ARMED FORCES INSTITUTE OF PATHOLOGY
WASHINGTON, D.C. 20306

REPLY TO: THE DIRECTOR
ATTN: AFIP-RRR

PATIENT IDENTIFICATION		
AFIP ACCESSION NUMBER	CHECK DIGIT	SEQUENCE
1915385-7	2	0
NAME	SSAN	
No Name WS-7-83 S		
SURGICAL AUTOPSY PATH ACCESSION #		
PLEASE INFORM US OF ANY PATIENT IDENTIFICATION ERRORS		

WRC/LR/gh

Dan E. Connor, M.D.
Associated Pathologists, P.C.
210-25th Avenue, North
Nasnville, TN 37203

DATE:
19 January 1984

CONSULTATION REPORT ON CONTRIBUTOR MATERIAL

AFIP DIAGNOSIS: WS-7-83, Liver: Angiosarcoma of liver
2. History of industrial exposure to vinyl chloride from 1963 to 1980

Submitted sections of the liver (WS-7-83) exhibit extensive replacement and destruction of normal liver by a malignant neoplasm conforming to the features of angiosarcoma. Patterns of neoplastic growth include cavernous, sinusoidal and fibrosarcoma-like types. Although there are several fields with ductular proliferation, we are unable to identify the presence of cholangiocarcinoma.

The occupational history of exposure to vinyl chloride from 1963 to 1980 supports an etiopathogenetic relationship between vinyl chloride and angiosarcoma of the liver. The following references may therefore be of interest:

1. Makk, L., et al JAMA 230: 64, 1974
2. Thomas, LB., et al New Eng. J. Med. 292: 17, 1975
3. Gedigk, R., et al, Ann. N.Y. Acad. Sci 246: 278, 1975
4. Berk, P.D., et al, Ann. Int. Med. 84: 717, 1976
5. Popper, H., et al, Amer. J. Pathol. 92: 349, 1978
6. Fortwengler, H.P, et al, Gastroent 80: 1415, 1981

If available, we should like to obtain a representative section or sections of formalin-fixed liver tissue (or paraffin blocks) for teaching purposes.

The opportunity to review this case is appreciated.

Examining and Reporting Pathologist:
Lionel Rabin, M.D.
Dept. of Hepatic Pathology

WILLIAM R. COWAN
Colonel, USAF, MC
The Director

AP00034031

INTERNAL MEDICINE CONSULTANTS, P.C.
356 24th Avenue North
NASHVILLE, TENNESSEE 37203
(615) 329-3871

JOHN S. JOHNSON, M.D. — RHEUMATOLOGY
ROBERT M. JOHNSON, M.D. — HEMATOLOGY AND INTERNAL MEDICINE
JOHN S. SERGENT, M.D. — RHEUMATOLOGY
JOSEPH W. HUSTON, M.D. — RHEUMATOLOGY

February 17, 1984

Owens & Graves Chartered Attorneys
Legal Arts Building
730 Clark Street
Post Office Box 2757
Paducah, Kentucky 42001

Attention: Karen Alderdice

Re: [redacted]
Chart #7367

Dear Sirs:

I am writing in response to your letter requesting information on [redacted] I received it February 9. I apologize for the slight delay in mailing it, but I wanted to formally dictate his discharge summary in light of new information obtained regarding his post mortem liver biopsy.

I am enclosing copies of his final hospitalization discharge summary. I informed medical records at West Side Hospital to send all other pertinent records from their chart.

You will see that his death is attributed to hepatic failure based on the presence of an angiosarcoma of the liver. The angiosarcoma diagnosis was made by the pathologist at West Side Hospital and was confirmed after reevaluation of the tissue by pathologists at the Armed Forces Institute of Pathology. There was some concern that he may have a mixed tumor until it was reviewed by the AFIP. The final conclusion is that he has a pure angiosarcoma of the type usually associated with previous vinyl chloride exposure.

I am in hopes that the enclosed data will be sufficient for your purposes, but if additional records are needed, please don't

RECEIVED FEB 22 1984
KA

Re: [redacted]

hesitate to contact me directly. It may be of some help to contact Dr. Daniel Conner, who is the pathologist at West Side Hospital who obtained the post mortem liver biopsy and has a copy of the AFIP report.

Sincerely yours,

Joseph W. Huston, M.D.

