

Renal Block

Lecture Seven

Renal Allograft

Important notes & MCQS



Objectives:

- Recognize the concept of renal allograft.
- Describe the pathology of rejection
- Differentiate between acute and chronic rejection.
- Recognize the principal infections inherent to renal transplantation.
- Recognize acute and chronic drug toxicity.

Two major things that may kill the patient if the clinician says the opposite:

- Rejection
- Infection

<u>Why?</u> Because if we say that its rejection and we give more immunosuppressant and it turns out to be an infection the patient may die

- Most important transplantation that doesn't get rejected is <u>pregnancy</u>.
- o The most important thing in allografts is **HLA compatibility**.

Types of rejection:

Hyperacute:

The patient already has the antibodies circulating, after entering the kidney it becomes hemorrhagic, necrotic and polymorphonuclear cells infiltrate after a few hours and this isn't supposed to happen.

Hyperacute under the microscope (antibody mediated):

- 1- Necrosis
- 2- Hemorrhage
- 3- Polymorph nuclear cells infiltrate

Acute rejection:

Happens in days or two weeks and is two types T cell mediated (cellular mediated) and antibody mediated. They differ in treatment.

1. Acute T cell mediated:

- <u>Grade 1</u> will look like tubulointerstitial inflammation and if it got more severe the wall of the vessels will thicken and endothelialitis and is grade 2.
- If really severe inflammation lymphocytes infiltrate, thickening endothelialitis and fibrinoid necrosis type 3 may be t cells or antibody mediated.
- The T cell mediated has a better prognosis than the antibody mediated
- 2. Acute Antibody mediated rejection to diagnosis we need 3 criteria:

1- Cellular injury:

- May appear as dilatation of the peritubular capillaries and polymorph or lymphocytes within them.
- Another type is only acute tubular injury and necrosis no inflammation or anything else but is c4d positive or lastly blood vessels fibrinoid necrosis and inflammation and infiltration.

2- C4d positive in immunofluorescence.

3- Circulating antibodies:

We can find Circulating Donor specific antibody in the blood but not as high as in the hyperacute.

o Chronic:

- Fibrosis and sclerosis of blood vessels.
- Antibody or t cell mediated

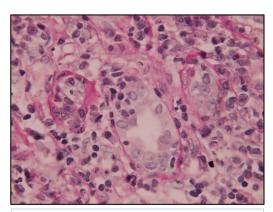
• All in t cell:

- Borderline changes will need clinical investigation (creatinine).
- Grade 1 A or 1 B: depending on the tubulointerstitial inflammation severity between both.
- Grade 2: endothelialitis we see a few lymphocytes beneath the endothelium and thick wall.
- Grade 3: may be t cell mediated or antibody mediates more severe than the rest

Chronic allograft nephropathy:

Fibrosis, arterial sclerosis, and scarring.

- → May be from rejection, drug toxicity (cyclosporine) or other things
- → We see double contouring in the glomeruli like membranoproliferative glomerulonephritis but membranoproliferative it is induced by immune complex but here it is because the circulating antibodies injured the capillary of the glomeruli they went to the endothelial cell and then recuperated and came back and made another capillary wall and made

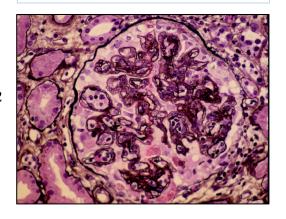


Tubulitis and lymphocytes (the black dots) and rupture of the tubular basement membrane.

transplant glomerulopathy.

Transplant glomerulopathy:

Chronic lesion more than a hundred days and 2 double contouring.



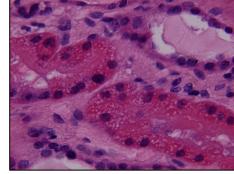
Chronic Allograft nephropathy:

In capillary we also see narrowing of the lumen and atherosclerosis like in hypertension to differentiate between them we do silver stain and if we see silver elastic multilayering it is hypertension if not then it is chronic allograft nephropathy.

Drug toxicity:

Cyclosporine toxicity:

- Acute we see the isometric vacuolization in the tubules.
- If chronic we see strip fibrosis or nodules in the wall of the blood vessels, hyaline nodules.



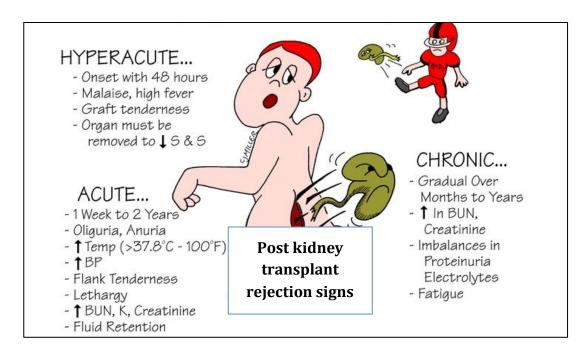
We see vacuolization and isometric due to acute drug toxicity.

