No. nephrotic syndrome

S4:-

* endothelial cells above LRI are fenestrated for filtration
* Podocytes have thousands of foot Processes which is Important to prevent proteinuria
* L=lumen & U=urinary space

S8:-

* Under arrows the slit diaphragm which lies between podocytes

S11:-

* NS is essentially massive proteinuria
* In the normal adult there is no protein in the urine or as little as 150-300 mg/24 hours more than that it is called (significant proteinuria)
* some NS patients have (massive proteinuria) without edema
* hypoalbuminemia is Variable due to activity of liver

S12:-

* patient could have pleural effusion

S13:-

* MCNS could present at any age
* Genes are not for memorizing

No.

S 14 & 15 :-

* face has loose bissues which is prone to edema at night but it is relieved at day time when patient ambulates
* anasarca is an extreme generalized edema

S16 :-

* these days we don't use the first two methods because it is hard to collect urine in pediatrics and it is invasive to use catheters
* in third method we use a random sample preferrably in the morning because with ambulation we tend to increase the amount of protein in the urine

S17 :-

* So it is important in treating edematous patients to correct oncotic pressure and give them albumin

S18 :-

* these are more in long standing patients
* factors B & D are responsible for opsonization particularly for encapsulated organisms so patient will be susceptible to them
* NS patients could present with infections as peritonitis or cellulitis or bacteremia also due to immunosuppressant
* hypecoagulability is more seignificant in Patients with Proteinuria (massive amounts) or membranous nephropathy who are unresponsive to therapy or have long standind disease

No.

S19:-

* In pure nephrotic patients without nephritis can have significant contraction in intravascular compartments due to hypoalbuminemia
* Vomiting or diarrhea can endanger kidneys to AKI

S20:-

* malaria could cause secondary NS
* MCGN is the most common type
* FSGS have two subtypes, primary which is more related to heritable disorders and causes and they can present at any age (even in infancy), and acquired (primary acquired) which needs nephritis-nephrosis sort of therapy and it presents suddenly
* MCGN have many types, could present in childhood or adulthood, they usually have a bad response to therapy, and they may start presenting as nephritis (hematuria, HTN, and decreased kidney function) or present with microscopic hematuria and nephrosis and developing into gross hematuria.
* membranous nephropathy is charectarized by MASSIVE proteinuria which makes these patients more prone to complications due to decreased protein

S24:-

* there is no need for biopsy especially if the urine analysis shows proteinuria without RBC's, normal BP, normal KFT & SCr, and responsive to treatment it is MCNS

No.

S25:-

* if RBC's or casts are seen in urine then we are dealing with nephritis not nephrosis (it could be nephitic nephrotic)
* protein normally should be zero (nill) but if it was (1+) it could be fine
* Protein > (2+) is usually considered in nephrotic range
* nephritis patients tend to have abnormal KFT
* CBC can show evidance of anemia or microangiopathy
* complements when nephritis is suspected
* increased lipids indicates long term nephrosis

S26:-

* RBC's in urine
* if there were RBC's casts then we are dealing with nephritis

S27:-

* lipid casts in urine -lipiduria- (does not have that significance)

S28:-

* HBsAg +ve disease can be associated with membranous nephropathy and we have to avoid steroid therapy for these patients

No.

S29:-

*in patients that seems not MCNS or in nephritic-nephrotic patients

*in some diseases (as SLE) each stage have a specific therapy

S30:-

*in relapse we dont need to wait for edema to occur to define it as relapse and start treatment it is sufficient to define it by increased protein

S33:-

*if albumin has been given alone it will drag fluids out of tissues to blood vissels which will increase BP and possibly lead to HF

*we usually avoid thiazides but it can be used with chronic nephrosis patients

*PCV-13 = pnemococcal conjugate vaccine

*they should take PCV-13 vaccine because they are susceptible to pneumococcal infection

S34:-

*oral steroids will be given every day for a month then tapered to be every other day for another month

*immunosupressive agents are used with steroid resistant patients or patients with frequent relapses

No.

S34 cont. :-

* Calcineurin inhibitors are used with transplantation
* monoclonal AB are expensive but efficient
* levamisole is used for the treatment of worms and sometimes for MCNS, it is cheap
* in nephritis-nephrosis ACEi & ARB's can decreas proteinuria

S36 :-

* more with patients with steroid toxicity, frequent relapses, or long term steroid therapy

S37 :-

* if after 1month the patient still not responsive so the chance to be MCNS < 2%

S39 :-

* levamisole is not used in other than MCNS