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THE CONCEPT OF ASSIMILATORY SYSTEM OF PAULESCU DATING FROM 1912

Constantin IONESCU-TÎRGOVIŞTE[1]

[1]"Prof. Nicolae Paulescu" National Institute of Diabetes Nutritional and Metabolic Diseases, Bucharest, Romania
Corresponding author: C. Ionescu-Tîrgovişte
E-mail: cit@paulescu.ro

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CHAPTER III
HEPATOPATHIES CAUSED BY BIOTIC AGENTS

Liver diseases caused by biotic agents divide into several categories, depending on the origin of the parasites causing them: animal, vegetal and microbial.

ART. I – ANIMAL PARASITES

Given its position on the trajectory of the blood coming from the intestine, the liver receives directly the eggs or germs of animal parasites from food or drinks.

Hydatid liver cysts

Etiology – The cause of hydatid liver cysts is *taenia echinococcus,* a parasite which is commonly met in the dog and, exceptionally, herbivores' intestine.

We have already described in volume I (page. 288) of this work the evolution of the embryo of this worm up to the stage of hydatid bladder, so we will not repeat ourselves and we shall only indicate the special points of the hepatic localization of the cyst.

Tapeworm eggs present on vegetables together with dog feces or carried out by drinking water can be swallowed by the man and can form hydatid cysts of the liver.

The canine origin of these cysts was proven by observations and experiments (LEUCKART). In Iceland, where this disease is most common, the inhabitants, sheep owners, have many dogs with which they live without complying with the most elementary rules of hygiene. We had various opportunities to check this statement: thus, for instance, a 55 year old woman, affected by a hydatid liver cyst, kept for four years in a room she occupied together with her four children, a dog that used to defecate there.

The influence of sex is less marked; out of 44 personal cases, 19 are men and 25 are women. The age, according to our statistics, varies between 19 and 66; but we also have to mention that in the hospitals where we conducted our research, there were also patients under the age of 16.

Pathologic anatomy – Tapeworm eggs reaching the stomach are attacked by the gastric juice; thus, their walls weaken and they dissolve. From each of them, an embryo with spicule exits, perforates the tissues and reaches the starting point of the portal vein, being carried by blood to the liver. There it loses its hooks, closes and forms a hydatid bag, whose volume grows progressively.

The irritation determined by this bag in the hepatic conjunctive tissue causes the formation of a fibrous membrane, 1–3 mm thick; this membrane has an abundant vascular network, which serves to feed the cysts.

Under this cover there is another membrane, similar to coagulated albumine, made of amorphic layers.

The hydatid membrane which belongs to the parasite is covered by another internal layer, full of granules (fertile membrane), which creates the vesicles and echinococci. Inside the cover there is a distinct number of ovoid vesicle, as large as a pigeon egg, sometimes a hen egg and echinococci which have the shape of whitish granulations, made of a head, four valves and approximately 30 hooks.

The vesicles float in a clear liquid, transparent like the water of a rook, neutral, with the density of 1000 to 1010, which does not contain albumin. Sometimes, this liquid is opaque and, when the hydatids die, it becomes similar to albumin.

Usually, there is only one or two cysts in the liver; more seldom their number can be three, five, six or even more.

The usual localization of these cysts is the posterior edge, in the median part, the upper surface and, sometimes, the lower part of the liver.

They are round, spherical or flat due to compression of the thorax and the diaphragm. The size varies from that of a cherry stone to that of an adult's head; sometimes, they have an excessive growth. In some cases, they get to the surface of the liver, where they have a whitish characteristic color.

The hydatid cysts which occupy the posterior edge of the liver penetrate the upper region of the diaphragm, in the pleural cavity, compress the lungs and sometimes move the heart. The cysts of the left lobe generally move up towards the epigastrium. Those on the lower surface spread towards the abdomen, compress various organs (stomach, intestine, portal vein, vena cava) and cause multiple disorders.

As the cyst grows, the hepatic tissue atrophies, so that sometimes a lobe is destroyed, and the remaining gland hypertrophies in order to replace the compromised function. A slight degree of cirrhosis can develop around the cyst and, if there are several cysts, this sclerotic lesion can be pretty extended; but when it is generalized with just one cyst, as in the case of a wine drinker we treated, it must be deemed a coincidence.

The hepatic vessels usually remain untouched; however, branches of the portal vein can alter and cause hemorrhages. The hepatic veins ulcer can create heart vesicles; if the cyst suppurates, it becomes the starting point off pyogenic pulmonary embolisms, which cause abscesses. In a personal case, the lower vena cava was obstructed by a stone which, present at the level of an enormous liver cyst, caused ascites and edema of the limbs.

Bile ducts, most often compressed, can ulcer the bile as well, which spreads in the cystic bag and kills the echinococcii. Sometimes, hydatids enter the bile ducts and generate bile retention, which is accompanied by purulent angiocholitis if the cyst suppurated.

As a matter of fact, the rupture of hydatid cysts can occur in the stomach, intestines, peritoneum, pleura, bronchi, pericardia and, as an exception, the abdominal wall. It rarely has a cure; most often, it generates very serious accidents.

The echinococcus dies following the extraction of the hydatid liquid and through the thickening of the fibrous membrane, with or without calcification; this membrane, when withdrawn, compresses the vesicles and chokes them, transforming them into a yellowish semifluid mass, similar to mastic for window panes, which on the microscope, reveals echinococci hooks, fat drops and cholesterine crystals.

Together with the hydatid cysts mentioned above, there are other parasite productions which also occur, where vesicles are no longer grouped into one bag, but develop in large numbers (*multiocular or alveolar echinococci*).

On the whole, hydatids represent tumours of various sizes, from the size of an egg to that of the head of an adult. They are irregular and have an unequal consistency, tough and cartilage-like sometimes, soft and fluctuating other times. Sometimes, multiple tumours spread like beads.

Tumours are surrounded by a conjunctive stroma, made of liver or other organs. This fibrous structure presents free spaces of multiple alveoli, with various dimensions, from that of a grain to that of a pea, an egg and each contains one hydatid vesicle, usually bended; large alveoli contain two or three vesicles.

These hydatids have relatively few echinococci and the small ones are generally sterile. They suffer the same modifications as regular cysts and turn into a gelatinous or fat mass which contains hooks and crystals.

The liver volume is so large that it can weigh up to 10 kg. The hepatic tissue is also icteric. The portal vein is sometimes obliterated and the bile ducts, compressed.

Symptomatology – Some hydats cysts appear, grow and die in the depths of the liver, without drawing the patient's attention and without betraying their presence in any way, so that they are usually discovered upon necropsy.

Most often, cysts manifest themselves through the increase of the liver volume. The dullness of this organ grows up to the 4th rib and down towards the navel. Exceptionally, upon percussion, it produces a special vibration, called *hydatid murmur,* which one of us noticed only twice in more than 60 patients. Upon palpation, elastic, fluctuating and protuberated surfaces are sometimes felt, and, more rarely, a piriform, pediculate tumour.

At the same time, the abdomen grows and reveals, as applicable, a circumscribed lump or a general tumefaction. If the lower surface of the liver is the location of the cyst, the compression of the portal vein causes ascites and that of bile ducts causes jaundice, symptoms which are similar to those of wine drinkers' cirrhosis.

We indicate the disorders coming from the compression of neighbouring organs, such as the movement of lungs and heart, pleural overflows etc.

Functional disorders consist in pains which manifest first through a sensation of fullness and weight and, later one, if the tumour rises up to the surface of the liver and the cyst is swollen, through acute pains, with irradiations towards the right shoulder.

The appetite is average; digestion is slow and, if the stomach is compressed, vomiting occurs. Usually, the patient does not lose excessive weight.

The disorder is apiretical and fever only occurs when the cyst suppurates because of the infection of intestinal origin. Fever crises are short and remittent, starting with shivers and ending in perspiration. The temperature oscillates between 40° and 36°.

The evolution of hydatid cysts is insidious, slow and continuous. The duration, according to our personal statistics, varies between 3 and 10 years, since the beginning of clinical manifestations.

The death of echinococci happens quite often and necropsy reveals liver cysts where the hydatids are turned into an atheromatous mass.

More often, cysts break and the symptomatic image varies depending on the point where the rupture occurs.

If the cyst empties in the stomach or intestine, it causes vomiting or evictions of alveoli containing hydatids.

When it perforates the diaphragm and enters the pleura, it causes painful pleurisy, usually purulent, which overflows and contains hooks, gelatinous hydatid remains and even bile, as we saw in three of our cases of the recent years.

When the cyst invades the basis of the lung, it creates slow evolution pneumonia, limited to the lower part of the organ to a cavern sound and hectic fever; when a bronchia is ulcerated, it creates abundant expectoration, containing remains of hydatids, bile and sometimes puss.

