UNDERSTANDING FSGS

Definitions – terminology - classification

Pathogenesis

Predicting prognosis

Choosing therapy
DEFINING & CLASSIFYING GLOMERULAR DISEASE

Histopathology

Clinical

Disease mechanisms
DEFINING & CLASSIFYING GLOMERULAR DISEASE

- Histopathology
- Clinical
- Disease mechanisms
FOCAL SEGMENTAL GLOMERULOSCLEROSIS

IgM
FOCAL SEGMENTAL GLOMERULOSCLEROSIS

An histological pattern ....... *not* a diagnosis
Pathological definition:
SEGMENTAL OBLITERATION OF GLOMERULAR CAPILLARIES
BY EXTRACELLULAR MATRIX
Pathological definition:
SEGMENTAL OBLITERATION OF GLOMERULAR CAPILLARIES
BY EXTRACELLULAR MATRIX

PODOCYTE INJURY
Pathological definition:
SEGMENTAL OBLITERATION OF GLOMERULAR CAPILLARIES
BY EXTRACELLULAR MATRIX

PODOCYTE INJURY

Stereotypic podocyte response to injury is:

PODOCYTE FOOT PROCESS EFFACEMENT
Actin cytoskeleton rearrangement
Pathological definition:
SEGMENTAL OBLITERATION OF GLOMERULAR CAPILLARIES BY EXTRACELLULAR MATRIX

PODOCYTE INJURY

Clinical correlate is proteinuria

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LOSS OF PODOCYTES
Detachment or cell death
Pathological definition:
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Podocytes in urine: evidence of ongoing injury in primary & recurrent FSSGS

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Actin cytoskeleton rearrangement

Clinical correlate is proteinuria

Podocytes in urine: evidence of ongoing injury in primary & recurrent FSSGS

LOSS OF PODOCYTES
Detachment or cell death

GBM is denuded
Healthy PODOCYTE *does not replicate*

Very high expression of cyclin kinase inhibitors
Healthy PODOCYTE *does not replicate*

Very high expression of cyclin kinase inhibitors

**REPAIR**

Some degree of GEC proliferation in all FSGS

- Mostly migrating parietal calls

Progenitor cells can arise from parietal layer:

- Can they become mature podocytes?
DEFINING & CLASSIFYING GLOMERULAR DISEASE

- Histopathology
- Clinical
- Disease mechanisms
NOS
Not otherwise specified

Perihilar

Cellular

Collapsing
NOS
Not otherwise specified

Perihilar

Cellular

Tip lesion

Collapsing
TIP LESION

Is it a variant of minimal change?

Is it a variant of FSGS?

Does it matter?
Suggested mechanisms of injury

NOS
Not otherwise specified

Perihilar

Cellular

Tip lesion

Collapsing
Suggested mechanisms of injury

- Tip lesion
- Cellular
- Collapsing
- Podocyte depletion
- Perihilar

NOS
Not otherwise specified
Suggested mechanisms of injury

Podocyte depletion

Perihilar

Cellular

Podocyte depletion

Tip lesion

Podocytes are flow sensitive
Tip podocytes subject to greater turbulence

Collapsing
**Suggested mechanisms of injury**

**Podocyte depletion**

**Cellular**

Podocytes are flow sensitive. Tip podocytes subject to greater turbulence.

**Tip lesion**

**Not otherwise specified**

**Perihilar**

**Collapsing**

Immature podocyte phenotype

? Hypercellularity = dedifferentiated podocytes and mature parietal cells.
NOS
Not otherwise specified

Podocyte depletion

Perihilar

Podocyte depletion

Suggested mechanisms of injury

Tip lesion

Podocytes are flow sensitive
Tip podocytes subject to greater turbulence

Cellular

Speculation only

Collapsing

Immature podocyte phenotype

? Hypercellularity = dedifferentiated podocytes and mature parietal cells
PODOCYTE INJURY & FSGS

Why are the lesions segmental?

