

# Renal Manifestations of Systemic Conditions

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# Disclosure

- Kim Squires has no conflicts to disclose
- Patti Ring has no conflicts to disclose

# Overview

- Systemic Lupus Erythematosus
- Diabetes Mellitus, Type 1 and 2
- Sickle Cell Anemia
- Prematurity
- Spina Bifida



**American Family Children's Hospital  
Madison Wisconsin**

# Systemic Lupus Erythematosus

- SLE affects 5,000-10,000 children in U.S.
- Girls > boys (8:1)
- Common initial presentation - malaise, fever, rash, joint pain, weight loss

# SLE clinical manifestations

- Hematologic (leukopenia, anemia, thrombocytopenia)
- Mucocutaneous (malar rash/ oral ulcers)
- Musculoskeletal (arthritis and arthralgias)
- Neurologic (headache)
- RENAL DISEASE – LUPUS NEPHRITIS
- Pulmonary disease
- Cardiac (pericarditis, valvular)
- Gastrointestinal

# SLE classification criteria

Systemic Lupus International Collaborating Clinics (SLICC) – group classification criteria

+Diagnosis if 4 or more of the 17 SLICC criteria

+At least one clinical and one immunologic criterion



# Lupus Nephritis - assessment

- Regular screening – UA, urine protein:creatinine, serum creatinine
- Additional laboratories – ANA, anti-ds-dna, anti- ENA, C3, C4, ESR, CRP
- Renal biopsy



# Renal Biopsy Findings

- Glomerular deposits that stain dominantly for IgG as well as IgA, IgM, C3 and C1q (Full House) immunofluorescence staining
- Glomerular deposits in the mesangial, subendothelial and subepithelial locations simultaneously
- Extra-glomerular immune-type deposits within tubular basement membranes, interstitium and blood vessels
- Tubuloreticular inclusion in the glomerular endothelial cells

# Lupus Nephritis Classification

- 1982 WHO (World Health Organization)
- 2004 ISN classification (International Society of Nephrology)
- Six different classes
  - Patients may evolve from one class to another

# Lupus Nephritis - classifications

- Class I – minimal mesangial
  - Very mild, only mesangial immune deposits
  - Clinical: normal UA, normal creatinine
- Class II – mesangial proliferative
  - Mesangial hypercellularity and mesangial matrix expansion
  - Clinical: microhematuria and/or proteinuria

# Lupus Nephritis - classifications

- Class III – Focal
  - Less than 50% glomeruli affected
  - Immunofluorescence IgG and C3 involvement
  - Segmental active or inactive endocapillary or extracapillary glomerulonephritis
  - Immune deposits seen on EM
  - Clinical: microhematuria, proteinuria, some hypertension

# Lupus Nephritis – classifications

- Class III SUBSETS

- Class III A: active lesions.

- Focal proliferative

- Class III A/C: active and chronic lesions.

- Focal proliferative and sclerosing

- Class III C: chronic inactive lesions with scarring.

- Focal sclerosing

# Lupus Nephritis - classifications

- Class IV – Diffuse
  - More than 50% glomeruli affected
  - Endocapillary and/or extracapillary glomerulonephritis
  - Mesangial abnormalities
  - Subendothelial depositis seen on EM
  - Clinical: hematuria and proteinuria. Frequent nephrotic syndrome, hypertension, elevated creatinine, low C3 and elevated anti-ds-dna



# Lupus Nephritis – classifications

- Class IV SUBSETS
  - Class IV – S (glomeruli affected segmentally)
  - Class IV – G (glomeruli affected globally)
  - Inflammatory activity
    - A (Active)
    - C (Chronic)



# Lupus Nephritis Classifications

- Class IV – S (A). Active lesions
  - Diffuse segmental proliferative
- Class IV – G (A). Active lesions
  - Diffuse global proliferative
- Class IV – S (A/C) active and chronic lesions
  - Diffuse segmental proliferative and sclerosing
- Class IV – G (A/C) active and chronic lesions
  - Diffuse global proliferative and sclerosing

# Lupus Nephritis – classifications

- Class IV – S (C). Chronic inactive lesions with scars
  - Diffuse segmental sclerosing
- Class IV – G (C). Chronic inactive lesions with scars
  - Diffuse global sclerosing

# Lupus Nephritis - classifications

- Class V – Membranous
  - Diffuse thickening of glomerular capillary wall
  - Subepithelial immune deposits
  - Clinical: nephrotic syndrome, microhematuria, may have elevated creatinine

# Lupus Nephritis - classifications

- Class VI – Advanced sclerosing
  - Global sclerosis of more than 90% of glomeruli
  - Advanced stage of chronic disease, not active
  - Clinical: slowly progressive renal dysfunction, proteinuria

# Lupus Nephritis Treatment

- Glucocorticoids
  - Oral, alternate day when possible
  - Intermittent high dose IV
- Steroid sparing
  - Mycophenolate (Buratti et al, 2001)
- Diffuse proliferative GN (class IV) – worst prognosis
  - Cyclophosphamide
  - Rituximab

# Lupus Nephritis - Treatment

- Prednisone/ IV methylprednisolone
  - Cataracts
  - Decreased bone density
  - Irritability/moodiness
  - Increased appetite, weight gain, cushingoid



# Lupus Nephritis - Treatment

- Mycophenolate Mofetil  
400-600 mg/m<sup>2</sup>/dose BID. Max 3000 mg/day
- - Malignancy. lymphoma, skin
  - Infection
  - Teratogenicity



# Lupus Nephritis - Treatment

- Cyclophosphamide (Class III, IV, V – off label)  
0.5 - 1.0 g/m<sup>2</sup> given monthly for 6 months  
(adjust by 0.25 g/m<sup>2</sup> for wbc or anc)
  - Increase fluids at home 1-2 days prior
  - Infusion with pre and post hydration
  - IVF and monitor specific gravity (<1.010)
  - Mesna to protect bladder