The bag rupture in the pericardia causes purulent peritonitis, and in the peritoneum, a suppurated peritonitis, rapidly lethal.

Finally, the cyst opening into the vena cava is followed by embolisms and rapid death; that which occurs in the bile ducts is accompanied by pains similar to those of hepatic cramps, jaundice and infection phenomena.

Briefly, the fatal ending of hydatid cysts is the consequence of a rupture of infection of the bag; more rarely, it comes from hepatic insufficiency, when the glandular parenchyma is largely destroyed.

Semiology – Diagnosing liver hydatid cysts has often serious difficulties.

A large liver with a smooth, circumscribed, elastic or fluctuating curve suggests the presence of echinococci. But what makes the diagnosis certain is the extraction by puncture of a clear, transparent, non

almbuminoid liquid containing echinococcus hooks. We must know that we should not draw the conclusion that the disorder is absent following a puncture with a negative result.

Another highly important sign in this case if provided by the bile, which is not increased in the case of liver echinococcosis.

Cysts that go up the left hypochondrium simulate a lesion of the spleen; those of the anterior edge of the liver resemble a relaxed bladder; those going down the iliac fossa or invade the abdomen suggest an ovary cyst, a renal cyst or hydrophrenosis; finally, those exiting the diaphragm and going towards the thoracic cavity suggest pleural overflowing.

Moreover, the tumours of the big epiploon and the cysts of the mesenteric muscle are easily confounded with liver echinococcosis.

The prognosis of liver hydatid cysts is always serious, considering it can never be predicted how they will end, whether they heal through the death of the parasite, whether they will suppurate, whether they will open into a serous cavity in the lungs, in a vein, in the bile ducts or in the digestive tube. The anterior and surface bags are less serious than the bags in the profound and posterior regions, since they are more accessible to therapeutic agents. Progressive weight loss and phenomena of hepatic insufficiency (hemorrhages) must arise suspicion. On the contrary, the reduction of the liver volume or the spontaneous detachment of the cyst or following a puncture are positive and represent an almost certain sign of healing.

Prophylaxis and treatment – The prophylaxis of liver echinococcosis consists in avoiding drinking unfiltered water and raw, poorly washed and unpeeled vegetables. We should add that patients must not own dogs which carry tapeworm or should treat them so as to eliminate it.

The treatment must aim at killing the parasite through a puncture or removing it through an incision.

First, an exploration puncture is performed with a Parvaz syringe, in order to see whether the cyst content is suppurated or not.

The aspiration puncture is prepared with a special device such as the instrument of Potain, equipped with a long needle with the head one millimeter long. This sterilized needle is inserted in the protuberated point of the cyst; then, it is adjusted to the aspirating device; finally, it is slowly and progressively removed, so that to allow the bag walls to close and form the orifice. The patient must remain in bed after the puncture with the recommendation to take an opiate potion. It is best to completely empty the average cysts; for those extremely large it is recommended to leave part of the liquid so as to avoid syncope accidents which are, however, rare.

The aspiration of hydatid liquid changes the living conditions of the parasite which, generally, dies. Died vesicles dry up, the cyst retracts and the bag is impregnated with calcareous salts. Sometimes, the liquid reappears and a new puncture is necessary.

For instance, in a personal case, a 35 year old woman came to the hospital with a pain in the left hypochondrium and a progressively growing excrescence in the epigastrium, this tumour lifted the heart and pushed it outward. The liver was huge; the anterior edge crossed the iliac fossa, the navel and the 9th right rib. The spleen was normal. A puncture eliminates 1.300 ml clear, transparent and unalbuminoid liquid; this puncture was followed by vomiting and urticaria. One month later, the bag acquired again contour, so a new puncture was performed and 2 liters of yellowish, opalescent liquid were extracted, containing a large amount of albumin. After that, the tumour stopped reproducing; the liver volume dropped, the pains stopped and the patient's general condition rapidly improved.

In order to be certain that they killed the parasite, after extracting the liquid, some physicians insert in the cystic bag 100 mg Van Swieten liquid which they remove after 10 minutes.

The aspiration of the cystic liquid is not usually followed by any accident, however, there are patients who, shortly after the puncture, manifest signs of nausea, vomiting, hiccups and fever, followed by an episode of rash caused by the absorption of a certain amount of hydatid liquid. Other times, these accidents end in rapid death.

If a hydatid cyst does not disappear after two or three aspiration punctures, surgery is necessary. It is imperious in the case of suppurated cysts, old cysts whose bag lost elasticity, cysts with numerous vesicles.

After removing the hydatids, scaring is sometimes slow, particularly if the bile enters the bag and the patient can die because of a hectic condition.

Liver cysticercus

The cysticercus or taenia solium larva is rarely found in human liver, where it does not cause considerable disorders, given the small number of individuals which develop simultaneously. In animals, for instance in pigs, where these larvae are more numerous, they often cause hepatic sclerosis.

Liver haematobia

Etiology – The haematobic dystome is usually found in the veins of the bladder and urethra; sometimes, it is hosted in the portal vein. The eggs, which it lays in large amounts, spread into the hepatic veins and break them causing hemorrhages. Other times, the embryos cross the vascular walls, infiltrate in the tissues and determine more or less serious sclerosis.

Pathologic anatomy – The liver, increased, is hardened and sclerosed. The dystome eggs which cause this form of sclerosis occupy the veins in the portal spaces, so that the cirrhosis they cause is similar to that of wine drinkers.

Symptomatology – The clinical manifestations of this disorder are little visible at the beginning and consist of lack of appetite and dyspeptic disorders. In a more advanced stage, the signs of ordinary cirrhosis are visible: tumefaction of the spleen, meteorism, ascites, jaundice. This form of cirrhosis grows slowly and progressively; it is unsettling, since it prevents circulation in the portal vein.

Semiology – It is very difficult to diagnose this disorder; it is often possible to do so due to the hematuria resulting from the presence of the parasite in the urinary ducts.

The prognosis is very serious, since the disease usually ends up in death. However, if the dystome dies, healing occurs spontaneously.

Prophylaxis and treatment – In Egypt, where haematobiosis is found in edemic condition, physicians recommend to avoid drinking Nile water, unless it is perfectly filtered and not to each fruit and vegetables unless they have been thoroughly rinsed.

There is no cure for the disease, given that we do not know of any substance that might kill haematobia and release the organism from this parasite.

Liver pentastome

In the human liver, two distinct species of pentastome have been noted: *pentastoma constrictum,* found in Egypt, and *pentastoma denticulatum,* found in several cases in Germany.

Denticulate pentastoma is the first stage of development of the worm which lives in the nostrils and frontal sinuses of the dog (LEUCKART). It has white, flat, lance-shaped body, 5 mm long, 1 mm wide, denticulate at the surface.

Pentastoma has an elongated cylindrical body, 15 mm long, 2 mm wide, with transversal constrictions at the periphery.

The mechanism through which these parasites enter the human body is unknown; probably the eggs or larvae are swallowed with drinks or vegetables that were not properly rinsed.

These parasites, once reaching the liver, close within a fibrous capsule and take the form of a tubercle, 3 or 4 mm in diameter. The cysts are usually impregnated with calcareous salts; they contain the curved worm.

The low number of these cysts, their small volume, the superficial localization explain the liver tolerance. They do not cause any significant disorder and go unnoticed, being discovered at the necropsy.

Liver coccidae

Ovoid coccidae which are present in rabbits and hares determine hepatic lesions which consist in sclerosis with whitish tubercles, whose content, analyzed on the microscope, is made of small ovoid bodies, similar to helminth eggs (MEGNIN). These coccidae were isolated, cultivated and studied by BALBIANI.

Similar parasites have been met in humans. A 50 year old woman was seized by remittent fever, with pains in the limbs, light diarrhea, albuminuria and tumefaction of the liver and spleen. She died six weeks later and the liver revealed numerous caseified centers, whose content presented coccidae that could have been grown (SILCOCK).

ART. II – VEGETAL PARASITES

The liver is the location of vegetal parasites; it is one of the organs where actinomyces are located.

Liver actinomycosis

Etiology and pathogenicity – Hepatic actinomycosis results from the insertion in the digestive tube of actinomyces, radial-shaped fungi that grow on certain plants, usually certain grass grains (see vol. I, pag. 847). This disease, sometimes primary, is mostly secondary, depending on the localization of the parasite in the intestine, mainly in the cecum.

Pathologic anatomy – The volume of the liver affected by actinomycosis grows and presents, in one or several points, lumps where an incision reveals masses with sizes varying from that of peanut to that of a fetus head, with a liquid with the consistency and aspect of pus. A more thorough analysis reveals that this liquid comes from a large number of cavities which give the parasitic mass the aspect of a sponge.