Why are juxtamedullary glomeruli usually affected first?
DEFINING & CLASSIFYING GLOMERULAR DISEASE

Histopathology

Clinical

Disease mechanisms
DEFINING & CLASSIFYING GLOMERULAR DISEASE

Histopathology

Clinical

Disease mechanisms
CLINICAL PRESENTATIONS OF FSGS

PRIMARY FSGS

Typically ‘like minimal change’:
Sudden onset of nephrotic syndrome
Any age – but commonest in children and young adults

SECONDARY FSGS

More often:
Asymptomatic proteinuria
Normal serum albumin
Any age
CLINICAL PRESENTATIONS OF FSGS

SECONDARY FSGS

More often:
- Asymptomatic proteinuria
- Normal serum albumin
- Any age

PRIMARY FSGS

Typically ‘like minimal change’:
- Sudden onset of nephrotic syndrome

Any age – but commonest in children and young adults


Lennox Hill Hospital, New York 1986 - 2002
Secondary FSGS
Secondary FSGS

Perihilar pattern more common
Drugs

Intravenous heroin
Pamidronate
Interferon-α, β, γ
Anabolic steroids

Secondary FSGS
Viruses
- HIV
- Parvovirus B19

Drugs
- Intravenous heroin
- Pamidronate
- Interferon-α, β, γ
- Anabolic steroids

Secondary FSGS
Viruses
- HIV
- Parvovirus B19

Drugs
- Intravenous heroin
- Pamidronate
- Interferon-α, β, γ
- Anabolic steroids

Adaptive response to reduced renal mass
- Renal agenesis/dysplasia
- Oligomeganephronia
- Surgical renal ablation
- Reflux nephropathy
- Cortical necrosis
- Chronic allograft nephropathy
- Any advanced renal disease with reduction in functioning nephrons

Secondary FSGS
**Adaptive response to reduced renal mass**

- Renal agenesis/dysplasia
- Oligomeganephronia
- Surgical renal ablation
- Reflux nephropathy
- Cortical necrosis
- Chronic allograft nephropathy
- Any advanced renal disease with reduction in functioning nephrons

**Adaptive response with initially normal renal mass**

- Obesity
- Sickle cell nephropathy
- Congenital cyanotic heart disease
- ? Very low birth weight

**Drugs**

- Intravenous heroin
- Pamidronate
- Interferon-α, β, γ
- Anabolic steroids

**Viruses**

- HIV
- Parvovirus B19

**Secondary FSGS**
**VERY LOW BIRTH WEIGHT AND FSGS**

4 men, 2 women. Mean age 32 yrs
Born at 22-30 weeks gestation
Birth weight 450-1420g

<table>
<thead>
<tr>
<th>Condition</th>
<th>Value</th>
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</thead>
<tbody>
<tr>
<td>Nephrotic range proteinuria</td>
<td>3/6</td>
</tr>
<tr>
<td>Hypoalbuminaemia</td>
<td>0/6</td>
</tr>
<tr>
<td>Oedema</td>
<td>0/6</td>
</tr>
<tr>
<td>GFR</td>
<td>70-130</td>
</tr>
</tbody>
</table>

Glomerulomegaly

FSGS – perihilar variant 5/6

Hodgin J *et al.* CJASN 2009; 4: 62
Primary
( Idiopathic )
FSGS

Secondary
FSGS
Primary
( Idiopathic )
FSGS

Genetic
FSGS

Secondary
FSGS
Primary (Idiopathic) FSGS

Genetic FSGS

Secondary FSGS
Primary (Idiopathic) FSGS

Genetic FSGS

Secondary FSGS
Molecules identified in genetic forms of FSGS

Schell C, Huber T. NDT 2012; 27: 3406

- Slit diaphragm
  - Nephrin
  - Podocin
  - CDA2P

- Actin cytoskeleton
  - alpha-actinin-4
  - inverted formin 2
  - Non-muscle myosin 1E