# Lupus Nephritis - Treatment

- Cyclophosphamide Side Effects
  - Bone marrow suppression
  - Impaired fertility (dose and duration dependent)
  - GI upset. Nausea and / or vomiting
  - Hemorrhagic cystitis, hematuria

# Lupus Nephritis – Treatment

- Rituximab (off label)
  - small observational studies
- 375 mg / m<sup>2</sup> weekly for 4 weeks  
(Melander, 2009)

# Lupus Nephritis Treatment

- Rituximab side effects
  - Fever, fatigue, chills, headache, rash, angioedema
  - Infusion reaction
  - Nausea
  - Mucocutaneous reaction, stevens-johnson syndrome
  - Lymphopenia, cytopenia, anemia, thrombocytopenia
  - Progressive multifocal leukoencephalopathy
  - Hepatitis B reactivation

# Lupus Nephritis – Treatment

- Rituximab Lab Monitoring
  - CBC with diff and platelets
  - BMP
  - Screen for HBV prior to initiation

# Prognosis Lupus Nephritis

- Canadian Study (Hagelberg et al, 2002)
  - Diffuse proliferative (class IV)
    - 10 year mortality 9%,
    - 19 year mortality 12%
    - 10 year ESRD 25%
    - 19 year ESRD 40%
  - Kidney failure resulting in transplant do well



# Lupus Nephritis Prognosis

- Canadian study (Hagelberg et al, 2002)
  - Mesangial (class II) or focal proliferative (class III)
    - No ESRD up to 21 years post diagnosis





**Bascom Hill**  
**University of Wisconsin - Madison**

# Diabetic Nephropathy

- Standards of Medical Care in Diabetes 2014  
(American Diabetic Association)
- Children and Adolescent Screening
  - **Nephropathy**
  - **Hypertension**
  - Dyslipidemia
  - Retinopathy
  - Celiac Disease
  - Hypothyroidism

# Diabetic Nephropathy Screening (ADA 2014)

- Annual albumin:creatinine when age 10, puberty or 5 years diabetes T1DM (at diagnosis for T2DM)
- Albuminuria 30 -300 mg/G creatinine
- Repeat 2 additional times, 1<sup>st</sup> AM on separate days
- Overt proteinuria can progress to diabetic nephropathy and risk of ESRD

# DN – Risk Factors

- Increased risk in patients with diabetic sibling or parent with diabetic nephropathy (Type 1 and Type 2)
- Increased risk with smoking, hypertension and poor glycemic control

# Proteinuria assessment

- Albuminuria.
  - Evaluate for proteinuria
- Labs – CBC, BMP, C3, C4, ANA, urinalysis
- Blood pressure
- Only proteinuria -> start ACE

# Diabetic Nephropathy Treatment

- ACE inhibitor. Start 5 mg/day
- Continue regular monitoring of urinalysis, urine protein:creatinine and labs (cbc, bmp)
- Improve glycemic control
- Smoking cessation, if necessary



# DN Renal Biopsy Pathophysiology

- Histologic Changes
  - Mesangial expansion
  - Glomerular basement membrane thickening
  - Glomerular sclerosis (may be associated with hyaline deposits in the glomerular arterioles)



# Diabetic Nephropathy in T2DM

- More prevalent than T1DM (Pinhas-Hamiel & Zeitler, 2007)
  - 14 to 22% albuminuria at presentation of T2DM
  - 60% albuminuria within 10 years from diagnosis
  - T2DM 4X more likely to develop renal failure than T1DM (Dart et al, 2012)



**Memorial Union Terrace  
University of Wisconsin - Madison**

# Diabetes - Hypertension screening

- Blood pressure with every health care visit
- Diagnosis of pre-hypertension or hypertension based on 4<sup>th</sup> Report (Pediatrics, 2004)
  - Pre-hypertension: systolic and/or diastolic  $> 90^{\text{th}}$  but  $< 95^{\text{th}}$  percentile. 3 separate occ.
  - Hypertension: systolic and/or diastolic  $> 95^{\text{th}}$  percentile. 3 separate occasions

# T1DM - Hypertension

- Increased incidence of hypertension in T1DM
  - (Rodriguez BL, Dabelea D, Liese AD et al, 2010)
- Patients with albuminuria had higher BP than those without albuminuria
  - (Guntsche Z, Saravi FD, Reyals EA et al, 2002)

# Hypertension treatment

- Pre-hypertension – Start non-pharmacologic
  - Low sodium diet
  - Exercise
  - Weight loss
  - Start pharmacologic if no improvement in 3-6 months
- Hypertension – start pharmacologic
- Goal is bp less than 90<sup>th</sup> percentile or < 120/80
- ACE inhibitor

# ACE/ARB guidelines

- Mechanism of Action
  - Inhibitor of angiotensin-converting enzyme / angiotensin receptor blocker
- Side Effects
  - Angioedema
  - Cough
  - Hyperkalemia
  - Syncope/hypotension
  - Neutropenia
  - Increased creatinine level
  - Fetal teratogenicity



# ACE monitoring

- Blood Pressure
- Labs within 3 months and regularly
  - BMP
  - CBC with diff
  - Urinalysis and urine protein:creatinine



**Camp Randall Stadium**  
**University of Wisconsin - Madison**

# Case Study

- Patient MH – 13 y.o. female with T1DM for 6 months, referred with albuminuria
  - History of one UTI when 8 yo
  - RUS normal
  - BP 96/54

# Case Study labs

- Creatinine 0.70
- Electrolytes normal
- Albumin normal
- CBC normal
- Hgb A1C 8.0
- C3/C4 normal
- ANA 1:80
- Anti – ENA low positive SSB
- 24 hour urine protein 150 mg (4.7 mg/m<sup>2</sup>/hour)