Fine cuts made along a fresh actinomycosis focus allow the recognition of *nodules* (see vol. I, pag. 848). These nodules are made of a mycelium and sporiferous organs; they have a specific radial structure and are surrounded by an area of young cells. Soon, under the influence of diastases secreted by the parasite, the nodule focus diminished and tends to open outward. Finally, the mycosis focus gradually grows in volume and reaches, in some cases, the abdominal wall or the diaphragm, pleura, lung and opens outward through fistula trajectories, which present a purulent liquid, often mixed with bile. Other times, it enters the intestine.

Symptomatology – The hepatic localization of actinomycosis is slow at the beginning. Later on, it translates into the increase of the liver and pains in the right hypochondrium because of peritoneal adherences with the diaphragm, the abdominal wall or other organs. Simultaneously, digestive disorders, progressive weight loss and weakening occur.

In the second stage of the disease, when the foci suppurate, remittent fever with shivers and perspiration occurs, which exhausts the patient. Finally, if there are intestinal lesions, unstoppable diarrhea installs and the patient succumbs to marasmus.

The evolution of this disease is slow and progressive and the duration spreads from several months to several years. Healing is possible in the beginning, when the parasite dies. Subsequently, suppuration generates death quite rapidly. Sometimes, purulent peritonitis speeds up the end.

Semiology – It is very difficult to diagnose hepatic actinomycosis. Identifying the parasite on the microscope is a pathognomonic sign; but this is only possible if there is a fistula or, at least, if there was an aspirating puncture.

Hepatic abscesses are distinct from actinomycosis through the absence of actinomyces.

Syphilis does not cause fever and ends in scaring.

The tubercles coexist with similar lesions in other organs.

In order to recognize actinomycosis during lifetime a highly careful analysis is necessary and we must not be surprised if in most cases, this disease is recognized only at the necropsy.

The prognosis is among the most serious, since hepatic actinomycosis usually ends up in death.

Prophylaxis and treatment – Given the etiologic conditions of this disease, we must warn persons who handle straws and spikes of grasses not to chew and swallow these items which can carry parasites.

The treatment consists in administering 3–6 g of potassium iodide a day. In case of failure, laparotomy must be performed.

ART. III – MICROBIAL PARASITES

A large number of microbes cause these hepatic diseases, related either to the presence of such agents in the liver or to the action of the toxins they release in this gland.

We shall discuss these diseases as *hepatic localization* of microbial disorders and we shall particularly insist upon their symptomatology, semiology and treatment.

§ 1.

The evolution of **paludism** has two distinct stages: an acute one, called paludic fever and a chronic one. Each of these stages has a distinct *hepatopathy,* that we shall discuss below.

I – Acute pyretic paludic fever
(Syn.: *Hematuric bilious fever*)

The hepatopathy which occurs in the acute stage of paludism consists in the congestion and tumefaction of the liver, whose color turns dark brown, even black (pigment) and whose parenchyma acquires a soft and unctuous consistency. Small bruises sometimes appear at the surface of the gland. The bile ducts are free, but the bile is dense, sometimes almost solid and strongly colored in brown.

The hepatic epithelium, made of tumid and opaque cells, impregnated with pigmented granulations, suffers a granular-fat degeneration which can be felt or not and which translates, when significant, into phenomena of hepatic insufficiency: jaundice, hemorrhages etc.

The liver parenchyma is affected to various degrees during paludic fever.

Usually, its alteration has no visible symptom.

But sometimes, when the glandular cells are largely destroyed, the syndrome of *hepatic insufficiency* occurs. The patient turns yellow and has bilious vomiting. The feces, initially very dark, are later discolored. The urine, rare, contains bile; very often, hematuria occurs and total anuria becomes visible and rapidly leads to death. Soon, multiple hemorrhages occur which, besides very common hematuria, are abundant and difficult to stop: epistaxis, purpura, hematemesis, melena, etc. Increasingly weak, the patient falls into a condition of prostration; the tongue is very dry; he is delirious and dies in a coma.

However, healing is not impossible.

This hepatic complication, frequent in Madagascar and Senegal, determined us to call it *hematuric bilious fever* (see vol. I, pag. 369).

We hereafter present one of our cases[1]:

A 24 year old young man, in good physical shape, embarked on March 12th, 1895 on a ship to Madagascar. In June, he was seized by paludic fever which persisted until July 15th. Upon his return to France, he had again several fever episodes in October and November.

On December 9th, he was again seized with fever and the next morning his face turned yellow. Then he was seized by greenish vomiting and dark diarrhea; the urine, very rare, was almost black. Then it disappeared completely. The patient was very weak; his voice was faint, but he did not suffer from headache or delirium. He died on the night of December 18th, in a state of hypothermia (34°).

The necropsy revealed the liver weighing 2200 gr., smooth, soft, unctuous and very pigmented, with the uniform color of pus. The gallbladder contained highly viscous, semi-solid and black bile.

On the microscope, the hepatic cells were tumid: the cilia were disturbed and impregnated with pigmenting granulations. The secreting tubes of the kidneys had similar alterations, while the intra-renal excreting ducts contained a homogeneous hyaline substance which completely obliterated the light of these tubes.

Difficult to recognize in the beginning, before the occurrence of jaundice, this disease is then obvious due to its characteristic symptoms: yellow color, multiple hemorrhages and other phenomena of hepatic insufficiency.

The prognosis is always very serious and often fatal. The treatment of this hepatopathy is the same as that for paludism (see vol. I, pag. 379). Hepatic and renal insufficiency shall be fought against through the methods already mentioned.

II – Chronic sclerotic paludic hepatopathy
Syn: *Paludic cirrhosis, hypertrophic biliary cirrhosis*

The hepatopathy which occurs in the chronic stage of paludism was studied for the first time and described

[1] E. LANCEREAUX, *Traité des maladies du foie et du pancréas*, pag. 176.

by one of us[2], who, in his observations, relied on the observations collected from the hospital, in most cases.

Etiology and pathogenicity – This hepatopathy is caused by the paludism agent (see vol. I, pag. 355).

It is present in persons who spent time in marshy areas, especially those who lived for a while in Algeria, Senegal, Madagascar, Tunisia, Vietnam, Tonkin, Italy, in the region of the Lower Danube etc.

Most patients had before crises of intermittent fever, generally few in number; but there are others who do not remember having suffered from this fever, although they lived close to marshes or in paces suspect of paludism.

The influence of sex is considerable: out of 45 patients consulted by us, 38 were men and only 7 were women.

Age does not seem to be a factor worth considering; even children can get sick in the first years of life and their special physiology in marshy countries, coinciding with the tumefaction of the liver and spleen, has been known for long. Often, the disease seems to be congenital.

Food excesses causing hepatic congestions predispose to paludic hepatitis. The same happens with the excess of alcoholic drinks and wine abuse, which complete the action of the paludic agent, generate mixt, paludic and enolic cirrhosis characterized by bivenous sclerosis which is missing in simple paludism hepatopathy.

The pathologic anatomy, Symptomatology and Evolution of this disease are treated elsewhere (see vol. I, pag. 361, 370 and 375).

Semiology – This hepatopathy is easy to recognize at the beginning because of the simultaneous and progressive increase of the liver and spleen, which subsequently coincides with jaundice called hemapheic (with no biliary pigments in the urine and without discoloration of the feces). Moreover, the absence of any hepatic pain, the dilation of abdominal subcutaneous veins, any visible ascites coexisting with generally good health do not create any doubt on the diagnosis of paludic cirrhosis.

This disease can be mistaken, before the occurrence of jaundice, with amyloid degeneration or liver lymphedema; but the absence of leucocytosis and prolonged tubercular or purulent lesions, with diarrhea and albumimuria, help set the diagnosis.

The paludic hepatopathy[3] has analogies with the fat form of wine drinkers' cirrhosis; it is distinct from them through its slow evolution – enolic cirrhosis, when jaundice installs, which ends in a few days or weeks, and especially through the absence of phenomena of alcoholic poisoning.

A disease which is not always easy to separate from paludic hepatitis is the obstruction of bile ducts by a calculus, a narrowing, a cancerous tumour; but the features of jaundice, which is bilipheic, the discoloration of the feces and the absence of the increase of the spleen volume allow to set the diagnosis of this hepatopathy.

The prognosis of paludic hepatitis is serious; this disease, if allowed to develop, leads to death. But its slow evolution allows the efficient action through an appropriate treatment and diet and helps to obtain, at least in the first two stages, pre-jaundice and jaundice, improvements which are almost similar to complete recovery.

Prophylaxis and treatment (see vol. I, p. 380).

In recurrent fever, the liver is tumid, congested and its epithelium sometimes suffers a granular-fat degeneration.

When this alteration is very advanced, jaundice and multiple haemorrhages occur. Jaundice, noted in about a quarter of the cases, usually occurs in the third or fourth day. Haemorrhages are cutaneous, nasal, pulmonary, gastro-intestinal, renal and uterine, which in this last case can cause abortion.

