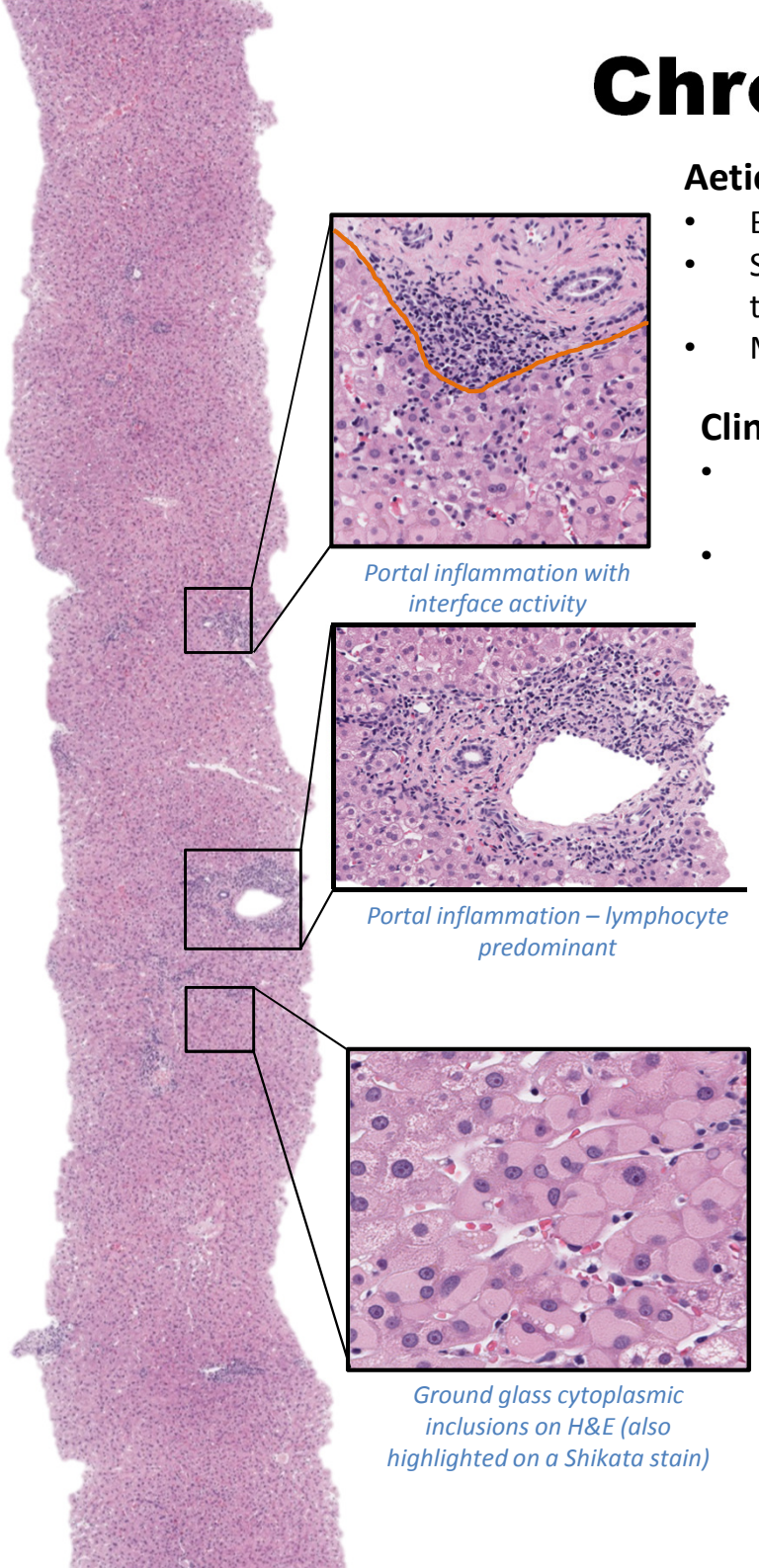
Chronic Hepatitis B



Portal inflammation with interface activity

Portal inflammation – lymphocyte predominant

Ground glass cytoplasmic inclusions on H&E (also highlighted on a Shikata stain)

Aetiology:

- Blood borne DNA virus
- Spread by sexual contact, intravenous drug abuse, contaminated blood, or vertical transmission from mother to infant
- May have hepatitis D superinfection

Clinical/biochemical picture:

- Often subclinical, but can present with acute liver failure, chronic liver disease & cirrhosis
- Hepatitic picture – elevated ALT

Liver Biopsy:

- Biopsy often performed to determine fibrosis or disease activity (grading & staging), and to determine treatment options

Histology:

- Hepatitic pattern of varying severity – ranges from minimal inflammation, to marked portal and lobular inflammation with areas of necrosis.
- Commonly: Portal inflammation, predominantly lymphocytic, with associated interface activity. May show scattered acidophil bodies & parenchymal inflammatory foci. Confluent necrosis suggests a flare or coinfection.
- Often show ground glass hepatocytes (but not always) – can be highlighted on Shikata Orcein/Victoria Blue special stains or Immunohistochemistry for HBsAg.
- With time, increasing portal inflammation, leading to fibrous septae and cirrhosis.

Clinical course:

- 30% develop cirrhosis in 30 years
- Risk factor for development of HCC (whether cirrhotic or not)
- Can be managed with antiretroviral therapy to suppress viral replication, but currently cannot be cured