JWH:QMT6

RECD FEB 17 1984

WEST SIDE HOSPITAL
Nashville, Tennessee

CLINICAL RESUME

NAME OF PATIENT: [redacted]
HOSPITAL NUMBER:
ROOM NUMBER: 702

DATE ADMITTED: 10-22-83
DATE DISCHARGED: 10-28-83 (Expired)

DISCHARGE DIAGNOSES:
(1) Death.
(2) Hepatic failure, secondary to angiosarcoma of th liver which is probably secondary to previous vinyl chloride exposure through his employment.
(3) Rheumatoid arthritis.
(4) Chronic prostatitis and prostatic obstruction with secondary E. coli urinary infections.
(5) Previous history of peptic ulcer disease.
(6) Persistent leukopenia and intermittent thrombocytopenia related to the liver disease and/or previous arthritis medications (gold and/or Penicillamine).
(7) Chronic external otitis.
(8) Chronic low dose Prednisone and full dose salicylate therapy for rheumatoid arthritis.
(9) Ex-tobacco user.
(10) Retroperitoneal hematoma.
(11) Pre-terminal hepato-renal syndrome with hyperkale

CONSULTANTS: Dr. Claude Workman (urology).
Dr. Dan Gremillion (gastroenterology).

PRESENT ILLNESS: This was the second and final West Side Hospital admission for this 61 year old, Caucasian male, from Dexter, Kentucky.

He has a complex past medical history which is well summarized in the admission note of his September, 1983 admission to this hospital. His history had been dominated by the presence of rheumatoid arthritis which had been active since 1976. This disease had required continuous therapy with a combination of anti-inflammatory (Prednisone and salicylate as well as remittive inductive (gold followed by Penicillamine) therapies. His treatment had been complicated by the unexplained persistence of leukopenia and intermittent thrombocytopenia which until recently, had been felt to be related to his medications. He was admitted to this hospital in September, 1983 because of the rather abrupt onset of anasarca associated with increasing abdominal girth and ascites. That hospitalization had revealed abundant evidence of severe hepatic insufficiency, manifested by hypoalbuminemia and secondary anasarca with ascites, severe, prolonged prothrombin time not correctable with vitamin K, and abnormal liver functions as well as an abnormal liver scan. Gastroenterology consultation with Dr. Daniel Gremillion had been obtained. However, the prothrombin time was not correctable enough to allow liver biopsy.

Cont'd------

He had gradually diuresed during that hospitalization with a combination of bed rest, sodium restriction, Aldactone, and Lasix. The total diuresis reached 23 pounds with near total resolution of his obvious pedal edema and moderate improvement in his ascites. He had been evaluated during the hospitalization by Dr. Claude Workman for persistence of prostatosis and evidence of urinary inflammation. He was anxious to go home in spite of his obvious poor prognosis and was finally released on September 23, with instructions to continue Prednisone 5 mg. daily, Aldactone 50 mg. t.i.d., Lasix 20 mg. daily, Geocillin, one gram q.i.d. and Entex LA, one b.i.d. for nasal congestion.

He had been reasonably comfortable at home, despite no change in his liver functions and prothrombin time at a repeat visit, half-way between hospitalizations. He was re-admitted on 10-22-83 with a four day history of increasing abdominal girth and pain as well as decreasing urinary volume and deterioration in higher intellectual functioning. He was admitted by Dr. Hagan who had been him in the emergency room because of the urinary symptoms. However, his care was immediately transferred to Dr. Gremillion and myself.

PHYSICAL EXAMINATION: On admission, blood pressure was 100/60. Pulse 80. Respirations 20. Temperature 98. Generally, an icteric, Caucasian male, complaining of right flank pain, but reasonably well oriented and no other acute distress. Examination of the skin revealed icterus and telangiectasi: Head,EENT- revealed conjunctival icterus and some inflammation of the left, external, auditory canal. Lungs were clear and cardiac exam was unremarkable. Abdomen was distended with bulging flanks and a positive fluid wave indicative of ascites. Right flank and lower quadrant tenderness was elicitable on palpation ofthe abdomen. Extremity exam revealed the previously described changes of rheumatoid arthritis as well as three plus pitting edema in the lower extremities. Neurological exam showed asterixis and occasional subcutaneous muscle twitching of the arms and legs.