When the ending is fatal, the tongue dries, the urine disappears, the patient is weak and succumbs to comma.

It is very easy to diagnose this disease and the prognosis is serious.

The treatment is similar to that for recurrent fever (see vol. I, pag. 386).

[2] E. LANCEREAUX, *Atlas d'anat. path,* Paris, 1871, pag. 60, pl. 7.

[3] E. LANCEREAUX, *Traité des maladies du foie et du pancréas,* pag. 351 and *Atlas d'anatomie pathologique,* pag. 96, pl. 13.

§ 2.

In **smallpox**, the liver is congested, tumid, yellowish, soft and lax; it is the location of an alteration of glandular elements which suffer some sort of coagulation (unclear tumefaction), followed by fat transformation. Sometimes, this alteration causes the destruction of hepatic cells; the result is hepatic insufficiency which translates into multiple hemorrhages (hemorrhagic smallpox). These hemorrhages occur in the smallpox vesicles, in the derma or in the cellular tissue; they also occur in the organs and cause stomatorrhagia, epistaxis, hemoptysis, hematemesis, metrorrhagia and, more rarely, hematuria and enterorrhagia. Hemorrhagic foci are also noted in the muscles, along the nerves, in joint cavities, on the serous surface etc.

This hepatic complication in smallpox is sometimes noted at an early stage, in the third or fourth day of the disease, other times later, after the vesicle starts to suppurate (see vol. I, pag. 394 and 397).

In *early black smallpox* – which refers to organism poisoning with toxins of the agent of this disease, the initial symptoms are completed by multiple hemorrhages. The temperature is very high, the pulse is fast, and breathing accelerated; there are conditions of agitation and even delirium. The rash is purple; vesicular eruption becomes violent; vesicles dry and are impregnated with blood serosity which gives them a black color, while among them, they form petechias and bruises and hemorrhages occur: epistaxis, hemoptysis, hematema, melena, metrorrhagia, hematuria etc. At the same time, the patient has the impression of choking with excessive anxiety: the lips turn black; the tongue dries; urine disappears; finally, the patient falls into a coma and succumbs towards the fourth or fifth day. Sometimes, death occurs even on the second day (fast black smallpox).

In *late black smallpox* caused by the suppuration agents and which is very rare, pustules fill up with blood and are simultaneous with hemorrhages: purpura, epistaxis, hematemesis, metrorrhagia, etc. The patient is agitated and raves; he feels a sensation of choking and the tongue dries, urine disappears and he succumbs to coma.

It is not difficult to diagnose this disease when there is a smallpox epidemics. The prognosis is very serious, since this disease rapidly causes death.

The treatment is specified in the article *smallpox* (vol. I, pag. 400).

During **measles**, the hepatic cells suffer sometimes a more or less significant coagulation (unclear tumefaction). Subsequently, there is the syndrome of hepatic insufficiency: purpura, epistaxis, melena, hematuria, etc. This hemorrhagic measles is extremely serious: it almost always ends in death.

During **scarlet fever**, the liver is often affected; it becomes soft, lax, yellowish and its epithelium has coagulation alterations (unclear tumefaction) and fat degeneration, with a fragmentation of tubercles, which no longer have columns made of closely interconnected cells, but lines of cells separated by free spaces[4]. When these lesions are more serious, multiple hemorrhagic foci are also noted on the skin, in the mucous membranes, in the muscles, along the nerves etc.

In this case, the symptoms of scarlet fever (see vol. I, pag. 420) are completed by tumefaction of the liver which exceeds by one or two finger lengths the costal margin, headache, weakness, insomnia, delirium, dry tongue. These phenomena are often associated with a yellowish color and various hemorrhages; the rash becomes petechial and is followed by epistaxis, hematuria, metrorrhage, melena, etc. This hemorrhagic scarlet fever, which is rapidly lethal, is mostly noted in individuals whose liver is already altered (alcoholism, enolism, paludism).

During **exanthematic typhus,** the liver is often affected; it is soft, lax, with an unctuous consistency and its cells are the location of more or less serious coagulation (unclear tumefaction). This liver alteration translates into symptoms of hepatic insufficiency which add to those of the primary disease (see vol. I, pag. 430).

Typhoid fever is sometimes accompanied by lesions of the liver which is, in the beginning, congested, and then becomes soft, lax, with an unctuous consistency, with the color of mustard and its cells suffer a degeneration consisting of the unclear tumefaction of the protoplasm and, later on, a fat transformation.

[4] E. LANCEREAUX, *Traité des maladies du foie et du pancréas*, Paris, 1899, pag. 163.

Moreover, the liver sometimes presents small necrosis foci invaded by lymphatic cells, which form specific nodules. The bile, abundant in the beginning, becomes scarce and less colored when the glandular parenchyma is affected.

When the hepatic cells are deeply affected, the symptoms of typhoid (see vol. I, pag. 442) are completed with those of hepatic insufficiency, which translate into a slight yellow color of the sclerotic membrane, excessive weight loss with delirium and carphology and multiple and early hemorrhages (epistaxis, purpura, stomatorrhagia, metrorrhagia, etc.). In this case, death usually occurs fast.

§ 3.

Since the hepatopathies of yellow fever and serious feverish jaundice (hepatosis) are described in detail in volume I of this paper (pag. 461 and 466), we shall not insist upon them here, reserving the right to discuss a highly frequent hepatic disease called catarrhal jaundice, which we associated with the previous ones due to their common features.

Apyretic jaundice
(Syn: *Catarrhal jaundice*)

This disease, described as *catarrhal jaundice*, is usually attributed, according to VIRCHOW, to the obstruction of the choledoc by a mucous stopper, which seems to originate in the congestion of the mucous membrane of the last bile ducts and the duodenum.

Given the weak intensity of the yellow color, the early tumefaction of the liver and of the spleen and especially the clearly defined evolution of the disease, we shall consider this form of jaundice as the effect of an alteration of hepatic cells connected with a general disorder and not as the result of a simple local disorder.

Etiology and pathogenicity – The analysis of 50 cases studied by one of us[5] for over thirty years reveals that catarrhal jaundice affects young and especially old people. The analysis of our observations shows that we had 5 cases under 20 years of age, 30 cases between 20 and 30, 9 cases between 30 and 40 and 6 cases between 40 and 45.

Sex is relevant, since out of 50 cases, we had 43 men and 7 women.

As regards their profession, we noted the frequency of this disease among masons, merchants, wine merchants, bakers etc.

It is worth noticing that this form of jaundice mainly occurs in June, July and August.

The cause of this disease is unknown.

As an occasional cause, 21 of our patients accused the excess of food and drink; 5 invoked a cold, while others could not provide any information in this respect.

This form of jaundice is most often sparse; sometimes, it occurs in a large number of individuals, which gives it the appearance of an epidemic disease.

At any rate, apyretic jaundice seems to have a food origin, many rare cases and most epidemics, especially those noted in the military field, being attributed to a negligent diet.

Pathologic anatomy – The study of hepatic lesions in catarrhal jaundice continues, none of our cases ended in death and we do not know of any necropsy report published by other authors.

Symptomatology – After a few days of weakness and muscular pain, when the appetite decreases, the tongue becomes dirty, whitish, nausea and constipation occur which make the patient eliminate urine; he notices that his urine acquires a darker color and the sclerotic liquids starts to turn yellow. In some cases, this pre-jaundice stage seems to be missing.

[5] E. LANCEREAUX, *Traité des maladies du foie et du pancréas,* Paris, 1899, pag. 209.

The skin acquires an intense yellow color which lasts for the entire duration of the disease. The abdomen is slightly affected by meteorism or not at all; the liver, with increased volume, exceeds the false ribs by 2 or 3 finger widths and is not painful upon percussion. The spleen is also tumid and has 14–7 cm on the transversal diameters and 21–25 cm on the longitudinal diameter. The tongue is white and relaxed; anorexia is complete and disgust for meat is often absolute; digestion is difficult; eructation and even vomiting are present. The constipation persists; it is sometimes replaced by mild diarrhea; the feces are discolored and clayish. The urine, rare, does not exceed 500–800 ml; it is dark, colored brown by bile pigments; its density varies between 1020 and 1030; it contains relatively little urea and does not contain any sugar or albumin.

Fever is constant, the axillary temperature fluctuates between 36° and 37,5°; the pulse, almost always slow, is between 50 and 70 beats per minute. Breathing is normal. Moreover, there is a feeling of exhaustion, sadness and insomnia. Sometimes, mild rash and even epistaxis occur.

This jaundice stage lasts for fifteen days on average, but it can also prolong to 4 weeks, after which the overwhelming stage disappears, the strength and joyfulness reappear as does the appetite. At the same time, the color of the urine turns paler and more abundant, while the feces regain color. The patient is convalescent. However, the liver and the spleen reduce their volume more slowly, going back to normal after 1 month or 6 weeks.

