

An Autopsy Case of Weil's Disease

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Weil in 1886, described several human cases of leptospirosis. Weil's disease is caused by *Leptospira icterohaemorrhagiae*. It is characterized by an acute onset with headache, severe muscular pain, anorexia, icterus, conjunctival injection and etc.

Recently we sectioned the patient with leptospirosis hebdomadis (subgroup of Weil's disease) by chance and found numerous leptospirae in the liver and kidneys.

CASE REPORT

K. M. 55 year-old man. Restaurateur.

Chief complaints: Icterus, general fatigue, nausea, muscular pain, conjunctival injection and anorexia.

Present illness: He was acutely ill with high fever, influenza, on Jan. 1, 1969 and then after 3 days anorexia, nausea, icterus and decrement of urine volume were noted. Crisis appeared, but general fatigue became severe so he was admitted to Shimonoseki National Hospital on Jan. 8, 1969.

Status praesens at admission: He was good nutritious and the consciousness was clear. Severe icterus and conjunctival injection were prominent. Abdominal pain was severe, especially tenderness was striking on the right hypochondrium.

Laboratory findings at admission: RBC 398×10^4 , WBC 23,200, Hb 81 %, Total bil. 16.4 mg/dl, I. I. 55 u., Serum prot. 5.1 g/dl, Al. 37.6 %, α_1 -Gl. 10.0 %, α_2 -Gl. 21.7 %, β -Gl. 10.4 %, γ -Gl. 20.5 %, Al. phos. 14.5 u., Acid phos. 7.1 u., GOT 300 u., GPT 140 u., LDH 680 u., BUN 95.5 mg/dl, Bleeding-time 2 min., Coagulation time more than 30 min. Urinarysis; Specific gravity 1.001, pH 6.0, Sugar (-), Protein 30 mg/dl, Sediment; RBC 1-2/f, WBC 4-5/f, Squamous epithel. 2-4/f. Spinal fluid; Not examined.

Clinical course: After admission immediately the patient was catheterized but excreted little urine. Next day diagnosis was considered as Weil's disease, therefore,

antibiotics (SM, TC) and transfusion (1,500 ml/day) were administered. He vomitted 3 days after admission. Being taken care of bleeding, he was administered steroids (Rinderon) and hemostatics were added to transfusion. Cramp and a confused state of mentality occurred. He fell into coma P. M. 5:00 on Jan. 10. Hepatic coma was considered and Ancoma (glutamic acid derivative) was dropped, and a little chrolpromadin was administered. He was improved transiently, but suddenly died with carmp P. M. 7:00 on Jan. 11. The serum obtained on second day of admission was found to have a titer of 1:160 against an antigen of *Leptospira hebdomadis* (Akiyami B), 9th day of illness.

POSTMORTEM EXAMINATION

A postmortem examination was performed on Jan. 11, 1969.

Gross anatomical findings

General: The body was that of a moderately built, nutritious, 55 year-old man measuring approximately 159 cm. in length and weighing about 58 kg. Rigor mortis was present and there was dependent lividity. Severe icterus, conjunctival injection, scattered subcutaneous hemorrhages and excoriations meaning itch were noticed. No swelling of superficial lymph nodes was noted.

Abdomen: Subcutaneous hemorrhages and excoriations were scattered. Abdominal swelling was not noted.

Primary incision: A typical "I" incision was made. Subcutaneous fat tissue and muscle were well developed.

Abdominal cavity: Peritoneum was smooth, glistening and icteric. Swelling of lymph nodes was not seen. Ascites, icteric and slightly bloody, measured about 200 ml.

Pleural cavities: There was dense, old, fibrous adhesion, right lateraly.

Pericardial sac: 20 ml. of clear, icteric pericardial fluid was contained.

Heart: Weight, 300 gms. The subepicardial fat tissue was well developed. The myocardium was red-brown in color. The walls of the left and right ventricles measured up to 1.2, 0.2 cm. in thickness, respectively. The endocardium was smooth. The ostia were not remarkable. Subendocardial and subepicardial petechiae were scattered. Coronary arteries showed slight tortousities.

Lungs: Weight, left, 580 gms., right, 800 gms. Grossly, both lungs looked approximately the same findings. In general the lungs were dark red in color, voluminous and edematous.

Spleen: Weight, 80 gms. The capsule was slightly thickened. Consistency was moderately soft.

Liver: Weight, 1,600 gms. It was enlarged, firm and yellowish in color. Architecture of the lobules on the cut surface was obscure.

Kidneys : Weight, left, 200 gms., right, 200 gms. It was yellowish. The capsules stripped with ease leaving icteric surfaces and clear venulae stellatae. Sections through the kidneys revealed sharply demarcated cortical and medullary tissue. Medullary striations were slightly indistinct. Pelvic mucosae were distinctly hemorrhagic. The ureters showed no gross abnormalities.