LABORATORY DATA: Admission laboratory included slightly abnormal CBC with hematocrit of 39.2 and hemoglobin 13.6. Her indices were normal. Platelet count diminished at 115,000. White cell differential was normal. Urinalysis showed 1-3 WBCs but was otherwise unremarkable. Blood clotting studies showed protime of 19/12 while PTT was normal. Admission chemistry profile showed hyponatremia with sodium of 130 as well as hyperkalemia with potassium of 5.4. Other abnormalities included chloride of 95. Bicarb 23. BUN 38. Uric acid 11.5. Inorganic phosphate 5.1. Total protein 5.4. Albumin 3.1. Alkaline phos. 171. Total bilirubin 3.7. SGOT 71. Subsequent laboratory values included an elevated SGGT at 248 with a normal SGPT. Subsequent serum creatinine was elevated at 2.3. Blood typing showed blood type A positive with an anti-C antibody identified. Admission EKG showed left axis deviation and non-specific ST-T wave changes. Liver, spleen scan showed diffuse, severe liver disease which was totally non-specific. Abdominal ultrasound showed a large amount of ascitic fluid and multiple focal hyperechoic lesions throughout the liver. The ascites compromised the quality of the full exam. Abdominal C-T scan showed the interval development of a huge, right retroperitoneal hematoma.

Cont'd.....

WEST SIDE HOSPITAL

CONTINUATION OF CLINICAL RESUME NASHVILLE, TENNESSEE PAGE 3

NAME OF PATIENT: [redacted]

HOSPITAL COURSE: It soon became apparent that his flank pain and change in clinical status was brought on by the evolution of a retroperitoneal hematoma. This was the consequence of his low prothrombin time. His serial CBC indicated the evoluation of significant anemia with hematocri openly dropping to 29.7. He required blood transfusion as well as the administration of fresh, frozen plasma to temporarily halt the bleeding diathesis and aloow successful transfusion. He did not respond to continued vitamin K therapy.

Additional laboratory monitoring revealed the evolution of continued hyponatremia and hyperkalemia which ultimately responded to intermittent therapy of potassium retriction and intermittent use of D-50W, insulin, sodium bicarb, and enemas with Kayexalate and Sorbitol.

Patient had undergone endoscopic examination per Dr. Gremillion prior to the discovery of the retroperitoneal hematoma. This was done in an effort to see if his evolving anemia and pain was related to upper intestinal bleeding or variceal bleeding. Varices were noted but no bleeding was identified.

[redacted] showed improvement after transfusion but his situation deteriorated rapidly beginning on the morning of 10-27-84. He developed clinical and laboratory findings indicative of the hepatorenal syndrome with sudden worsening in renal function and hyperkalemia. His potassium did respond to withholding Aldactone and the above measures. However, at 1:00 PM on 10-27-83, he developed ventricular tachycardia and subsequently became hypotensive and semi-conscious. He responded initially to Lidocaine and increased potassium lowering measures but ultimately required IV Dopamine for additional blood pressure support. He showed a rapid deterioration in all systems from that moment on, and by 9:00 PM, on the night of the 27th, he had markedly diminished renal output and cardiac output as well as persistently elevated serum potassium. By the next morning, he was moribund and essentially totally unresponsive, requiring high dose Dopamine and other supportive measures. He then went through a series of cardiac arrhythmias and also lost all cardiac output and signs of life, leading to the pronouncing of death at 9:05 AM on 10-28-83.

Permission was requested and received from the family to perform an open liver biopsy as part of a post-mortum study. The initial impression was angiosarcoma was present but possibly mixed with other tumor tissue. The biopsy specimen was then forwarded to the Armed Forces Institute of Pathology who recently returned their opinion that angiosarcoma was the only tumor type present and that the pattern was highly suggestive of that induced by vinyl chloride exposure.

JH/bt
D: 2-11-84
T: 2-13-84

Joseph Huston, M D

DIGESTIVE DISEASE CONSULTANTS
337 21ST. AVENUE NORTH
NASHVILLE, TENNESSEE 37203
615 327-4242

ROBERT C. DUNKERLEY, M.D., P.C.
DOUGLAS P. MITCHELL, M.D., P.C.
DANIEL E. GREMILLION, JR., M.D., P.C.

FELLOWS OF THE AMERICAN COLLEGE OF PHYSICIANS
AMERICAN BOARD OF INTERNAL MEDICINE
CERTIFIED IN GASTROENTEROLOGY

January 23, 1984

Mrs. [redacted]
Route 1, Box 2
Dexter, KY 42036

Dear Mrs. [redacted]n:

The post-mortem liver biopsy on your husband, [redacted] did confirm angiosarcoma of the liver which is a type of malignancy.

It is in my opinion that this angiosarcoma was the prime cause of his liver failure and death.

Please give this information to your insurance company and have them correspond with me if there are any further questions.

I hope you are doing well. I am very sorry that [redacted] passed away.

Very sincerely yours,

Daniel E. Gremillion, M.D.
DANIEL E. GREMILLION, JR., M.D.

DEG/cs