aetiology

- Viral (HBV, HCV)
- Alcohol
- Metabolic
- Autoimmune
- Genetic

1. persisting parenchymal injury

2. perpetuation of injury (ex. hypoxia)

3. chronic cell & tissue injury
   → HC death by necrosis or apoptosis

4. perpetuation of injury

5. inadequate hyperplastic proliferation of HC

6. activation of MF-like cells

7. pro-inflammatory mediators

- increased deposition of ECM and ECM altered remodelling

GFs, CKs and mediator’s pattern favouring tissue repair and ECM producing cells