Infections:

Most important infection:

- o **Polyoma** virus:
 - In chronic transplant because they are taking a lot of immunosuppressants.
 - We can see inclusion in the nuclei ground glass appearance and gray.
 - It is mainly in the tubule.
- Cytomegalovirus:
 - It makes the cells big and is in the glomeruli and nuclei (everywhere not specific).

<u>Note:</u> Any disease that leads to kidney transplant may return to the new kidney or even de novo (a new disease) may happen.



Now Check Your Understanding:

B. Acute rejection

C. Chronic rejection

D. Hyperacute rejection

1.	What is the most important transplantation that doesn't get rejected?
	A. Pregnancy
	B. Kidney
	C. Liver
	D. Heart
2.	A patient had recurrent kidney disease after his kidney transplantation. How correct is this
	scenario?
	A. Not correct at all
	B. Correct
3.	A patient was given immunosuppressant therapy after kidney transplantation. He died a week after
	the therapy started. What could have been the cause of his death?
	A. Rejection
	B. Infection
	C. All of the above
4.	A patient had an infection after his kidney transplant. Histological picture shows ground glass
	appearance, what infection could that be?
	A. TB
	B. Polyoma
	C. Cytomegalovirus
5.	A patient had an infection after his kidney transplant. Histological picture shows infiltration
	throughout the glomeruli, tubules and interstitium, what infection could that be?
	A. TB
	B. Polyoma
	C. Cytomegalovirus
6.	Which of the following causes antibody mediated endothelial damage?
	A. Normal kidneys

1. A	2. B	3. B	4. B	5. C	6. D

7. Vasculitis is associated with?

- A. Normal kidneys
- B. Acute rejection
- C. Chronic rejection
- D. Hyperacute rejection

8. A cyanotic kidney is a feature of?

- A. Normal kidneys
- B. Acute rejection
- C. Chronic rejection
- D. Hyperacute rejection

9. Which of the following is T-cell mediated?

- A. Normal kidneys
- B. Acute rejection
- C. A & B
- D. Hyperacute rejection

10. A patient had a successful renal transplant and in a year he bad routine check up that revealed progressively high serum creatinine levels. What could that be an indication of?

- A. Normal kidneys
- B. Acute rejection
- C. Chronic rejection
- D. Hyperacute rejection

11. We can see mild arteritis in:

- A. Grade IA
- B. Grade IB
- C. Grade IIB
- D. Grade III

12. We can see fibrinoid necrosis in:

- A. Grade IA
- B. Borderline changes
- C. Grade IIB
- D. Grade III

7. B	8. D	9. B	10. C	11. C	12. D

13	Where	can	WA SAA	doub	le contour?)
1.0	. wilete	Lan	we see	uvuv	ie contoui :	

- A. Chronic allograft nephropathy
- B. Amyloidosis
- C. DDD
- D. RPGN

14. Where can you see transmural arteritis?

- A. Grade IA
- B. Grade IB
- C. Grade IIB
- D. Grade III

15. How can you treat cyclosporine toxicity?

- A. Gastric lavage
- B. Reduce dose
- C. Alkalization of urine

16. What can you use in the treatment of grade II?

- A. Steroids
- B. Cyclosporine
- C. Statins

13. A	14. D	15. B	16. A

Contact us: Pathology435@gmail.com

Team Members

Nouf Altwaijri
Aljohara Almazroua
Amjad Alduhaish
Budoor Julaidan
Deema Alfaris
Kayan Kaaki
Khawla Alammari
Lamees Altamimi
Nojood Alhaidri
Noura AlBulushi
Noura AlKharraz
Nouf Alabdulkarim
Reem AlAqeel

Ahmad Taha Alkhiary

Ammar Saleh Almansour

Anas Baleegh Mohammad Ali

Faris Ibrahim Alwarhi

Hamzah Abdullah Alfiar

NaiF MoHaMMeD AlHaDi

Qusai Abdulbaqi Ajlan

Rayan Abdulrahman Almuneef

Zeyad Abdulaziz Alsalem

قال صلى الله عليه وسلم: (من سلك طريقًا يلتمس فيه علمًا سهّل الله له به طريقًا إلى الجنة). دعواتنا لكم بالتوفيق.