Immunohistology of the renal biopsy shows irregular,granular deposits of IgA, C3 and fibrin in the glomeruli. Deposits of IgA and C3 are also found in the skin, even in non-affected areas, and are diagnostic of the condition.

As in IgA nephropathy, the available evidence suggests an IgA dominant immune-complex pathogenesis with complement activation occurring via the alternative pathway. A variety of bacterial or viral antigens could be involved, as there is an association with preceding upper respiratory tract infection. In addition, HSN is a seasonal disease: most patients present during the winter. The clinical and immunological similarity between HSN and IgA nephropathy suggests that IgA nephropathy is a renal limited form of HSN.

Lecture No. 16

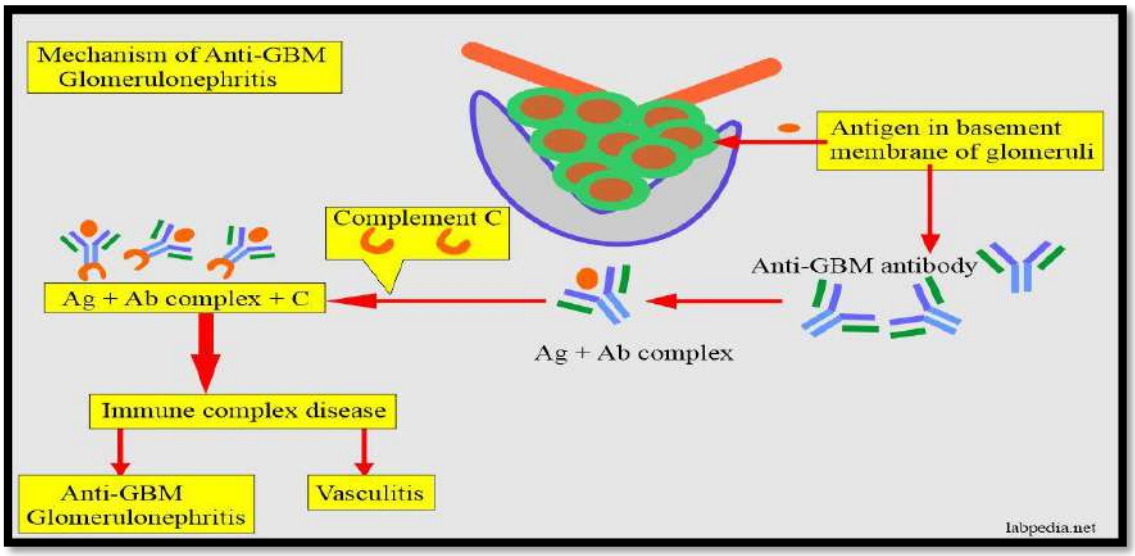
Anti-glomerular basement membrane disease

Acute glomerulonephritis mediated by anti-glomerular basement membrane (anti-GBM) antibody account for about 1-2% of all cases of glomerulonephritis. Anti-CBM nephritis is more common in men and in those who possess HLA-DR2. Patients present with nephritis alone or, more commonly, with glomerulonephritis and lung haemorrhage, a combination termed Goodpasture's syndrome. However, rapidly progressive nephritis and pulmonary haemorrhage can occur in other multisystem disorders such as SLE or Wegener's granulomatosis so the combination of renal and lung involvement is not synonymous with anti-GBM disease.

The target antigen is the α3 chain of type IV collagen, a major constituent of the GBM. Lung damage results from antibodies to antigens common to both alveolar and glomerular basement membranes. In Goodpasture's syndrome, respiratory symptoms often precede renal disease by 1 year or longer. Haemoptysis, usually leading to anemia, is a prominent feature and the sputum typically contains haemosiderin-laden macrophages. Lung biopsies show intra-alveolar haemorrhage and necrotizing alveolitis.

Etiology

Although the cause is unknown, anti-GBM disease follows upper respiratory tract infections in 20-60% 0f patients, or exposure to certain hydrocarbons. These agents may damage alveolar basement membrane, generating new and potent antigens able to stimulate autoantibody production. Alternatively, the agent responsible (e.g. a virus may cross-react with basement membrane antigens. Pulmonary haemorrhage in anti-GBM disease is strongly associated with cigarette smoking.



Figure(1):Mechanism of Anti-GBM glomerulonephritis

Diagnosis

1. **Renal involvement includes**
   - **a)** Gross or microscopic hematuria, proteinuria, a decreased 24-hour creatinine clearance, and elevated blood urea and serum creatinine levels.
   - **b)** Abnormally shaped RBCs and casts can be found in the urine sediment.
2. **In those patients with pulmonary involvement,** decreased total lung capacity and increased uptake of carbon monoxide is evident. An iron deficiency anemia with decreased hemoglobin and hematocrit can develop if pulmonary hemorrhage is severe.
3. The ESR and CRP level may be normal or increased.
4. Circulating antibodies to the GBM (anti - GBM) glomerular basement membrane can be detected in about 87% of patients. These antibodies can be identified by IIF, ELISA, or Western blot.

Lecture No. 17

Respiratory disease

Drug-Induced Pulmonary Disease

Drug-induced pulmonary disease is lung disease brought on by a bad reaction to a medicine. Pulmonary means related to the lungs.

What is the most common drug-induced respiratory problem?

Interstitial pneumonitis (ie, inflammation of the lung interstitium, such as the alveolar septa) is the most common manifestation of drug-induced lung disease.

The Common Types of Drug-induced Pulmonary Diseases