- Podocyte-GBM integrity
  - Laminin β2
  - Phospholipase Cε1

- Calcium homeostasis
  - Transient receptor potential cation 6

- Apical membrane
  - GLEPP-1

- Mitochondrial products
# Utility of Genetic Testing in Steroid-Resistant Nephrotic Syndrome

Genetic testing for: NPHS1, NPHS2, TRPC6, CDA2P, PLCE1, INF2, WT1, ATN4

<table>
<thead>
<tr>
<th>Age of onset</th>
<th>Causing mutations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital</td>
<td>100%</td>
</tr>
<tr>
<td>Infantile</td>
<td>57%</td>
</tr>
<tr>
<td>Early childhood</td>
<td>24%</td>
</tr>
<tr>
<td>Late childhood</td>
<td>36%</td>
</tr>
<tr>
<td>Adolescent</td>
<td>25%</td>
</tr>
<tr>
<td>Adult</td>
<td>14%</td>
</tr>
<tr>
<td>Adult – FSGS &amp; ESRD</td>
<td>8%</td>
</tr>
</tbody>
</table>

Santin S et al. CJASN 2011; 6: 1139
Buscher AK et al. Clin Nephrol 2012; 78: 47
PREDICTIONS

FSGS associated with podocyte protein mutations
PREDICTIONS

FSGS associated with podocyte protein mutations

... will be steroid resistant
FSGS associated with podocyte protein mutations

... will be steroid resistant

......unless the ‘second hit’ is steroid responsive
PREDICTIONS

FSGS associated with podocyte protein mutations

... will be steroid resistant

... will not recur in transplants
Variants at chromosome 22q13 confer susceptibility for FSGS & HIVAN in African Americans

APOL1 variants *not* MHY9 are associated with risk for FSGS & HIVAN in African Americans

.... and protect against trypanosomiasis

Genovese G et al. Science 2010; 329: 841
*etc.*
DEFINING & CLASSIFYING GLOMERULAR DISEASE

- Histopathology
- Clinical
- Disease mechanisms
NEPHROTIC CHILDREN

Steroid-sensitive

Steroid-resistant
Steroid-sensitive NEPHROTIC CHILDREN

Steroid-resistant NEPHROTIC CHILDREN

Minimal change disease

Focal segmental glomerulosclerosis

Other glomerular diseases

NEPHROTIC ADULTS
NEPHROTIC CHILDREN

Steroid-sensitive

Steroid-resistant

NEPHROTIC ADULTS

Minimal change disease

Focal segmental glomerulosclerosis

Other glomerular diseases
Steroid-sensitive NEPHROTIC CHILDREN

Steroid-resistant NEPHROTIC CHILDREN

Minimal change disease

Focal segmental glomerulosclerosis

Other glomerular diseases

NEPHROTIC ADULTS
NEPHROTIC SYNDROME

Normal BP – usually

Stick-positive haematuria ~ 10%
MINIMAL CHANGE DISEASE

NEPHROTIC SYNDROME

COMPLETE REMISSION

WITH

CORTICOSTEROIDS
MINIMAL CHANGE DISEASE

NEPHROTIC SYNDROME

COMPLETE REMISSION

WITH

CORTICOSTEROIDS

Why do steroids work?
Steroid response in minimal change disease

- Children
- Adults

Complete remission (%)

Weeks from beginning corticosteroid therapy

0 2 4 8 16

Histologic patterns and steroid responsiveness in diseases causing nephrotic syndrome

Steroid responsive
Mineral change disease (MCD)
Mesangial hypercellularity
Focal segmental glomerulosclerosis (FSGS)
IgM
Histologic patterns and steroid responsiveness in diseases causing nephrotic syndrome

- Steroid responsive
- Minimal change disease (MCD)
- Mesangial hypercellularity
- Focal segmental glomerulosclerosis (FSGS)
- IgM