The evolution of this disease is almost cyclic, its average duration is 3 weeks, more rarely 2 or 4 and finally the patient heals.

Semiology – The diagnosis of catarrhal jaundice relies on apyrexia, tumefaction of the liver and spleen and knowledge of its evolution.

This disease is easily differentiated from serious jaundice which is accompanied by fever and has a shorter duration. It is also distinct from enolic cirrhosis through the absence of alcoholism signs, from paludic cirrhosis through jaundice which is not hemapheic, from mere biliary obstruction through splenic tumefaction.

The prognosis of catarrhal jaundice is benign, since it does not cause death. By continuing his milk-based diet, the patient does not lose much weight and his health is restored within 4–5 weeks.

Prophylaxis and treatment – If there are any uncertainties referring to the existence of etiological conditions of this disease, it is difficult to formulate efficient prophylactic recommendations. Everything makes us believe that it comes via digestive paths, that is why we recommend to monitor food and drinks and avoid any excess.

The patient will have to remain in the room or even in bed; at any rate, he must avoid strenuous effort and tiredness; moreover, he must protect himself from cold. He will have an exclusive milk diet plus alkaline waters such as Vichy.

We must also fight constipation with lavages, saline purgatives or calomel. The patient shall be administered 2 tablets of 1 g of quinine sulfate every evening, at an interval of a quarter of an hour. We shall prescribe a cold bath every morning, or at least a cold alcoholized lotion followed by dry massage.

In case of **plague, cholera, flu,** the liver sometimes suffers alterations similar to those noted in eruptive fever, which lead to coagulation and fat degeneration of glandular cells.

These very serious and often lethal conditions manifest through a yellow coloration of teguments and multiple hemorrhages (purpura, epistaxis, etc.).

The same applies to **diphtheria hepatitis.**

Dysentery is often followed by hepatic abscesses, some caused by amoeba, others, by pus-forming agents. We shall discuss them in detail when we reach the hepatopahties caused by pyogenic microbes.

§ 4.

Erysipelas and particularly pneumonia sometimes cause hepatic alterations, frequent in wine drinkers. These alterations consists of unclear tumefaction of the cells which can be affected by fat degeneration, more or less advanced.

Under the circumstances, generally mild jaundice occurs accompanied or not by signs of hepatic insufficiency (purpura, epistaxis, etc.) and a general bad condition often leading to death.

§ 5.

Suppuration microbes (streptococci, staphylococci, colibacilli) generate two types of hepatopathies, some being the effect of organism poisoning by diastases and remains of food of such agents, others resulting from the presence of such microbes in the hepatic parenchyma.

1. Toxic hepatopathies with pyogenic microbes

These hepatopathies are met within *pyemia* (MARÉCHAL), ulcerous endocarditis (LANCEREAUX)[6], puerperal infections, phlebitis of the umbilical cord in new born babies etc.

In all these diseases, the remains of nutrition of pyogenic microbes are drained in the blood and poison the organism, altering the hepatic cells.

Under certain conditions, the volume of the liver increases, being soft, lax and yellowish. The glandular cells are tumid, impregnated with protein or fat granulations and sometimes destroyed. The bile is slightly colored and yellowish. The spleen, congested, is often, like the liver, the focus of small pyogenic infarctions.

After several episodes of violent fever, the occurring jaundice is distinct through a light yellow color and is soon generalized to reach the entire tegument.

The urine, scarce, contains bile pigment and often albumin. Sometimes, the patient has vomiting sensations, diarrhea, especially preceding jaundice; the feces are yellowish or discolored.

Then, the skin covers with petechias and multiple hemorrhages occur: epistaxis, hematemesis, melena, etc.

The patients, exhausted and agitated, are seized by delirium followed by coma and death.

The diagnosis of these hepatopathies relies on liver tumefaction and especially jaundice and multiple hemorrhages which coexist with a suppuration focus.

The prognosis is among the most serious.

Aseptic bandages, rigorous measures against puerperal accidents in women who recently gave birth, care in treating the ligature of the umbilical cord prevent from such hematopathies.

The treatment consists of quinine in large doses (1–2 gr.) and, if there are any signs of hepatic insufficiency, purgatives and diuretics.

2. Purulent hepatopathies

Liver abscesses are always secondary and result from the transport of suppuration microbes to this organ.

Besides the trauma itself, this transport can follow several different paths:

1. arterial path;
2. venous path;
3. biliary path.

[6] E. LANCEREAUX, De l'endocardite suppurée, etc. *Gaz. med.*, Paris, 1864.

A – Arterial purulent hepatopathies
(Syn: *Metastatic hepatic abscess*)

Etiology and pathogenicity – Once frequent before the practice of asepsis, such abscesses are almost inexistent at present, except for when pyogenic agents, entering pulmonary veins or the heart, are carried by blood up to the hepatic artery, where they stop. Such suppurations are quite frequent in abscesses of the lungs and ulcerous endocarditis.

Sometimes, metastatic hepatic abscesses are met after furuncles, anthrax, nail infections, bone abscess and especially purulent phlebitis, and the pulmonary capillary network cannot always stop the microbes which reach the liver together with venous blood.

Pathologic anatomy – The liver, highly congested, is considerably increased in volume and weight. Its parenchyma, which is violet, is dotted with bruised, black or yellow stains. Abscesses, usually multiple, are spread or grouped at the surface and in the organ; generally, they are small and their size varies from that of a pea to that of an olive; seldom, they are larger when, fewer in number, they grow and unite themselves.

The liver section analyzed on the microscope shows that the hepatic tissue is covered by deep cavities creating abscesses (vol. I, pag. 670); these purulent foci sometimes host streptococci, as in a case of angina complicated by multiple suppurations, other times staphylococci, as was the case of a liver abscess which occurred within several days during the convalescence after anthrax, while other times colibacilli, etc.;

The hepatic cells are unclear and tumid and sometimes partially destroyed. The portal veins and the bile ducts are intact. Other organs are suppuration foci as well.

Symptomatology – Arterial hepatic abscesses manifest through remittent fever, with large abscesses and typhoid or adynamic phenomena which rapidly lead to coma. The fever is often accompanied by jaundice related to the alteration of glandular cells and sometimes hepatic insufficiency disorders (multiple hemorrhages).

The liver is tumid and exceeds the false ribs by one or two finger widths; it hurts when the physician resorts to pressure or percussion in one or several points. Very rarely, notable lumps are visible accompanied by a sensation of friction (peri-hepatitis). The spleen is large and other organs, particularly the lungs, translate the suppuration through specific symptoms.

This hepatopathy develops very fast and ends in a few days in death. Healing is only possible if there is a single abscess which opens to the exterior.

Semiology – Metastatic hepatic abscesses do not raise serious diagnosis problems. They can be mistaken for typhoid fever and acute tuberculosis; but the febrile type (remittent), the painful liver tumefaction, the occurrence of jaundice allow to avoid such an error. In order to recognize the initial focus of the suppuration, various organs must be examined and, if it disappeared, its recent scar must be sought.

The prognosis, very serious, is almost always lethal.

Prophylaxis and treatment – Prophylaxis consists in emptying, disinfecting and carefully bandaging the suppuration foci, especially when they are close to a vein.

The treatment, which is null, only consists of administering quinine sulfate, alcoholic aconite solution and cold baths.

B – Venous purulent hepatopathies
(Syn: *Hepatic abscess*)

Etiology and pathogenicity – These hepatopathies are caused by purulent lesions located in the original area of the portal vein, originating usually in dysenteric ulcers, gastro-intestinal ulcers and suppuration foci of the pelvic organs.

I – The coexistence of dysentery with hepatic abscesses has been known for long; the geographic distribution and the epidemic recrudescence of these two diseases coincide[7] and ROUIS admitted that in Algeria, hepatic abscesses are preceded by dysentery in nine cases out of ten.

In case of dysenteric intestinal ulcers, the branches of the portal vein are the door of entrance for dysenteric agents and pyogenic microbes. These microbes are carried by blood to the hepatic capillaries, where they cause abscesses. Alcohol abuse and an excessive meat diet are not, contrary to general claims, causes which predispose to hepatic suppurations in warm countries.

The two sexes are subject to this disease which is more common in men than in women. It can affect any age group; but adults are more prone to hepatic abscess.

II – Another important cause of hepatic suppuration is *the ulcer of digestive paths*[8]*, appendicitis and abscesses of the pelvic organs.*

For instance, simple ulcers, cancer ulcer and generally any solution for the continuity of the digestive tube can generate liver suppurations. This suppuration is very rare in intestinal ulcerations of typhoid and tuberculosis, which undoubtedly refers to the fact that in such cases, an inflammatory obliteration of the veins occurs around the ulcer.

