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BUSINESS ADMINISTRATOR
J. W. [illegible]

May 14, 1969

American Mutual Liability Insurance Company
120 LaSalle West
South Bend, Indiana 46601

Attention: Mr. Robert E. Bigley

Re: Floyd Costner
Vs: U.S. Gypsum Co., East Chicago

Dear Mr. Bigley:

First, I would like to apologize for the delay in answering your letter.

I first saw Mr. Coster in June, 1967, at which time he was referred by Dr. S. J. Sroka with respiratory difficulty and epigastric distress. The patient was admitted to St. Margaret Hospital, Hammond, Indiana on August 5, 1967 and discharged August 11, 1967. Bronchoscopy was performed which was negative. A chest X-ray showed fibrotic changes of the upper half of the lungs. Laminagrams showed chronic fibrotic changes of the upper lobes with apical pleural capping. There was some deviation of the trachea to the right. Cholecystograms showed medium-sized radio-opaque calculi. Upper G-I X-rays showed a hiatus hernia. Pulmonary function studies were carried out which showed moderate reduction of function, restrictive. The patient was discharged back to the care of Dr. Sroka.

Because of persisting complaints of chest pain, Mr. Costner was re-admitted on December 8, 1967 for lung biopsy. He was discharged on December 24, 1967. Chest X-ray showed progressive fibrotic process with widening of the mediastinum and shift of the trachea to the right. Vital capacity was 1.7 liters with restrictive features. An electrocardiogram showed abnormal T-wave changes suggestive of diffuse anoxia. Laboratory examination was non-contributory. Skin tests were negative for fungus or acid-fast bacilli.

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On December 14, 1967, the patient was taken to the Operating Room. The 4th intercostal space was entered and exploration was carried out. This patient had marked parietal and pleural fibrosis as well as adhesions. There were numerous nodular densities scattered in the upper lobes. Lung tissue was somewhat emphysematous and there also was visceral pleural thickening. A specimen was obtained from the parietal pleural area along with lung tissue and this was sent to pathology for frozen section. This showed no malignancy and adequate tissue sampling. Final report of the pathologist was pleural and lung fibrosis with severe chronic inflammation and foreign body type giant cell reaction and anthracosis with moderate bronchiectasis. These diagnoses were obtained from the pathological feature of marked interstitial fibrosis associated with severe collections of chronic inflammatory cells, mainly lymphocytes, plasma cells, and mononuclear cells. A few multinuclear foreign body type giant cells were also seen. The lung parenchyma showed marked thickening of the alveolar walls and evidence of interstitial fibrosis. The other area showed complete collapse. The small bronchioles showed dilated lumen and chronic inflammatory cell infiltrate. There was no definite evidence of asbestosis. However, with the polaroid light, several double refractible crystals were seen which suggested pneumonoconiosis of indeterminate nature. The patient's post operative course was satisfactory except for persisting small pneumothorax. He was discharged on December 24, 1967.

The patient was again seen by me on several occasions. He was also seen by Drs. Sroka, Reed and Ferry. His main complaints are those of persisting progressive pulmonary insufficiency, persisting chest pain and epigastric distress, and the incapacity to perform any gainful activity--even limited normal daily activity. A chest X-ray taken on February 18, 1969 showed a fluid level in the pneumothorax portion of the left chest. The patient was re-admitted to the hospital on March 7, 1969 and discharged on March 15, 1969. The main attempt of this admission was to determine further progression of his pulmonary disease, functional evaluation, and therapy for possible improvement of his pulmonary status. The fluid was removed and the laboratory report of culture and smears showed no pathological findings. The fluid appeared compatible with transudate. The patient was placed on intermittent positive pressure breathing and mucolytic agents, as well as dietary control for his epigastric difficulty. With this, some improvement took place although no marked benefit was noted. Chest X-ray showed what was interpreted to be slightly further progression of pulmonary fibrosis and incapsulated hydrothorax which disappeared after thoracentesis. An electrocardiogram at this time showed non-specific subendocardial ischemia of the posterolateral wall. An upper G-I series at this time showed no evidence of diaphragmatic hernia. However, it was noted that there was slight thickening of the fold of the lower esophagus. An attempt at pulmonary function testing was made which was totally unsatisfactory because of marked

restriction of ventilatory status. The only valid information one can gather for the patient's vital capacity at the maximum effort is less than one liter per minute, now showing both restrictive and obstructive features. I felt the maximum breathing capacity or any other information was meaningless under such circumstances. The patient was instructed to continue his diet and tracheobronchial hygiene. Steroid therapy was tried without much success.

The total picture of this patient is that of progressive pulmonary disease resulting in progressive fibrosis and restriction of ventilation to the extent that he is unable to maintain himself except for minimal activity. It is true that the lung biopsy showed no definite asbestosis. The findings are consistent with that of a noxious chemical or foreign body exposure. The double refractive bodies seen in the lung specimen and on polaroid light suggest the etiology is that of pneumonoconiosis. I am somewhat dismayed with the limitation of a pathological laboratory where no spectographic study can be carried out. I have to state, however, that the patient's disease is quite rapidly progressive, quite incapacitating, and is of recent origin, and have to assume exposure was of recent origin. I do not think this patient will be able to return to gainful employment, and it would be highly undesirable for him to return to an environment where dust and air pollution are prevelant. I cannot definitely state the etiological agent except for the suggestion as stated above.

If there is any further information you wish, I will be happy to comply with your request. I am most anxious for equitable compensation for this patient, as he certainly needs all the help he can get.

Sincerely yours,

YSK:gf

Y. S. Kim, M.D., F.A.C.S.

cc: S. J. Sroka, M.D.