# Case study assessment summary

- Type 1 DM
- Mild proteinuria
- Insignificant ANA, + anti-SSB
- Low normal BP
- Start low dose ACE. Lisinopril 2.5 mg daily



# Case Study continued

- 4 months later
  - 24 hour urine protein 240 mg
  - ANA 1:160
  - Increase lisinopril to 5 mg daily
- 1 year later
  - ANA 1:320
  - C3 93/ C4 22
  - Anti ENA still negative except SSB low positive
  - 24 hour urine protein 290 mg



# Renal Biopsy at 1 year

- Light microscopy
  - Mild increase mesangial matrix
  - Focal, segmental, mild increase in cellularity
- Immunofluorescence
  - Mesangial Immune Deposits
  - IgA +4, IgM +2, C3 +1/+2, C1Q +1, lambda +3
  - IgG trace, Kappa +1

# Renal Biopsy pathology

- Electron Microscopy
  - Mild segmental increase in mesangial matrix and cellularity
  - Mesangial paramesangial and focal subendothelial electron dense immune like deposits
- Diagnosis
  - Mesangial Lupus Nephritis Class II

# Case Study Treatment

- Start mycophenolate mofetil 750 mg twice daily
- 6 months later - Repeat 24 hour urine protein 177 mg
- Continued monitoring of mild proteinuria while on mycophenolate and lisinopril



