March 18, 2002. The Board may not consider evidence for the first time on appeal that was not before the Office at the time it issued the final decision in the case. 20 C.F.R. § 501.2(c).