Stomach : A little black-brown material was contained. An erosion and a little-finger's head sized ulcer were seen.

Intestines : Submucosal petechiae were scattered.

Urinary bladder : Submucosal petechiae were seen.

Tongue : Ulcer formation with blood coagula was seen.

Adrenal glands, pancreas, sexual organs, esophagus, thyroid glands, aorta : Not remarkable.

Central nervous system : Not examined.

Microscopic findings

Heart : The normal architecture was fairly preserved. But in some areas slight degeneration and swelling of the muscle fibers were noticed.

Lungs : Sections revealed hemorrhage and edema. There were no signs of pneumonia.

Spleen : Plasma cells, lymphocytes, numerous macrophages phagocytized fragments of erythrocytes were present in the pulp. Hyalinization of the central arteries was noticed.

Liver : The normal architecture was distinctly destroyed. Parenchymatous degeneration, destruction of the liver cell cords and conspicuous dissociation of the hepatic cells were characteristic. There was active proliferation of hepatic cells, that was, mitoses and polynucleated hepatic cells, particularly two-nucleated cells, were prominent. Slight necroses occurred in the center of the lobules. Bile stasis was not so striking. Enlargement of Kupffer cells was seen. (Fig. 1, 2.)

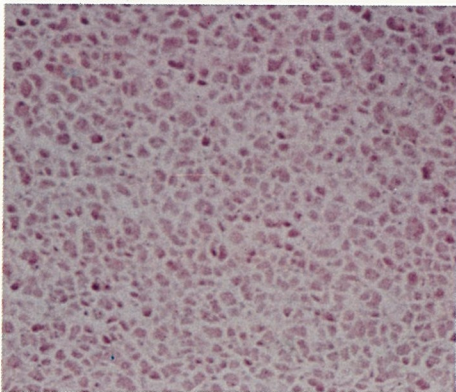


Fig. 1. See destruction of the liver cell cords and conspicuous dissociation of the hepatic cells. (HE. $\times 100$)

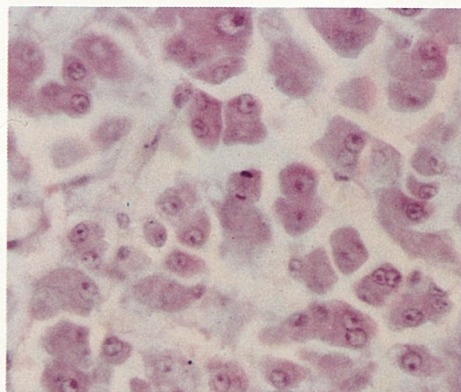


Fig. 2. Mitosis and two- or three-nucleated hepatic cells are seen. (HE. $\times 400$)

Kidneys: The normal architecture was fairly preserved. Slight chronic inflammatory cell infiltration, particularly lymphocytes, was present in the interstitial tissue. Slight degeneration of the tubular epithelia was noted. Submucosal hemorrhage was noticed in the pelvic mucosae.

Pancreas: The normal architecture was well preserved. Slight chronic inflammatory cell infiltration, lymphocytes and plasma cells, was present in the interstitial tissue.

Stomach: Ulcer formation that attained to the muscle layer was noted.

Intestines: Not remarkable. The normal architecture was well preserved.

Urinary bladder: Submucosal hemorrhage was prominent.

Tongue: Ulcer formation with fibrin and necrotic masses was noted.

Parotis gland: Lymphocytes and neutrophils infiltrated slightly in the interstitial tissue and glandular spaces.

Skeletal muscles: Not remarkable. Vacuolation, sarcolemmal cell proliferation and necroses were not noticed.²⁾ No findings were characteristic of Weil's disease.

In addition, numerous leptospirae were demonstrated by special staining (Levaditi's method)⁸⁾ in the liver and kidneys. In the liver organisms were present in the liver cells and interstitial tissue, whereas, in the kidneys, in the interstitial tissue, tubular epithelia and tubular lumens. (Fig. 3, 4.)

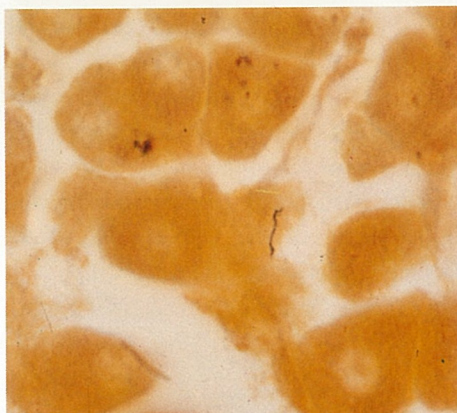


Fig. 3. Leptospira in the liver cell. (Levaditi's stain $\times 1000$)