Tip lesion
Histologic patterns and steroid responsiveness in diseases causing nephrotic syndrome

- Steroid responsive
- Minimal change disease (MCD)
- Mesangial hypercellularity
- Focal segmental glomerulosclerosis (FSGS)
- Collapsing
- IgM
Histologic patterns and steroid responsiveness in diseases causing nephrotic syndrome

- Steroid responsive
- Minimal change disease (MCD)
- Mesangial hypercellularity
- Focal segmental glomerulosclerosis (FSGS)
- IgM

Genetic forms?
‘Idiopathic’ nephrotic syndrome
‘idiopathic’ nephrotic syndrome

MCD-FSGS ‘spectrum’
Isn’t it steroid responsiveness that defines a ‘disease’?

Isn’t that more important than presence of FSGS?
POSSIBLE RELATIONSHIPS BETWEEN MCD AND FSGS

A spectrum?
POSSIBLE RELATIONSHIPS BETWEEN MCD AND FSGS

A spectrum?

Favoured by:
- Glomerular size
- Genetic factors
POSSIBLE RELATIONSHIPS BETWEEN MCD AND FSGS

A spectrum?

or

Two distinct podocytopathies

MCD

FSGS
RELATIONSHIPS BETWEEN MCD AND FSGS

A spectrum? 

or

Two distinct podocytopathies

MCD

CD80 overexpression on podocytes

IL-13 overexpression in T cells

Circulating factor(s)

Hemopexin
Angiopoietin-like 4

FSGS

Circulating factor(s)

CLC-1
Cardiotrophin-like cytokine 1
(IL-6 family)

suPAR
Soluble urokinase plasminogen activator receptor
(acute phase reactant)
CIRCULATING suPAR IN FSGS

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Circulating suPAR positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>North America</td>
<td>70</td>
<td>84%</td>
</tr>
<tr>
<td><em>CT trial</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Europe</td>
<td>94</td>
<td>55%</td>
</tr>
<tr>
<td><em>PodoNet</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>6%</td>
</tr>
</tbody>
</table>

CT trial
Reduction in suPAR
associated with
MMF therapy
reduction in proteinuria

PodoNet
Higher suPAR levels
associated with
NPHS2 mutation

Wei C et al. JASN 2012; 23: 2051
A FORTY YEAR OLD CONUNDRUM ....

‘Can minimal change evolve into FSGS?’

OR

‘It was FSGS all along but you just missed it?’
A FORTY YEAR OLD CONUNDRUM ....

‘Can minimal change evolve into FSGS?’

Steroid resistant *but* biopsy still
= Minimal Change

‘It was FSGS all along. Did it?’
A FORTY YEAR OLD CONUNDRUM ....

‘Can minimal change evolve into FSGS?’

Steroid resistant *but* biopsy still = Minimal Change

Does this mean the FSGS lesions are still being missed?

‘It was FSGS all along but you just missed it?’
A FORTY YEAR OLD CONUNDRUM ….

‘Can minimal change evolve into FSGS?’

Steroid resistant *but* biopsy still = Minimal Change

Does this mean the FSGS lesions are still being missed?

… *small biopsies*
… *superficial biopsies*
A FORTY YEAR OLD CONUNDRUM ....

‘Can minimal change evolve into FSGS?’

Early recurrent FSGS

Nephrotic
Foot process effacement

NO segmental lesions

‘It was FSGS all along but you just missed it?’
A FORTY YEAR OLD CONUNDRUM ....

‘Can minimal change evolve into FSGS?’

Does it matter in clinical practice?

‘It was FSGS all along but you just missed it?’
CLASSIFICATION OF FSGS

Aetiology & Pathogenesis

Genetics

Pathology

Clinical features

Response to corticosteroids
UNDERSTANDING FSGS

Definitions – terminology - classification

Pathogenesis

Predicting prognosis

Choosing therapy
PRIMARY FSGS

Can we predict outcome at presentation?

