GENITOURINARY SYSTEM

SUBJECT : ملخص محاضرة 3

LEC NO. :

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GENITOURINARY SYSTEM

Nephritis GN = inflammation= hypercellularity = proliferation
Most important Clinical features of Niphritis
1. Hematourua 2. hypertintion 3.Azotimia 3. RPC cast 4. little proteinuria and edema
The first disease in nephritis
1. Acute Post infectious (Post streptococcal) Glomerulonephritis(PSGN)
هو عبارة عن مرض بجي ب الاطفال بعد الإصابة ب اي نوع بكتيريا خصوصا streptococcal ب ٤ اسابيع بعد ما يتشافو من التهاب حلق او جلد "nephritogenic" strains of β-hemolytic streptococcal infection.

Pathogenesis of Acute Post streptococcal GN: Is immune complex deposition, because the typical features of immune complex disease are seen, including,
(1) Granular deposits of IgG & complement on the GBM

In LM : 1.hypercellularity =proliferation of endothelial and mesangial cells and neutrophilic infiltrate
2. Diffuse and uniform /affect all glomerui
In EM / "subepithelial"humps"***""in GBM****.
In IF : granular deposits of IgG & complement

PSGN-Clinical Course
•Acute onset .
•Gross hematuria with smoky brown rather than bright red urine .
•Mild proteinuria.
•Serum complement levels are low during the active phase of the disease.
•↑serum anti-streptolysin O antibody titers.
•Recovery occurs in most children.
ملخص المرض :
بتجيك ام بتحكيلك ابني قبل قبل ٤ اسابيع صابة التهاب حلق او جلد وتشافي منة بعدين مرض وصار لون البول مثل ال smoky
لما تعمل فحوصات بطلع ف LM عندة hypercellularity in endothelial and mesangial cells و على EM بطلع عنده subepithelial"humps مهمة جدا
وبكون هاض المرض عند الاطفال

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Second disease in niphritic GN

IgA Nephropathy (Berger Disease):IgA nephropathy is one of the most common causes of recurrent microscopic or gross hematuria &is the most common G disease revealed by renal biopsies worldwide *very important عبارة مهمة جدا

*هاض المرض زي اللي قبلة بس الفرق انه اصابتة البكتيريا قبل يوم او يومين غالبا بجي في الاطفال بكون عندهم اما upper RTI, or, less commonly, GIT بعد الإصابة ب grossor or microscope hematuria

Pathogenesis of IgA nephropathy

هون نتيجة الإصابة ب بكتيريا او فايرس زاد عندنا IgA بشكل غير طبيعي بالتالي الجسم اعتبر انه الزيادة هاي اشي abnormal وبدأ بتكوين autoantibody ل IgA وصار complex وترسل ب mesangial cell

In normal condition there is low levels serum IgA But in this disease there is genetic influence then incres IgA synthesis in response to respiratory or GIT lead to deposition of IgA & IgA-containing immune complexes in the mesangium,

So pathogenesis : abnormality in IgA products and clearance

In LM : NORMAL or (mesangioproliferative)

IF: mesangial deposition of IgA with C3 التشخيص يعتمد على وجود هاض

EM: deposits in the mesangium

Some cases occur in patients with celiac disease.

3' disease in niphritic /Rapidly Progressive (Crescentic) Glomerulonephritis

characterized by rapid & progressive loss of renal function with features of the nephritic syndrome, often with severe oliguria & (if untreated) death from RF within weeks to months.

Three types / group A /(Anti-Glomerular BM Antibody): as Goodpasture syndrome(with renal & pulmonary involvemen

Group B (Immune Complex) as SLE And IgA nephropathy

Group C (Pauci-Immune): Antineutrophil cytoplasmic antibody (ANCA) Associated:

- Idiopathic
- Wegener granulomatosis
- Microscopic angiitis

هلي التقسيمة بشكل عام وخلينا نعرف معنى crescents هو اختفاء ال Bomans space نتيجة تكاثر ال parital membrane

في عنا common features لل هذول ال ٣ disease وهي petechial hemorrhages و segmental) GBM break و ()necrosis

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Group A : Anti-GBM Antibody Crescentic GN
Characterized by linear deposits of IgG & C3 along the GBM (which
can be seen by immunofluorescence M
Anti-GBM Abs are present in the serum of all patients& are helpful in their diagnosis & patients benefit from plasmapheresis or immunoadsorption,which removes
 The disease is either:
(I) Idiopathic Anti-GBM Ab GN cases, in which the anti-GBM Abs bind to renal GBM only, without pulmonary lesions
(II) II) Goodpasture syndrome cases of Anti-GBM Ab GN, against kidney and lung

Group B: Immune Complex-Mediated Crescentic GN
This can be a complication of any of the immune complex nephritis as SLE
In all these cases, immunofluorescence studies reveal the characteristic granular ("lumpy bumpy") or wire loop
These individuals cannot usually be helped by plasmapheresis

Group C: Pauci-Immune Crescentic GN
Defined by the lack of anti-GBM Abs or significant immune complex deposition detectable by immunofluorescence & EM.
 Most of these individuals have anti-neutrophil cytoplasmic Abs in the serum, which have a role in some vasculitis.
immunofluorescence shows NO immunoglobulin or complement, & NO EM detectable deposits.

There is no immune reaction in this type

Disease 4 in niphritic is Hereditary Nephritis
caused by mutations in GBM proteins as Alport syndrome in which nephritis is accompanied by nerve deafness & eye disorders, including lens dislocation & cataracts.
The disease is NOT immunologically mediated disease

Pathogenesis:Mutation of any one of the α chains of type IV collagen
EM
• GBM thin and attenuated.
• GBM later develops splitting and lamination "basket-weave" appearance
Clinically: The inheritance is heterogeneous, being most commonly Xlinkedas a result of mutation of the gene encoding α5 type IV collagen.
Males affected more frequently & more severely & are more likely to develop RF than females
overt RF occurs between 20 & 50 years of age.

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هاي الأمراض اللي هون عبارة عن خليط بين ال niphritic و ال niphrotic لكنها تميل للـ niphritc اكثر شوية
الدكتورة حكت جاي علية سوال هو شوية معقد بس انشالله رح الخص الموضوع

Membranoproliferative GN(MPGN): (MPGN) is a pattern of glomerular injury on kidney, including hypercellularity and thickening of the glomerular basement membrane (GBM).
MPGN is a histologic lesion and not a specific disease entity+ and there hypocomplementemic,
Essential features: 1/hypercellularity+ mesangial and subendothelial deposits and duplicating of glomerular basement membrane.
The tram track appearance is caused by "splitting" of the GBM due to the inclusion within it of processes of mesangial & inflammatory cells extending into the peripheral capillary loops.

There are 2 type :
MPGN / Most cases of type I .classical
1.discrete subendothelial and mesangial electron dense deposits immune complex deposition indicating activation of classic complement
2.Distinguished from new category of C3 glomerulopathies by prominent Ig or C1q
Primary MPGN mostly affecting adolescents and young adults
3.MPGN due to a monoclonal gammopathy or complement mediated disease with a higher risk of graft recurrence than immune complex mediated MPGN secondary to infection or autoimmune disease

Type II MPGN (dense-deposit disease/ *excessive complement active + intra membrane)

*Essential diagnostic feature based on the presence of highly electron dense ribbon-like or chunky deposits of the glomerular basement membrane
*autoantibody against C3 convertase called C3 nephritic factor
Can be acquired by infections or monoclonal paraprotein
*Poorer prognosis than type I