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Perspective

Lobulation Caused by Coarse Fibrosis

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DESCRIPTION

The liver is divided into several lobules to suggest a slight similarity to the situation seen in some animals. This lobulation is more obvious on the surface of the bigger right lobe than on the left lobe, and it differs from the foetal lobulation of the kidneys. Upto 16 lobules have been discovered. While it is important to separate these lobules from pons cirrhosis abnormalities and hobnails as well as from cicatrices that are clearly syphilitic, it is likely that this lobulation is caused by pathological conditions rather than being a morphological characteristic. Syphilis, prenatal peritonitis, or even tuberculosis could be to blame. The alteration is likely caused by syphilitic hepatitis of congenital origin in many cases, but there is frequently a lack of conclusive evidence to support this. It is likely that peritonitis during foetal life altered or interfered with the growth of the organ, resulting in fissuring and lobulation of the organ's surface, in cases when lobulation of the liver is accompanied with peritoneal adhesions.

A small flap of hepatic tissue may occasionally be all that is seen when the left lobe is shrinking. In these circumstances, the falciform ligament emerges from the left edge of the liver and the left lateral ligament is correspondingly weak. Due to this anomalous exposure of the stomach, the entire gallbladder is visible from the front and protrudes from the liver's left lateral edge. The gallbladder may be so dislocated as to lie with its long axis in the transverse axis of the body as a result of the disrupted relationship of the lobes. Rarely is there a particularly obvious cause for the left lobe's atrophic state, though it has been proposed that it may be because of foetal or early-life circulation disturbances. Other congenital problems may be linked to the alteration. In a case described by Caned, severe left lobe dwarfism was linked to lobulation of the right hepatic lobe and the existence of two rather than three aortic valves. In some cases, the left lobe atrophy may be brought on by syphilis or other pathological alterations that develop later in life.

Small liver material protrusions, around the size of the forefinger's terminal phalanx and mimicking the caudate lobe in miniature, are quite frequent and have no pathogenic or clinical relevance. Their typical location is close to the portal and longitudinal fissures on the underside of the right lobe. They may develop "accessory livers" if they are noticeably pedunculated. There are situations when the spigelian lobe is oddly pedunculated. A cast of a jushinculated lobe joined to the left border of the left lobe with a hepatic tissue pedicle can be found in the Cambridge Anatomical Museum. In a female infant aged fourteen weeks, scientists described an aberrant process emanating from the left lobe of the liver in conjunction with the lack of the inferior vena cava.

A peritoneal ligament connects the accessory liver to the main organ as a result of the liver cells in the pedicles of the tiny accessory lobes, which are frequently visible on the underside of the liver, atrophying. Accessory livers can also develop in this way. It appears possible that some detached lobes observed in adults may be caused by pressure effects or the atrophy of some liver tissue as a result of blood supply interference. Under the heading of liver abnormalities caused by tight lacing, constriction lobes attached to the lower extremity of the right or, less frequently, the left lobe are reported.

A pedunculated lobe may develop atrophy at the base of the Spigelian or Iwo-sibly of the frontal lobe. A spigelian lobe with peduneules was also described. It should be noted that although the liver cells have vanished, the remnants of a rudimentary lobe may be detected in the left lateral ligament's peritoneal layers along with hepatic and portal arteries.

Dark purple stains are frequently visible on the liver's surface where it has come into touch with the stomach or colon. The action of gases, including sulphuretted hydrogen, which seep through from the colon and stomach and meet with iron in the liver causes these stains, which are created after death and are fairly superficial. As a result, a monte compound like sulphide of iron is produced. When a fever or other infection is present, the surface of the liver develops irregular white patches that are a sign of congestion and degenerative changes.

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