All the abdominal organs, tributary to the circulation of the portal vein, even those that only have anastomotic relations with this system, can be the starting point of the hepatic abscess. This is the action of periprostatic phlegmons, periuterine abscesses, especially those following a birth[9].

New born babies also present hepatic suppurations following the septic ligature of the umbilical cord.

Pathologic anatomy – I. – In dysenteric suppurations, the liver is generally large, which is dictated by the size of the abscess and the congestion of the organ. Usually, the anterior edge of the liver reaches the vicinity of the iliac crest.

No part of the gland escapes the suppuration, but the right lobe, at the convex and the posterior edge, is mostly affected. For instance, ROUIS' statistics presents the following figures: right lobe, 154 cases; left lobe, 33 cases; Spiegel's lobe, 9 cases.

Most often, hepatic abscess is single; sometimes, it is double or multiple. Out of 146 necropsies, ROUIS found a single abscess 110 times; multiple abscesses, 36 times.

Generally, suppuration is limited to a part of the gland; sometimes, it disappears almost completely as a result of purulent drainage.

When the abscess opens outward, naturally and artificially, the purulent cavity gradually withdraws, the walls draw near and finally unite. The resulting scar forms at the surface of the liver and in depth a depression similar to that left by liver syphilis. In general, open abscesses scarify rapidly; SANX quotes a case when an incised abscess scarified 8 days after the surgery. Sometimes, when the orifice does not allow complete drainage, a fistulous trace is left which persists for months or even years.

According to some authors (CAMBAY), in certain situations, the pus present in the hepatic abscesses could resorb on the spot.

II. – Liver suppuration subsequent to gastro-intestinal ulcerations (other than those of dysentery), appendicitis, abscesses of the pelvic organs etc. is only distinct from the previous ones through the multitude of purulent foci.

Besides the suppuration focus, the hepatic parenchyma is slightly modified; however, in some cases of acute suppuration with intense fever, the glandular cells are touched by unclear tumefaction.

The bile has a normal aspect; the bile ducts are intact.

Sometimes, adherences, false membranes and even purulent proximity collections occur in the pleura, peritoneum, lungs, spleen, kidneys and brain.

Symptomatology – Liver suppurations start with more or less intense fever, which translates into intermittent or remittent crises, staring with shivers and ending in abundant perspiration.

[7] E. LANCEREAUX, *Traité des maladies du foie et du pancréas,* Paris, 1899, pag. 227.
[8] Idem, *Mémoires d'Anat. path.,* Paris, 1863. *Traité d'Anat. path.,* Paris, 1879–1881, vol. II, pag. 962.
[9] E. LANCEREAUX, *Traité des maladies du foie et du pancréas,* Paris, 1899, pag. 231.

Simultaneously, the patient has a feeling of weight, tension, pain in the right hypochondrium, which worsens in two or three days, becoming acute, pulsatile and irradiating towards the corresponding shoulder and at the back. The intensity is so high, that it changes the patient's features and forces him to take a position which allows him to relax the muscle, especially the diaphragm, which can compress the liver. The result is an acute breathing discomfort, which the liver tumefaction stresses even more. The pulse is fast during febrile crises.

Anorexia occurs; the tongue is dirty; bilious vomiting, diarrhea and reduction of the amount of urine appear.

These symptoms last for the duration of the abscess, after which the pain lowers in intensity and dyspnea diminishes. The liver, large, increases volume further, widens the thorax basis spreading the ribs and lifts the abdominal wall which presents a number of lumps, particularly at the epigastrium level.

Later on, the symptoms continue with rapid weight loss with changes of the face features; jaundice, usually mild, which occurs only in one fifth of the cases, is a negative signal; vomiting, caused by hepatic insufficiency; adynamia; a typhoid condition; delirium; incontinence and finally coma which ends in death.

The evolution of hepatic suppurations is acute in warm climates, ending rapidly in death (90 out of 100 cases).

In temperate climate, the abscess evolution is slower and sometimes the duration is very long, the disease being almost silent. This duration varies between 10 days and 180 days (ROUIS).

Several months and even years are needed for the patient to recover.

Liver abscess sometimes spontaneously opens into the peritoneum, in the right pleura, through the lungs and bronchi, very rarely in the pericardium, the digestive tube, the bile ducts, the skin, especially at the level of sub-sternal belt.

In some cases, purulent foci heal after they spontaneously open. For instance, in a personal case, a 33 year old man who contacted dysentery at the age of 20 and seemed to have recovered, was seized by strong pains in the right hypochondrium accompanied by fever, bilious vomiting and jaundice. Palpation allows to recognize at the right edge of the epigastrium hard and rounded tumefaction, from which an exploration puncture aspires pus. A few days later, the feces are covered by pus; then, fever drops and disappears and the patient recovers completely.

Recovery is exceptional when no intervention is made; but currently intervention occurs in almost all cases, due to surgery. Even in these cases, death can occur because of septicemia, complications (peritonitis, purulent pleurisy), hepatic insufficiency or following a prolonged aspiration.

Semiology – The diagnosis of hepatic abscesses relies on the existence of remittent fever, with pain in the right hypochondrium and liver tumefaction, which follow dysentery or ulcerous and purulent lesions of the digestive tube or the pelvic organs.

When the purulent collection is visible through the abdominal wall, an exploratory puncture can be performed to extract pus.

Sometimes, friction is felt as a result of peri-hepatitis which occurs in the focus.

Other times, accidents are rare and diagnosis is achieved by exclusion.

Purulent hepatopathy can be easily mistaken for sub-diaphragm abscesses.

Other disease such as cirrhosis, lymphomatosis and amyloid degeneration of the liver, hepatic tumours (sarcoma, epithelioma), serous cysts and non purulent hydatid cysts distinguish themselves through the absence of fever and a distinct evolution. Purulent hydatid cysts and the suppuration of bile ducts are also different due to their evolution, as well as the absence of the etiologic conditions of hepatic abscesses.

In order to determine the localization of the purulent bag, we rely on the presence of the circumscribed curve of the abdominal wall, a pain limited to one point of the liver, a slight perihepatic friction, persistent cough which indicate a convexity abscess; jaundice and frequent vomiting are signals for an abscess of the lower liver surface. But the result of an exploratory puncture, which can be repeated, allows to establish the precise localization of the abscess.

The prognosis of this disease is among the most serious, because of the functional importance of the liver which is partially destroyed and the difficulty to evacuate the pus. Abscesses which open into the bronchi,

the pleura, the digestive tube have changes of healing; those draining in the peritoneum or the pericardium are more dangerous. Multiple abscesses are very serious.

Prophylaxis and treatment – The prophylaxis of venous hepatic suppuration is dictated by the etiologic conditions of this disease. In the presence of dysentery and a purulent or ulcerous lesion in the portal vein, the physician should resort to purgatives, washings in order to clear the intestine from feces and to avoid the absorption of pyogenic microbes; the use of calomel, charcoal, bismuth salicylate, lactic acid, benzonaphthol is recommended to prevent hepatic suppuration.

The treatment consists of quinine (1–2 g) during the formation of pus and morphine injections to calm down pains.

When the pus bag is formed, surgery is necessary, since we cannot rely, as we did in the past, on the disappearance of a hepatic abscess or on its spontaneous opening.

Sometimes, a simple aspiration puncture can be enough. For instance, a 23 year old young man who contracted dysentery in Vietnam presented a hepatic abscess a few months later. We performed a puncture and extracted over one hundred ml of pus; the fever dropped and the patient, now healed, left the hospital after 2 weeks.

Surgery, often efficient in the case of a single abscess, provides little help in the case of multiple abscesses.

Gangrenous hepatopathies

The agents of gangrene, as those of suppurations, generate two types of hepatopathies: some are related to the poisoning of the body with microbial toxins, others are caused by the presence of the agents in the liver parenchyma.

The former hepatopahties, those generated by the gangrene of an organ, consist in an alteration of glandular cells, which become tumid and unclear.

They manifest through violent fever episodes, followed by mild jaundice and multiple hemorrhages.

The diagnosis, which is very difficult to set, relies on the existence of a gangrene focus; the prognosis for the evolution of the disease is among the most serious.

The treatment is similar to that of gangrene (vol. I, pag. 708).

Gangrenous hepatopathies resulting from the presence of pathogenic agents in the liver parenchyma occur after the arrival of microbes on three paths: the arterial path, the venous path and the biliary path.

Sometimes, they originate in a neighboring gangrene focus.

A – Arterial gangrenous hepatopathies

Etiology and pathogenicity – These hepatopathies are particularly noticed following gangrenous lesions of the bronchi or lungs. The microbes coming from here enter the pulmonary veins and are carried by the blood to the hepatic artery, where they stop.

Sometimes, their point of origin is another organ (mouth, skin) and gangrenous agents must cross the pulmonary capillaries before reaching the liver.