**Go Badgers!**

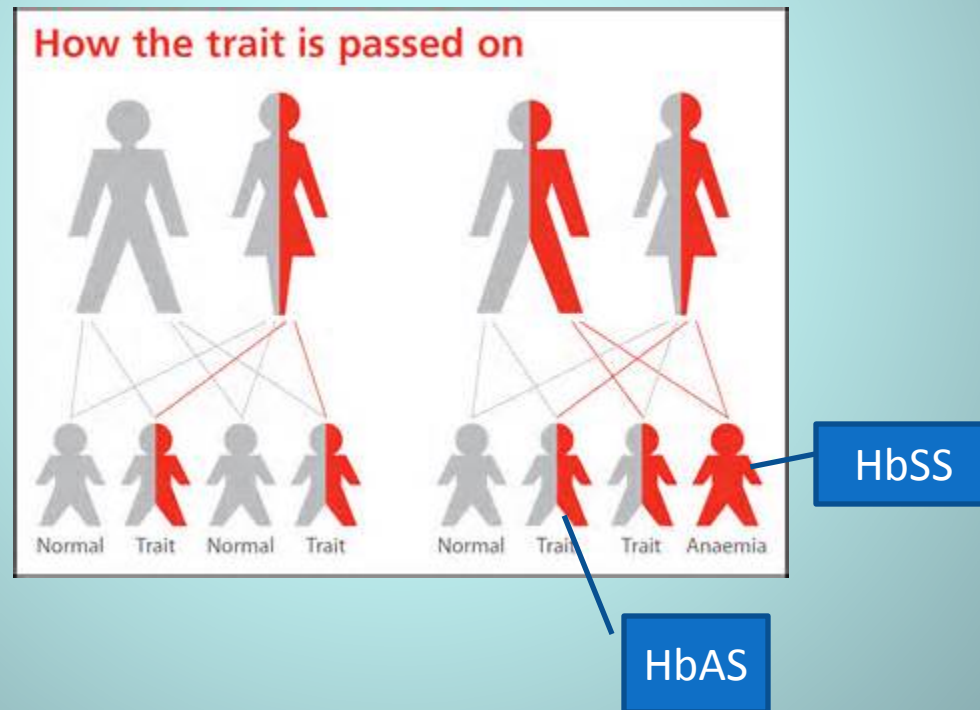


Children's Hospital of Wisconsin  
Milwaukee, WI

# **SICKLE CELL ANEMIA**



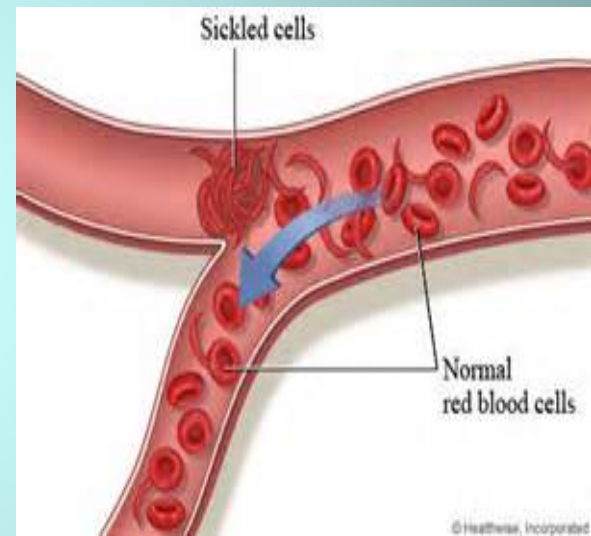
# Sickle Cell Disease-Inheritance



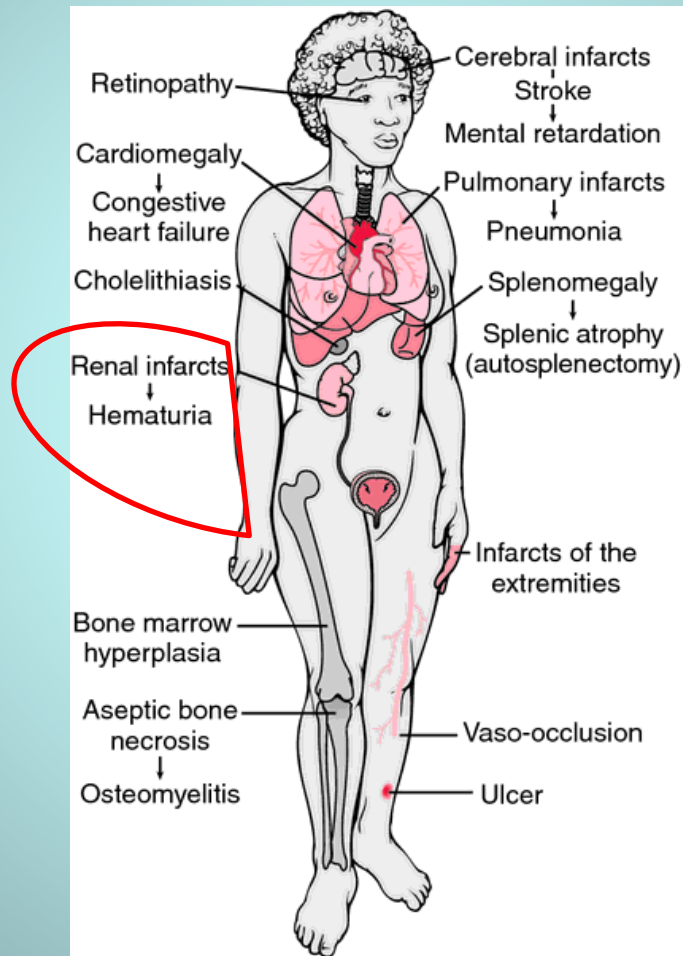
# Sickle Cell Disease

Sickle hemoglobin (HbS) is present in the RBC's

When deoxygenated, RBCs can develop a sickle or crescent shape. These cells are inflexible and increase blood viscosity



# Sickle Cell Disease-Complications



# Manifestations of Renal Disease

- Impaired urinary concentrating ability
- Hyperfiltration
- Hypertrophy
- Microalbuminuria/Proteinuria
- Hematuria
- Medullary cell carcinoma
- Hypertension
- Acute kidney injury
- End Stage Kidney Disease

# Blood Pressure and SCD

- Many children and adults have lower clinic blood pressures than age matched peers
- Pre-hypertension associated with increased risk of stroke and death
- Lack of normal nocturnal dip on ABPM common

Blood Pressure Abnormalities in Children with Sickle Cell Anemia. Becker AM, Goldber JH, Henson M, *et al.* *Pediatr Blood Cancer* 2014; 61: 518-522.

Masked Hypertension is Prevalent in Children with Sickle Cell disease: a Midwest Pediatric Nephrology Consortium Study. Shatat IF, Jakson SM, Blue AE, *et al.* *Pediatr Nephrol* 2013; 28: 115-120.



# Management of Sickle Cell Disease

Treatment	Rationale
Hydroxyuria	Improved/stabilization of GFR Decreased proteinuria
Inhibition of renin angiotensin system (Ace-I or ARB)	Reducing proteinuria may slow the progression of CKD
RBC transfusion	Outcomes have varied in effectiveness of delaying onset of proteinuria. May slow progression of established CKD
Erythropoiesis-stimulating agents	May require very high doses Hgb generally kept <10mg/dL to minimize risk of triggering venous occlusion
NSAID's	Used for pain control-can be damaging to kidneys
Aggressive treatment of hypertension	
Hemopoietic stem cell transplantation	



# Sickle Cell Trait



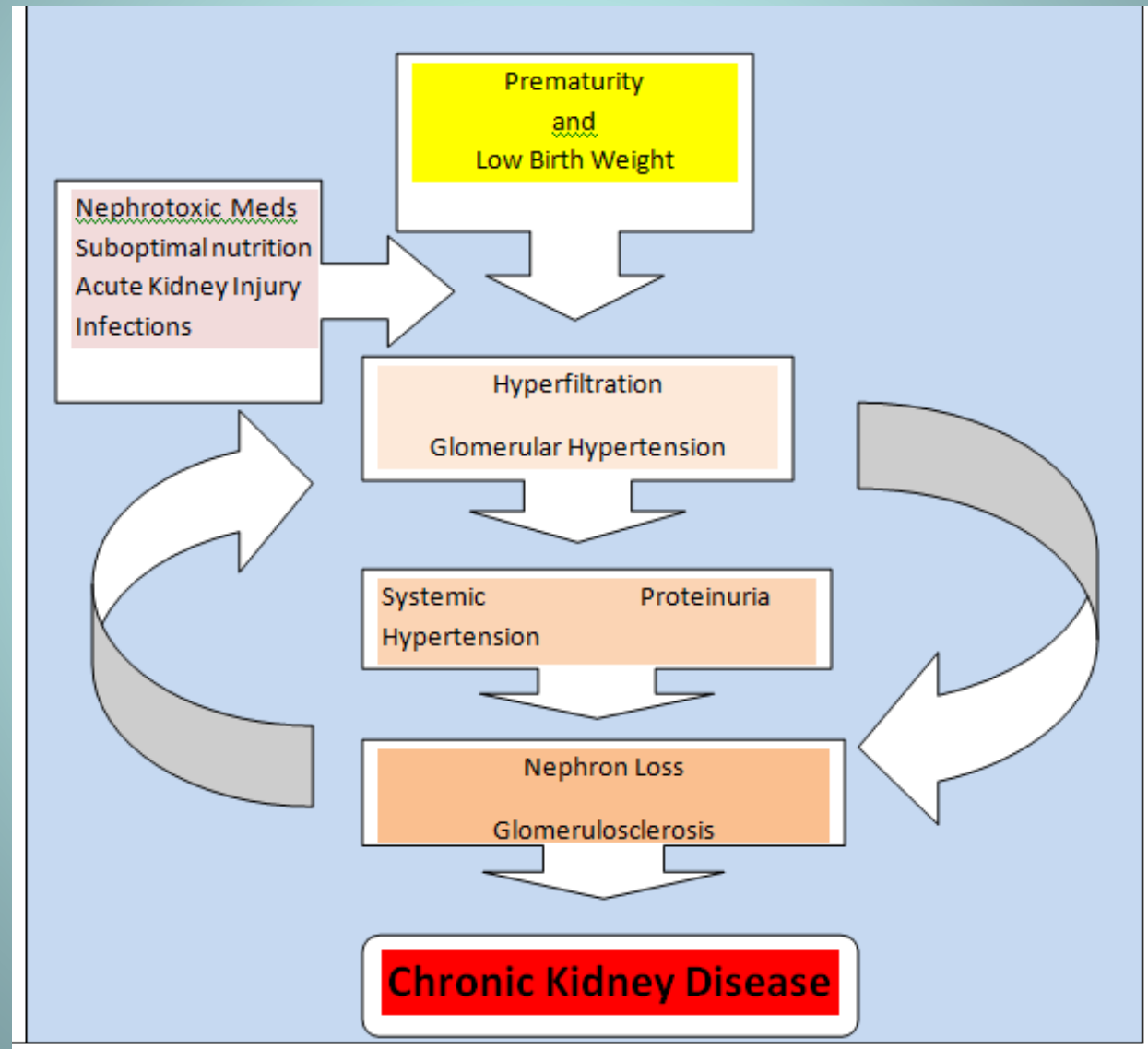
- Generally thought to be a benign condition with a normal life expectancy, however SCT can be associated with adverse outcomes
- Impaired urinary concentrating ability (less than SCA), asymptomatic hematuria and papillary necrosis
- Higher than expected prevalence of SCT among participants with ESRD
- Screening for CKD: Unsure if beneficial



