DE QUERVAIN THYROIDITIS
(GRANULOMATOUS THYROIDITIS)

- It was first described by Dr.Fritz de Quervain (1868-1940) swiss surgeon
- De quervain thyroiditis is virus triggered, immune mediated destruction of thyroid follicles associated with HLA genetic predisposition
- also called Granulomatous thyroiditis
- occurs much less frequently than does Hashimoto thyroiditis
- Age - common between 40 and 50 years
- Sex- more commonly affects females (F:M ratio of 4:1)

Pathogenesis

- Viral infection provides an antigen, which is either viral or a thyroid antigen that is released secondary to virus induced host tissue damage
- Exact pathogenesis is not clear
- Genetic predisposition is essential and is associated with HLA B35 and HLA B15/62
- Process is preceded by viral infections like
  - Mumps
  - Adenovirus
  - Epstein Barr virus
  - Coxsachie virus
  - Cytomegalo virus
  - Influenza
  - Echo virus
  - Enterovirus
- Majority of the patients have history of respiratory tract infection
- When virus enters the thyroid, macrophages and dendritic cells engulf the virus
- Viral proteins are processed, bind to HLA B35 and present to cytotoxic T cells to induce death of infected cells
- This results in rupture of thyroid follicular epithelial cells and release of thyroid hormones T3, T4 and specifically thyroglobulin

- **Activated macrophages also release cytokines which attract CD4+ T cells and these cells further differentiate and stimulate B cells to produce auto antibodies which further leads to destruction of follicular epithelial cells**

H/O Upper respiratory tract infection

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Virus enters into thyroid and infect thyroid follicular epithelial cells

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Viral particles are ingested by macrophages and dendritic cell

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Viral antigens are presented to cytotoxic T cell via HLA B35 on activated macrophages and dendritic cells

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Cytotoxic T cells destroy the infected follicular epithelial cells and release self antigen

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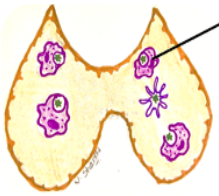
Release of T3, T4 and thyroglobulin



Activated macrophages release cytokines

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Activate CD4+ T cells which proliferate, differentiate and further activate B cells which produce antibodies to self antigens and viral antigens

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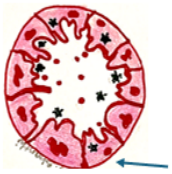
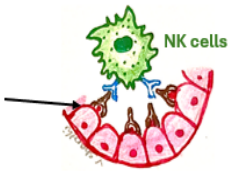
Further destruction of thyroid follicles by NK cells

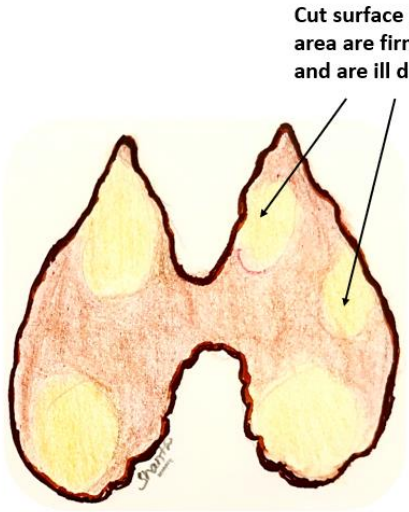


Thyroid follicular epithelial cell destruction



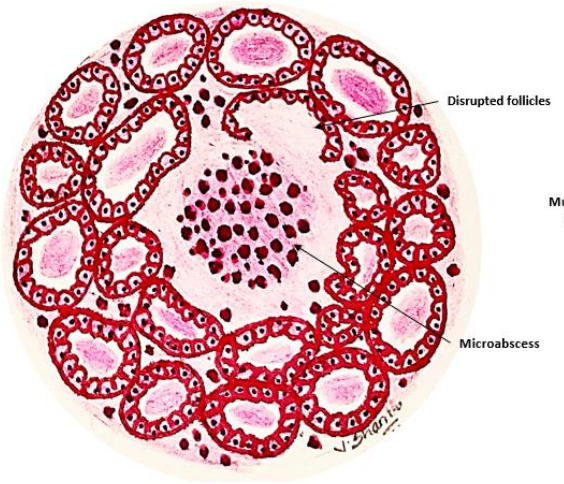
Morphology

- **Gland may be unilaterally or bilaterally enlarged and firm, with an intact capsule that may adhere to surrounding structures**
- **On cut section, the involved areas are firm and yellow-white and stand out from adjacent normal rubbery, brown thyroid gland**



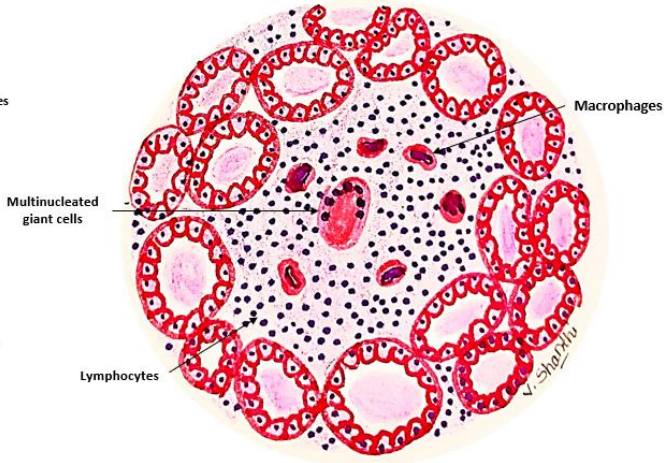
MICROSCOPY

Early in the active inflammatory phase

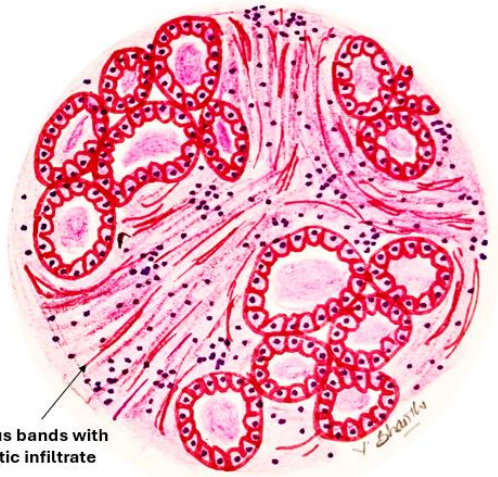


Scattered follicles may be disrupted and replaced by neutrophils forming microabscesses

Later phase



Characteristic features appear in the form of aggregates of lymphocytes, activated macrophages, multinucleated giant cells and plasma cells associated with collapsed and damaged thyroid follicles – Granulomatous thyroiditis



- In later stages of the disease, a chronic inflammatory infiltrate and fibrosis may replace the foci of injury
- Different histologic stages are sometimes found in the same gland, suggesting waves of destruction over a period of time

Clinical Features

- Pain and thyroid enlargement
- Immune response is virus-initiated and the process is limited
- Inflammation of the thyroid and hyperthyroidism are transient, usually diminishing in 2 to 6 weeks, even if the patient is not treated
- However, unlike in hyperthyroid states such as Graves disease, radioactive iodine uptake is diminished
- After recovery, generally in 6 to 8 weeks, normal thyroid function returns

- **Disease is seasonal, with occurrences peaking in the summer**

Disease progression

- **Thyrotoxic phase - Initially due to follicular cell damage and hormone leakage**
- **Hypothyroid phase – resulting from follicular cell destruction**
- **Recovery phase – regeneration of follicles and restoring thyroid function**