

REDACTED

GPO 441-15-811.4

CLINICAL RECORD

TISSUE EXAMINATION

SPECIMEN SUBMITTED BY

John Veirling, M.D.

DATE OBTAINED

8/13/74

SPECIMEN

Outside liver biopsy slides

BRIEF CLINICAL HISTORY (Include duration of lesion and rapidity of growth, if a neoplasm)

43 year old male with two year exposure to vinyl chloride.

Biopsy thru hepatic vein

(See our S74-1853)

PREOPERATIVE DIAGNOSIS

OPERATIVE FINDINGS

POSTOPERATIVE DIAGNOSIS

SIGNATURE AND TITLE

PATHOLOGICAL REPORT

NAME OF LABORATORY

Surgical Pathology Section

ACCESSION NO(S).

S74-1902

(Gross description, histologic examination and diagnosis)

Gross:

The specimen consists of 1 submitted slide bearing the label of St. Anthony Hospital, Louisville, Kentucky and the accession number S74-2753.

Microscopic:

The section includes several fragments of hepatic tissue which includes portions of two small portal tracts. These are infiltrated by a few lymphocytes. There is mild fatty vacuolization of hepatic cells but otherwise the hepatic lobules, hepatocytes and sinusoidal cells all appear normal. Slight focal variation in staining is thought possibly to represent artefact.

Diagnosis:

1. Mild fatty metamorphosis, liver.

(OVER)

(Continue on reverse side)

SIGNATURE OF PATHOLOGIST

Louis B. Thomas

DATE

8/21/74

AGE

43

SEX

M

RACE

W

IDENTIFICATION NO.

PATIENT'S IDENTIFICATION (For typed or written entries give: Name—last, first, middle, grade; date; hospital or medical facility)

REGISTER NO.

10-35-16-2

WARD NO.

Submitted by: St. Anthony Hospital
Louisville, Kentucky

TISSUE EXAMINATION
HISTOLOGY Form 515
515-105

BFG67296

Note: The submitted section included in this report was reviewed by Dr. Popper who concurs with the diagnosis of mild fatty change. At the same time, liver sections previously reported under our accession no. S74-1853 were also reviewed and discussed with Dr. Paul Berk.

The conclusions from this review and discussion can be summarized as follows:

(1) The moderate portal tract and focal intralobular fibrosis seen in S74-1853 are not specific; i.e., they may be due to alcohol damage to the liver but some other type of chemical injury cannot be ruled out.

(2) Certain features which seem to be characteristic of the liver changes in vinyl chloride polymerization workers are absent in Mr. Kennedy's liver. These are: (a) no fibrosis of the hepatic capsule; (b) no proliferation or enlargement of sinusoidal and lining cells; (c) no sinusoidal dilatation; (d) no enlargement (megacalocytosis) of hepatocytes.

(3) The amount and the distribution of the hepatic fibrosis seen in the S74-1853 sections (biopsy obtained by peritoneoscopy) do not appear to be sufficient to explain the splenomegaly. It is theoretically possible that the hepatic fibrosis and the enlargement of the spleen have different etiologies. Whether the patient actually has portal hypertension and whether the splenomegaly is a result of hepatic lesions is not known at the present time.

Robert Stern, M.D.

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