# PREMATURITY

- Nephrogenesis begins around 4-5 weeks gestation
- 60% of nephrons are formed during the 3<sup>rd</sup> trimester
- Entire nephron complement is determined by 36 weeks gestation

- Nephrogenesis in infants born prematurely may continue postnatally, but may be altered (based on autopsy studies possibly prone to selection bias)
- Postnatal glomerulogenesis does not seem to occur after 40 days
- Nephrons must last a lifetime as they do not have the ability to regenerate



# AKI

- AKI in the NICU is common although incidence is difficult to determine
- Normal Cr at discharge is not a good indicator of long term CKD risk because increased tubular secretion can maintain a normal plasma creatinine until 25-50% of GFR has been lost



# Long Term Risk of Hypertension Associated with Prematurity

TABLE 2. Adulthood characteristics according to preterm birth and birth weight categories

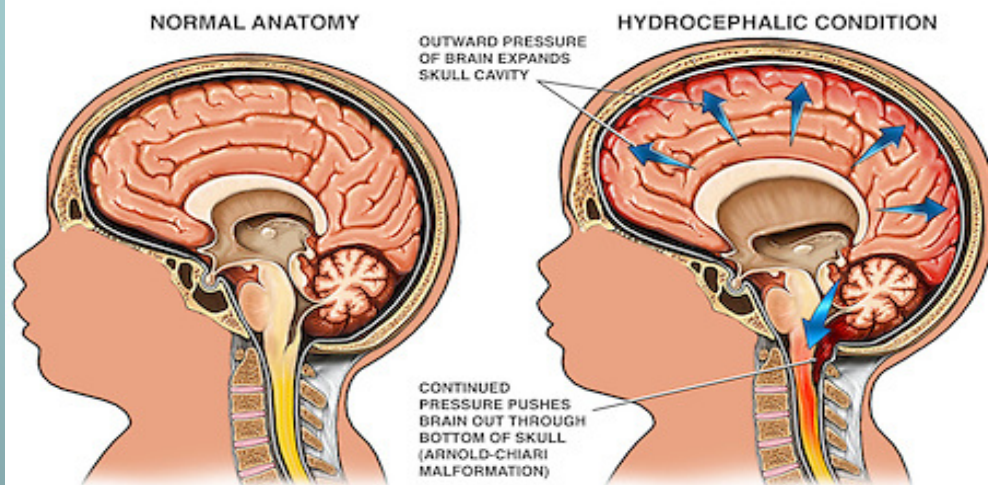
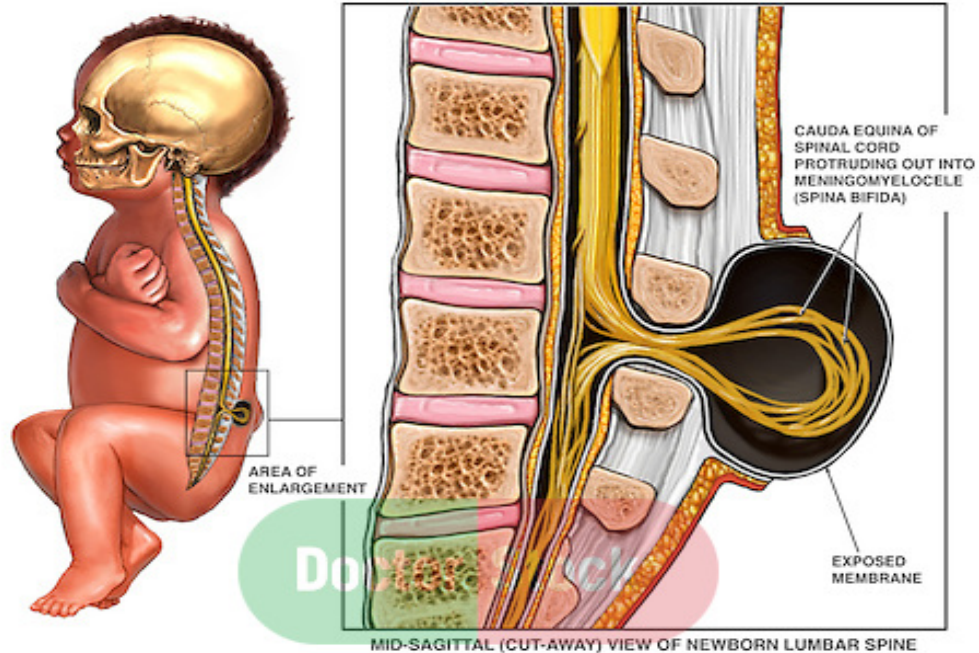
	Term (group I)		Preterm AGA (group II)		Preterm SGA (group III)		P I vs. II	P I vs. III	P II vs. III
	Mean	SD	Mean	SD	Mean	SD			
N	1630		87		39				
Males (%)	48.4		46.7		51.9		0.39	0.77	0.81
Age (years)	41.3	4.9	41.3	4.9	40.7	4.3	0.43	0.60	0.94
BMI (kg/m <sup>2</sup> )	26.5	5.0	27.3	5.6	27.3	5.1	0.10	0.25	0.96
Height (cm)	172.5	9.4	171.2	8.2	171.2	9.1	0.36	0.14	0.52
SBP (mmHg)	118.4	13.9	117.8	13	126.5	16.6	0.91	<0.001	0.004
DBP (mmHg)	74.4	10.4	74.8	10.9	78.7	12.3	0.57	0.004	0.13
LDL cholesterol (mmol/l)	3.25	0.82	3.16	0.81	3.49	1.04	0.40	0.08	0.07
HDL cholesterol (mmol/l)	1.31	0.33	1.34	0.34	1.35	0.31	0.78	0.44	0.74
Triglycerides (mmol/l)	1.32	1.22	1.5	2.1	1.47	0.68	0.17	0.45	0.90
Years of education	15.5	3.6	15.5	3.3	15	3.4	0.98	0.49	0.49
hs-CRP (mg/l)	1.7	2.7	1.6	2.7	1.4	1.7	0.73	0.51	0.80
Waist circumference (cm)	91.7	13.9	93.4	16.8	93.6	14.8	0.12	0.29	0.95
Glucose (mmol/l)	5.36	0.96	5.43	0.54	5.48	0.4	0.43	0.39	0.56
Insulin (mU/l)	10.1	14.9	10.6	8.5	10.8	6.7	0.86	0.91	0.81
HbA1C (mmol/mol)	36.6	5.2	36.8	3.7	37.4	3.2	0.70	0.29	0.31
Hypertensive BP levels (%)*	22.8		25.3		30.8		0.79	0.09	0.22
BP medication (%)	9.1		8.1		10.3		0.74	0.74	0.62