Pathologic anatomy – The liver is tumid and soft; usually, it becomes the location of several gangrenous foci, greenish or black, the size of a pea, an olive or, more seldom, a nut; these foci eliminate a bloody and fetid liquid. In a personal case, in a man suffering from gangrene of the bronchi, we found in the liver three gangrenous abscesses, out of which the largest was the size of a peanut.

The tissue surrounding this abscess is degraded and greenish on several millimeters. It contains leucocytes, altered blood, anaerobic microbes (gangrene agents) and aerobic agents (suppuration agents).

The hepatic cells are infiltrated with protein and fat granulations. The hepatic artery, the portal vein and the bile ducts are intact. The other organs are sometimes the location of similar metastatic abscesses; the lungs usually contain the initial focus of the gangrene.

Symptomatology – Clinical accidents start with intense fever, shivers and perspiration. The liver is tumid and painful when pressed; the spleen also has increased volume.

The patient starts to look typhic; he is agitated, has episodes of delirium, prostration and rapidly succumbs. The evolution of this disease is acute and the ending is fatal.

Semiology and treatment – Diagnosis considers the presences of a gangrenous focus in the lungs.
The prognosis is serious.
The treatment, that of gangrene (vol. I, pag. 708), is little effective.

B – Venous gangrenous hepatopathies

These hepatopathies occur during gangrenous infections of the digestive tube and particularly dysentery, where there are coincidences between the gangrene of the intestine and that of the liver (DUTROULAU).

The pathogen agent is carried to the liver through the blood of the portal vein.

The lesions consist of one or several gangrenous foci, generally large, with weakened walls and fetid content, containing anaerobic microbes.

The symptomatology, semiology and treatment of this disease do not differ from those of gangrenous hepatopathies of arterial origin.

Biliary gangrenous hepatopathies are little known and generally confounded with the suppurations of the same origin.

In some cases, gangrenous hepatopathies derive from a neighboring lesion. In a patient suffering from chronic dysentery, the liver presented a vast purulent and gangrenous focus, which communicated with the colon through a narrow perforation (EON).

Septic hepatopathy
(Syn: *Liver emphysema)*

Liver emphysema is characterized by the presence of gas bubbles disseminated in the liver parenchyma. It is visible after puerperal condition, traumatic erysipelas, biliary infections, smallpox and hemorrhagic scarlet fever etc.

I saw it several times, but only during summer time, coexisting with rapid putrefaction. But we accept, until better data, that hepatic emphysema could be caused by the *septic vibrio* which entered the blood.

The liver, tumid and discolored, creates a sensation of gas crepitation and even a sonorous sound upon percussion. When cut, a greenish liquid containing numerous gas bubbles flows; it is striated with small excavations and has a sponge-like aspect. Glandular cells are very altered; the parenchyma is full of microbes, mostly anaerobic.

The clinical manifestations of this disease are unknown, since they occur only when the patient dies, mostly during agony.

§ 6.

Syphilis spirochete generates two types of hepatic disorders: a toxic one, which occurs during the secondary stage of the disease, another which pertains to the presence of the pathogenic microorganism in the liver parenchyma, in the third stage of syphilis.

I. Toxic syphilis hepatopathy
(Syn.: *Syphilitic jaundice*)

This disorder manifests through jaundice which occurs in secondary syphilis, just like albuminuria, and these two accidents are the effect of the same process, namely poisoning of the body with spirochete remains. In fact, it often coexists with a febrile condition, exanthematic crisis and simultaneous tumefaction of the liver and spleen.

Hepatic lesions are little known, given that most of the time, this form of jaundice heals without leaving any trace. In some lethal cases, the liver was found soft, lax and yellow, with alterations of the glandular cells (tumefaction, fat degeneraton).

Patients are seized by a state of weakness, pains in the limbs, headache, lack of appetite, insomnia and fever. Five to six days later, jaundice appears and manifests first at the sclerotic membranes and then generalizes keeping a rather pale hue which can mask some erythematous and macular syphilides.

The temperature, sometimes normal, often rises to 38° or 39°; the pulse is fast when there is fever. The urine, little abundant, sometimes albuminoidal, is dark and contains biliary pigments. The subdural condition of digestive paths is accompanied by vomiting and diarrhea. The feces are yellowish, grey or discolored.

Abdominal examination reveals that the liver has increased volume, exceeding by at least two finger widths the costal margin; the spleen is also tumid.

For instance, in a young woman who had clear erysipelas, mild jaundice installed with discoloration of the feces and the presence of bile elements in the urine, spleen hypertrophy, intermittent fever which prolonged to over 3 weeks.

One or two months later, as happened in our case, sometimes after a longer interval, these phenomena diminish and disappear.

In some cases, we noted accidents of hepatic insufficiency that led to death.

It is easy to diagnose this form of jaundice, considering its occurrence in secondary syphilis and its evolution.

The prognosis is benign, most cases ending in complete recovery.

Medication with mercury and iodine is ineffective in the case of this form of jaundice, as in the case of albuminuria, and the treatment of these accidents requires a milk-based diet and cold baths or lotions.

II. Third syphilitic hepatopathy
(Syn.: *Syphilitic cirrhosis)*

This disease, well known due to the works of VIRCHOW, FRERICHS and one of us[10] – is one of the most well-known visceral manifestations of syphilis.

Etiology and pathogenicity – The cause of this hepatopathy is SCHAUDIN spirochete. The causes that predispose to it are trauma and excesses of food and drinks.

Pathologic anatomy – The agent of syphilis moves through the lymphatic system and the lesions of this disease develop around the arterioles, i.e. on the trajectory of lymphatic vessels. These lesions consist of the creation of a young conjunctive tissue which sometimes turns into an adult fibrous tissue, other times suffers a specific degeneration (vol. I, pag. 737). Hence, two varieties of syphilitic hepatopathies: a diffuse one and a gummous one.

[10] E. LANCEREAUX, *Traité de la syphilis,* Paris, 1866, ediţia a 2a, 1873, *Traité des maladies du foie et du pancréas,* Paris, 1899, pag. 378.

The lesions of the diffuse form are characterized, at the surface of the liver, by deep reeks which circumscribe unequal islands of hepatic substance, with the diameter of 3, 5, 7 cm and even more; this gives the exterior of the gland the aspect of intestinal circumvolutions. When dissected, fibrous ribbons are visible, which start from the depth of these reeks and spread into the depth of the hepatic tissue, limiting portions of the parenchyma, with variable width, similar to the islands at the external surface[11].

The liver sticks to the neighboring parts through fibrous peritonitis adherences, which appear in the reeks. The glandular cells are generally intact; sometimes, they are affected by amyloid degeneration.

The gummous form is specific through the presence at the surface of the liver and in the hepatic parenchyma of dry yellow nodules which, when resorbed, lose substance and acquire deep irregular depressions. These gums, softer or harder in the middle and dried at the edge, are surrounded by an area of grey fibrous tissue. They are round, the size of a pea, green bean, cherry or nut. They are generally multiple, isolated, grouped or confounded with masses of variable sizes.

The gummuous formations, made up of a conjunctive proliferation around the arterioles, namely in the portal spaces, suffer in the middle the diastasis action of the spirochete and liquefy; at the same time, the peripheral gum tissue goes into fibrous state and forms some sort of scar which covers the empty space resulting from the disappearance of the liquefied part. Thus, deep stellar depressions are formed at the surface and inside the liver and losses of substance which sometimes touch the left lobe and sometimes the right lobe of the gland which is reduced to the condition of fibrous stub.

These deformations are completed by the compensatory hypertrophy of the liver parts remained untouched.

The hepatic arteries, the branches of the portal vein and the bile ducts are normal; but when gummous formations appear around them, their caliber is narrowed and sometimes obliterated.

Syphilitic liver alteration is usually associated with the lesions of the same nature of teguments and organs (vol. I, pag. 739). The spleen is tumid; the hepatic lymphatic ganglions have increased volume; the digestive tube is the location of passive congestion, every time there is a blood stasis in the portal vein; the perihepatic peritoneum has false membranes and adherences which, in some cases, compress the gland up to the point when it generates ascites. Sometimes, peritonitis is generalized[12] and leads to the interconnected adherence of intestinal segments.

Symptomatology – The clinical manifestations of third syphilitic hepatopathy are sometimes so insignificant, that this disease goes unnoticed during lifetime.

Other times, the digestive disorders are the only apparent disorder. Patients complain of lack of appetite; they have slow digestion, with eructation, nausea, vomiting and diarrhea. Then meteorism occurs and patients keep losing weight. When the lesion is extended, these phenomena are completed by anemia and, finally, marasmus.

But generally patients feel a pain, more or less intense, in the right hypochondrium, which relates to perihepatitis. This pain manifests either through a sensation of weight, indisposition, or strong suffering, continuous and exacerbated by the movements of the thorax and irradiating towards the shoulder.