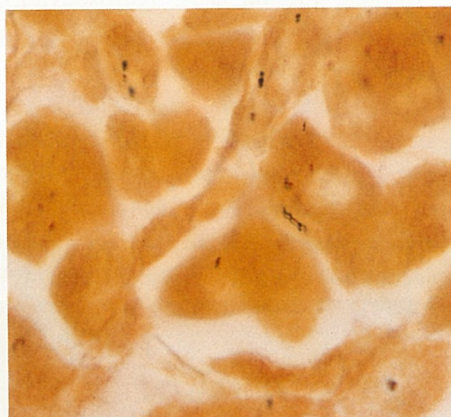


Fig. 4. Leptospira in the interstitial tissue in the liver. (Levaditi's stain $\times 1000$)

Pathological diagnoses ; 1) Weil's disease (Leptospirosis hebdomadis), 2) Interstitial nephritis (Leptospiral nephritis), 3) Hemorrhage and edema of the lungs, 4) Generalized petechiae ; renal pelves, urinary bladder, skin, conjunctivae, epicardium, endocardium, stomach, intestines, esophagus, 5) Gastric ulcer, 6) Lingual ulcer, 7) Acute parotitis.

COMMENT

The patient was suspected of suffering from influenza on Jan. 1, 1969, anorexia, nausea, icterus, decrement of urine volume, general fatigue, conjunctival injection and etc. (characteristic of Weil's disease⁴⁾⁷⁾) became prominent. He was admitted to hospital and treated under diagnosis of Weil's disease, but died on 11th day of illness. The serum obtained on 9th day of illness was found to have a titer of 1:160 against an antigen of *Leptospira hebdomadis* (Akiyami B). Immunological test confirmed the diagnosis, leptospirosis hebdomadis. High fever was not caused by influenza but leptospirosis hebdomadis. Microscopically the findings were noticed in the liver and spleen, that were characteristic of Weil's disease. Many leptospirae were also found in the liver and kidneys. Nevertheless he complained severe myalgia, we could not find the characteristic signs in the skeletal muscles.

Weil's disease was most prevalent 1910 to 1915, in Japan. But since the time the disorder have showed tendency to decrease gradually, especially, after the world war. In recent decade of years, the incidence of the disorder is showed in Tab. 1.

Table. 1. Sectioned Weil's disease within the recent decade, in Japan.
(1961-1970) (from "Bokensyuhō")

	Total sections	No. of cases
1961	13,276	12
1962	15,041	7
1963	16,350	9
1964	16,464	1
1965	19,476	5
1966	19,020	8
1967	20,902	9
1968	25,237	3
1969	25,224	4
1970	23,904	10

Weil's disease is classified into several types immunologically, but morphological identification of the specific organisms is impossible, even under the electron microscope. (Tab. 2) Although morphology identical with one another, they are completely different serologically.¹⁾⁵⁾

Immunological test is a Schüffner-Mochter agglutination test. It is estimated as an elevation of titer of antibody against specific organism. Serum obtained from non-infected man have a titer of O, whereas, those from infected man have a titer of 1:300 or more, after 1:5,000, rarely 1:20,000,¹⁾³⁾⁶⁾ in our case 1:160 against *Leptospira hebdomadis* (Akiyami B).

Table. 2. Immunological classification of leptospirae

Serogroups	First detection		Reservoirs
	The year	The land	
<i>L. icterohaemorrhagiae</i>	1914	Japan	mouse, white rat, rodents, pig, fox, monkey, dog, cat, horse, hen, calf, opossum
<i>L. canicola</i>	1931	Holland	dog, hamster
<i>L. hebdomadis</i>	1918	Japan	wild rat, opossum, dog, badger, skunk
<i>L. pomona</i>	1937	Australia	pig, dog, badger, skunk, wild cat, cattle
<i>L. grippityphosa</i>	1928	Russia	wild rat, rodents, badger, skunk
<i>L. autumnalis</i>	1925	Japan	wild rat, white rat, dog, guinea pig
<i>L. australis</i>	1937	Australia	wild rat, shrew, dog, pig, guinea pig, opossum, badger
<i>L. mitis</i>	1940	Australia	pig, cattle
<i>L. botavia</i>	1926	East Indies	white rat, cat, wild rat, pig, dog, guinea pig

Leptospirae are world-wide spread. Wild rats are the chief reservoir of infection, but organisms have been demonstrated in field mice, foxes, cats, pigs, horses, dogs and gophers. The role of rats and dogs in the transmission of these diseases has been confirmed and emphasized¹⁾⁶⁾. In our case the reservoir is not clear, but perhaps rat, dog or cat may be considered.

Because of an occupational predisposition, these infections would seem to be most prevalent among man. These disease occurs more frequently in sewer workers, fish handlers, miners, rice-field workers, slaughterhouse employees, sugar-cane cutters and farmers. In our case the patient was a male restaurateur.

SUMMARY

We have reported Weil's disease (leptospirosis hebdomadis) which we had recently sectioned by chance. In this report we have represented the pathology and comment of our case. We also detected numerous leptospirae in the liver and kidneys.

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