Effect of birth weight on life-course blood pressure levels among children born premature: the Cardiovascular Risk in Young Finns Study Juonala M, Cheung MMH, Sabin MA et al Jhypertension 33(8), 2015.

# Follow up of Premature/LBW Infants

- AAP recommends measuring BP at health maintenance examinations beginning at 3 years, however nearly half of extremely preterm infants have a systolic blood pressure >90% by age 2.5, so perhaps screening of high risk infants should occur at earlier visits
- Rapid growth in puberty often unmasks renal dysfunction because abnormal kidneys may be unable to accommodate the demands of increased growth
- Adolescents should be aware of their history of prematurity, understand their increased long term risk for CKD and receive counseling regarding modifiable risks (obesity, smoking, HTN)
- Earlier detection: treating HTN, microalbuminuria, and dyslipidemia could slow progression of CKD/facilitate education

# **MYELOMENINGOCELE (SPINA BIFIDA)**





# Myelomeningocele

## Neurosurgical Issues

Chiari II malformation  
Hydrocephalus-need for shunt  
Motor/Sensory deficit based on level of lesion

Tethered spinal cord

## Orthopedic Issues

Scoliosis  
Deformities of hip, knee, foot and ankle

## Skin Issues

Pressure Sores



## Neuropsychological Function

Learning disabilities  
Impairment of attention and memory

## Metabolic Syndrome

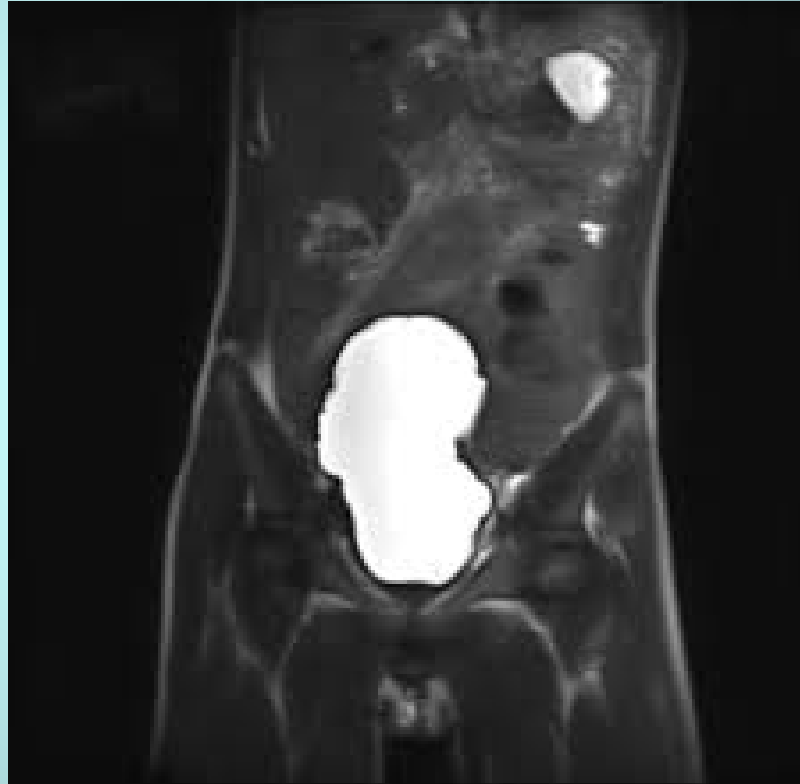
## Neurogenic Bowel and Bladder

Bladder sphincter dyssynergia  
High pressure bladder  
Flaccid, incontinent bladder