Another symptom noted in almost half of the personal cases is ascites, accompanied by dilation of subcutaneous veins of the upper abdominal parts. It usually develops slowly and progressively; sometimes, it grows considerably in only a few days. Very seldom it relates to circulation discomfort in intrahepatic capillaries; most often, it is caused by the compression of the trunk of the portal vein by gums or of the liver cover by false peritoneal membranes.

A more rarely noted phenomenon is jaundice, which is also the effect of the compression of bile ducts by a gum, by the ganglions of the liver hilum, by the false peritoneal membranes. It manifests through a slow evolution, for a prolonged period, of high intensity.

Other times, hemorrhages (epistaxis, hemoptysis, hematemesis, melena, purpura) appear during this disease which are often independent of jaundice.

The patient's examination reveals that the liver, first increased in volume, diminishes completely or partially. The atrophy affects both the left lobe, which becomes unperceivable upon percussion, and the right

[11] E. LANCEREAUX, *Atlas d'Anat. path.,* Paris, 1870, pag. 55, f. 1 and 2.
[12] E. LANCEREAUX, *Traité d'Anat. path.,* 1871–1881, pag. 311.

lobe, which, in the right hypochondrium, has an obvious sonority, as we noticed in a personal case. The volume decrease is little visible if the liver becomes the location of amyloid degeneration.

Palpation reveals in the liver a tough, unequal, irregular mass, made of lumps separated by deep depressions, limited by an irregular edge. This deformation of the organ is specific to syphilis.

Due to the adherences, the liver is immobilized and prevented from sliding on the abdominal wall during breathing movements.

Here is a typical example of syphilitic cirrhosis:

A 44 year old woman, who had two false pregnancies and a dead infant with syphilitic lesions, realized, at 36, that the volume of her abdomen was growing and that she had vague pains in the right hypochondrium. She came to the hospital weakened with meteorism and ascites. When we examined her, we discovered that the liver, large, presented lumps separated by deep depressions. The spleen had increased volume. The urine, with the density of 1.022, did not contain any abnormal elements. Subject to intensive treatment, her condition rapidly improved, she left the hospital and did not present any other symptoms for 6 years.

After this period, she was seized by repeated hematemesis, meteorism and ascites and finally succumbed. The necropsy reveals a shredded liver, with irregular lumps and deep channels. The esophagus had dilated veins, one of them ulcerated, 2 cm from the heart, and an orifice the size of a lentil pea through which blood was draining.

The evolution of this hepatopathy is slow and progressive and it spreads onto several years. Usually it ends up in recovery, which can be the effect of treatment, but sometimes it occurs spontaneously, as we noted in many cases during the necropsy. The fatal ending, relatively rare, is caused by hemorrhages and a progressive state of marasmus, amyloid degeneration of the organs, complications such as erysipelas, pneumonia etc.

Semiology – It is often difficult to diagnose this disease especially in the beginning, when there is no liver deformation.

In a more advanced stage, the irregularity of the surface and the anterior edge of the gland, the tumefaction of the spleen, metorism, ascites, hemorrhages, anemia, albuminuria and patient's weight loss which coexist or not with traces of syphilis in other organs allow us to confirm the nature of the hepatopathy.

Syphilitic cirrhosis can be mistaken for alcoholic and paludic cirrhosis, from which it differs through liver deformation, the symptoms it presents and its evolution; it can also be mistaken for tuberculous peritonitis from which it differs through the absence of abdominal pains, vomiting, diarrhea and tuberculous alterations of the lungs, pleura etc. The hydatid liver cyst is characterized by a transparent, non albuminoidal liquid, which contains hooks and which is extracted through an exploratory puncture. Hepatic epithelioma is differentiated due to the manifestation of jaundice accompanied by ascites, fast degradation and a progressive and rapid evolution.

The prognosis is not very serious, since this disorder, when well treated, heals in almost all cases. It is unsettling when the liver is destroyed by a gummous drain or becomes amyloid.

Prophylaxis and treatment – The prophylaxis and treatment of this form of hepatopathy are those of syphilis (vol. I, pag. 772), plus a diet which shall rule out any wine, alcohol and spices and good hygiene, exercise in the open, hydrotherapy.

III. Hereditary syphilitic hepatitis

This type of hepatopathy starts with the fourth months of intrauterine life. It starts in the fetus, which often dies between the seventh and the ninth month and it is also present in the new born. It is more rare in children and exceptional in teenagers and adults.

Hereditary syphilitic hepatitis has, similar to acquired hepatitis, two forms: diffuse and gummous.

The diffuse form translates into the increase of volume without liver deformation, whose surface is smooth and nongranular, firm consistency and yellowish grey color. The section surface has the same aspect as the exterior surface; sometimes, it is dotted with small smooth white stains. Histologically, this form of hepatitis is characterized by conjunctive proliferation which is very abundant especially at the level of the above-mentioned white stains. The lesion is diffuse and unequally distributed in the various regions of the gland.

The gummous form, mostly associated with the diffuse form, is characterized by white stains spread on the liver (like wheat grains) around the arterioles. These small gums can reach the size of a peanut; they are circumscribed by a grey fibrous region and their central part has the tendency to degenerate and liquefy. Glandular cells, compressed occasionally by the conjunctive proliferation, are often affected by fat-granular degeneration.

The spleen and the lymphatic ganglions are tumid; the lungs, testicles and especially bones display syphilitic lesions; the peritoneum contains a bloody liquid.

The symptoms of this hepatopathy go unnoticed within the multiple manifestations of the general disease. Usually, they consist of nasal, umbilical or oral, intestinal, cutaneous hemorrhage; we noted such hemorrhages in 12 out of 14 personal cases. The abdomen, large and meteorised, presents a certain dilation of tegument veins and generally mild ascites. The liver and the spleen are tumid, hardened, with smooth and irregular surface. Jaundice is rare.

The general condition soon worsens; the child loses appetite, has slow digestion, loses weight and becomes anemic; the features are withdrawn, eyes sink into orbits, the complexion is whitish and dull; then he has crises of vomiting and diarrhea, often green in new born babies, which exhaust it; it becomes skeletal and succumbs to marasmus.

This disease evaluates all the more rapid as the child is younger. However, if well treated, it can often heal.

The diagnosis is not difficult and the prognosis is generally serious.

The prophylaxis and treatment are those for hereditary syphilis (vol. I, pag. 781).

Tuberculous hepatopathy

Etiology and pathogenicity – This disease, which is more commonly met in children than in adults, is mainly caused by the bacillus of KOCH and malnutrition is its main predisposition.

The bacillus can reach the liver on two paths: arterial or venous. In the former case, the starting point of the infection is the lung; in the latter, the intestine and the mesenteric ganglions.

Pathologic anatomy – In its periarterial form, the liver has increased volume and the weight fluctuates between 1500 and 2500 g. Sometimes, it has a fat or cirrhotic aspect, in the case of simultaneous poisoning with alcohol and wine. Moreover, at the surface and deep in the parenchyma, we find grey or yellowish miliary granulations, often clustered on the trajectory of arterial vessels.

The venous form, which usually follows intestinal ulcers, is more specific to childhood; it consists of tumid liver with spread yellowish nodules the size of a pea or peanut. They are sometimes empty; they have worn edges, yellow color and their cavity often contains a liquid similar to pus, impregnated with bile.

This form of hepatopathy is sometimes complicated by tuberculous perihepatitis, consisting of fake membranes and adherences filled with miliary granulations and caseous masses.

Symptomatology – Arterial tuberculous hepatopathy does not have any special symptoms and usually goes unnoticed during lifetime – liver tumefaction could reflect hepatic adiposis – meteorism, digestive disorders, vomiting, diarrhea, ascites, which can be attributed to tuberculosis of the intestine or peritoneum.

Venous hepatopathy does not have any specific signs, either. But if a tubercle flows into a biliary canaliculus, the pains are similar to those caused by hepatic cramps.

General health is usually highly altered; there is fever, fast and progressive weight loss with alteration of the features and dark color of the skin.

The evolution of this disease is slow and the ending, lethal, is generally caused by pulmonary and intestinal disorders.

Semiology and treatment – It is difficult to diagnose hepatic tuberculosis. The painful crises that occur when a tubercle opens into a bile duct can be taken as cramps caused by calculi; it is different from it due to the regular absence of jaundice, the frequent recurrence of pain and a mild febrile movement.

The prognosis is serious, since in most cases, the ending is fatal.

The treatment is the same as for tuberculosis (vol. I, pag. 823).

Leprosy hepatopathy

The liver is rarely the location of leper lesions, which consist of nodules located on the trajectory of arterioles.

The hardly felt symptoms are anorexia, meteorism and sometimes ascites and jaundice. This disease has a slow and progressive evolution and usually lasts for several years.

Diagnosis is very difficult and the prognosis, serious.

The treatment is similar to that for leprosy (vol. I, pag. 838).