Primary Biliary Chirrosis: a Quantum Biophysical Semeiotic sign simple realiable in quick diagnosis even of its Inherited Real Risk

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"Primary Biliary Cirrhosis (PBC) is an autoimmune liver disease characterized by highly specific serum anti-mitochondrial antibody (AMA) and progressive destruction of the intrahepatic bile ducts resulting in chronic cholestasis, portal inflammation, and fibrosis that may lead to cirrhosis and ultimately liver failure. The disease predominantly affects women typically diagnosed in their fifth and sixth decade although younger patients have been described including rare paediatric cases. The loss of bile ducts leads to intrahepatic retention of detergent bile acids, resulting in liver damage through interaction with cell membranes and organelles. The derangement of the entero-hepatic bile acid circulation is likely the cause of other pathophysiological changes, which contribute to the extra-hepatic manifestations of the disease. (1).

The clinical features and natural history of PBC vary significantly among individual patients ranging from asymptomatic and stable or only slowly progressive to symptomatic and rapidly progressive. The typical clinical presentation has changed during the last few decades as the natural history has been modified by the recognition of earlier more indolent cases and the use of ursodeoxycholic acid (UDCA).

The autoimmune basis of PBC is supported by the highly specific anti-mitochondrial antibodies (AMA) and autoreactive T cells, the former being the basis for CURRENT diagnosis in the vast majority of cases

In following, I descirbe a simple, realiable Quantum Biophysical Semeiotic sign, which allows physician to bed-side diagnose the PBC since its first stage, Inherited Real Risk, namely from birth.

In healthy, intense manual pressure (1,000 dyne/cm.²), applied on liver projection area, brings about **immediately** rapid dilation of the gall bladder of **3 cm** of intensity (2, 3).

On the contrary, in PBC, starting from its Inherited Real Risk, the dilation of the gallbladder occurs after a latency time **greater than 5 sec**. Dilation is slow and **less than 2 cm**.

References

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