## Endocrine Issues

Precocious puberty  
Growth hormone deficiency

# Neurogenic Bladder

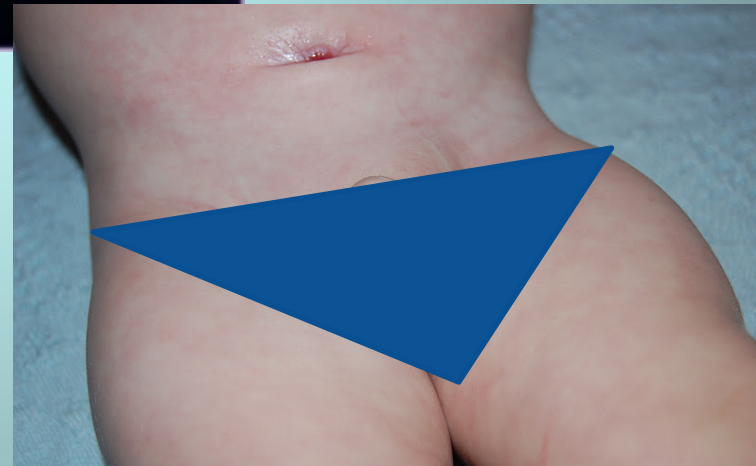




# Goals of Urinary Tract Management

- Identify lower urinary tract abnormalities
- Undertake strategies to preserve the upper urinary tract
- Provide continence at an appropriate age

# Bladder Drainage



# Pharmacotherapy

## Medication

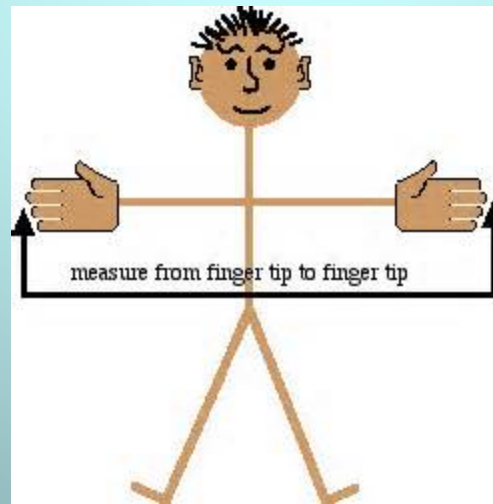
- Anticholinergic (oxybutynin chloride, tolterodine, trospium)
- Alpha-adrenergic blockers
- Botulinum toxin-A

## Reason For Use

- Used with CIC to lower filling and emptying pressures
- Facilitates bladder emptying
- Causes transient smooth muscle paralysis and improved urinary continence

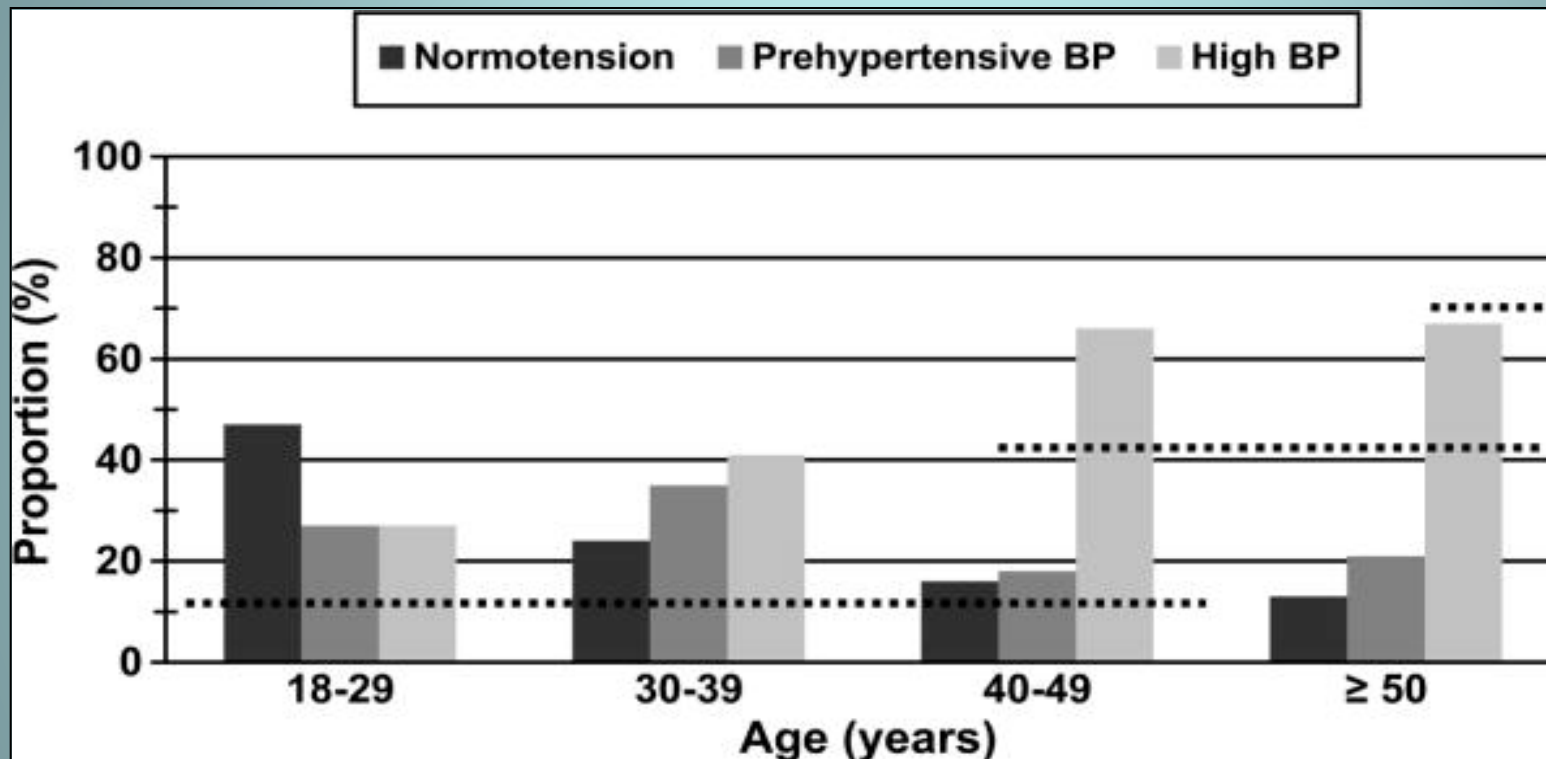
# Challenges in Assessing Renal Function in Children with Myelomeningocele

Finding ideal marker for measurement of renal function	GFR calculations (Modified Schwarz formula) based on height Creatinine based methods are insensitive because of low muscle mass and underdeveloped musculature in the legs Cystatin C: independent of muscle mass, height, size, and body mass Only reliable marker-but expensive
Determination of hypertension	Centiles based on height



# Occurrence of Hypertension and Prehypertension in Adults with Myelomeningocele

FIGURE 1 .. The dotted line represents the estimated prevalence of hypertension in adults 18-44 yrs old (10.5%), 45-64 yrs old (40.6%), and 65 yrs or older (70.3%) as reported by the National Health and Nutrition Examination Survey 2005-2008 cycle. BP indicates blood pressure.



**Young Adults with Spina Bifida May Have Higher Occurrence of Prehypertension and Hypertension.**

Stepanczuk, Beth; Dicianno, Brad; Webb, Thomas; MD, FAAP American Journal of Physical Medicine & Rehabilitation. 93(3):200-206, March 2014. DOI: 10.1097/PHM.0b013e3182a92b03

7 year old with sacral MMC

Ambulatory

No VP shunt

- At birth-UDM benign
- 11 months of age: bladder overactivity, dyssynergia and high pressures Ditropan and CIC to be started
- Family did not follow up
- Numerous attempts to contact family were not responded to
- One reported UTI
- Incontinent of urine and stool



2009



2015



	17 7/22/2014 0000	16 8/14/2014 1254	15 8/14/2014 1331	14 5/12/2015 1304	13 5/12/2015 1525	12 5/20/2015 1329	11 5/20/2015 1333	10 5/20/2015 1424	9 6/3/2015 1434	8 6/3/2015 1716	7 6/9/2015 1222	6 6/25/2015 1159	5 6/25/2015 1159
<b>GENERAL CHEMISTRY</b>													
Sodium Blood					140	140				142		142	
Potassium Blood					3.9	4.3				4.2		3.9	
Chloride Blood					109	108				108		110	!
Carbon Dioxide					14.5 *	19.6 *	!			20.9 *		18.0 *	!
Glucose Blood					95 *	79 *				108 *	!	84 *	
BUN					50	49	!			28	!	42	!
Creatinine Blood					2.43	1.85	!			1.64	!	1.58	!
Phosphate Blood						5.3				5.5		6.7	
Calcium Blood					9.6	9.3				9.3		9.1	
Albumin Blood						4.2							
Ferritin										6.0	!		
Iron Blood										104			
Iron Binding Capacity										402			
% Saturation										26			
<b>STERIODS &amp; HORMONES</b>													
25 OH Vitamin D Total													
25 OH Vitamin D To...						32 *							
25 OH Vitamin D D3...						32 *							
25 OH Vitamin D D2...						< 4 *							
PTH Intact Result							334.5	!					
<b>GENERAL HEMATOLOGY</b>													
WBC					7.5	5.2				8.9		7.8	
RBC					3.81	3.76	!			3.83	!	4.03	
Hemoglobin					10.0	10.1	!			9.9	!	10.3	!
Hematocrit					29.1	28.7	!			29.7	!	30.9	!
MCV					76	76				78		77	

Start CIC &  
anticholinergic  
medication

## Spina Bifida and Renal Function

Background and Review of Literature: Deaths from renal failure in patients with myelomeningocele (MMC) have decreased dramatically since the initiation of clean intermittent catheterizations and aggressive urologic management beginning in infancy. Although most infants with MMC are born with normal renal function, there are multiple risk factors for deterioration of renal function. These include:

- Vesicoureteral reflux (especially if female)
- Recurrent UTI-especially pyelonephritis
- Urolithiasis (increased incidence due to inactivity)
- Hypertension
- Bladder augmentation leading to metabolic derangements
- Late initiation of CIC (after 1 year of age))
- Obesity
- Neuropathic bladder
- Renal scarring
- Neurogenic bowel constipation increased risk of UTI
- Increasing age

Challenges to evaluating renal function in children with MMC: Serum creatinine is a poor marker for many children with MMC due to their decreased muscle mass in the lower extremities. Blood pressure norms and calculations of GFR are based on a patient's height. Arm span measurements have been used as a substitute for length/height for children with MMC, however arm span and height may not be interchangeable. Obesity, an independent risk factor for the development of hypertension is also difficult to assess in this population. Falsely low BMI's may occur for children with a loss of large muscle mass in the hips and lower extremities.

## Suggested Referral to Nephrology

Blood pressure above the 90% for age, gender, height on 3 occasions	Elevated automated blood pressures should be rechecked using the manual method Elevated blood pressures should be re-checked several times over the next few weeks
Abnormal renal parenchyma/significant hydronephrosis on renal ultrasound	Renal function tests (BMP, phosphate) should be done at least yearly for early identification of children with renal insufficiency/failure
Elevated serum creatinine or electrolyte abnormalities	
Bladder augmentation	Renal function tests (BMP, phosphate) should be done at least yearly for early identification of children with renal insufficiency/failure

# Why is this important?

- Opportunities to partner with other specialists for earlier identification of children with kidney disease