HISTOLOGY
NOS
Not otherwise specified

Perihilar

Cellular

Tip lesion

Collapsing
FSGS – HISTOLOGICAL VARIANTS & OUTCOME
# FSGS – HISTOLOGICAL VARIANTS & OUTCOME

RCT in 138 children and young adults with steroid-resistant FSGS in the USA

<table>
<thead>
<tr>
<th>Variant</th>
<th>%</th>
<th>ESRD @ 3 yrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>NOS</td>
<td>68%</td>
<td>20%</td>
</tr>
<tr>
<td>Perihilar</td>
<td>7%</td>
<td>-</td>
</tr>
<tr>
<td>Cellular</td>
<td>3%</td>
<td>-</td>
</tr>
<tr>
<td>Collapsing</td>
<td>12%</td>
<td>47%</td>
</tr>
<tr>
<td>Tip lesion</td>
<td>10%</td>
<td>7%</td>
</tr>
</tbody>
</table>

D’Agati V et al. CJASN 2013; 8: 399
RCT in 138 children and young adults with steroid-resistant FSGS in the USA
Treated with cyclosporin or MMF/dexamethasone

D’Agati V et al. CJASN 2013; 8: 399
PRIMARY FSGS

Can we predict outcome at presentation?

GENETICS

Not yet ....
PRIMARY FSGS

Can we predict outcome at presentation?

CLINICAL

Non-nephrotic 20% ESRD at 10 years

Nephrotic >50% ESRD at 5-10 years

Nephrotic >10g/day ~100% ESRD at 5-10 years
PRIMARY FSGS
Survival according to remission

Remission
No Remission

5yr survival
94% v 52%

Time to RRT or death (yrs)

REMISSION & ESRD IN FSGS

No Remission vs. Partial remission with a relapse

![Graph showing renal survival over years with PR rel+ and NR curves, with survival rates and numbers at specific years: PR rel+ 61, 36, 14, 6; NR 108, 43, 13, 5.](image)

p=0.001 NR<PR rel+

Troyanov S. et al. JASN 2005; 16:1061
UNDERSTANDING FSGS

Definitions – terminology - classification

Pathogenesis

Predicting prognosis

Choosing therapy
PREDICTING RESPONSE TO CORTICOSTEROIDS IN FSGS

Histology

**Tip lesion** – steroid responsive

**Collapsing variant** – steroid resistant
PREDICTING RESPONSE TO CORTICOSTEROIDS IN FSGS

Genetics

- More steroid resistance in African Americans
- Familial FSGS – usually steroid resistant
Does spontaneous remission occur?

- United States: 4-6%
- European study: 16%
- UK 5 centre study: 23%
Evidence from randomised controlled trials...
CORTICOSTEROIDS IN FSGS

No RCTs - retrospective cohort studies

1980’s

8 weeks oral prednisolone ~ 1mg/kg/day [based on MCNS in children]

Complete remission ~ 20-30%
CORTICOSTEROIDS IN FSGS

No RCTs - retrospective cohort studies

1980’s
8 weeks oral prednisolone ~ 1mg/kg/day  [based on MCNS in children]
Complete remission ~ 20-30%

1990’s
At least 4 months - prednisolone starting 1-2 mg/kg/day
➢ 4 months treatment - complete remission – 50-60%
INTERPRETING RETROSPECTIVE STUDIES OF TREATMENT IN FSGS

Are the demographics typical?

Is the histology defined?

Were causes of secondary FSGS excluded?

Was the genetics defined?

How was response defined?
INTERPRETING RETROSPECTIVE STUDIES OF TREATMENT IN FSGS

Any immunosuppressive therapy must add benefit to ‘best supportive therapy’…

BP 125/75
ACE inhibitor ± ARB
Statin

Were these achieved in retrospective studies?
Is a ‘response’ to 6 months of corticosteroids just a spontaneous remission?

What is acceptable toxicity?
Steroid-sensitive NEPHROTIC CHILDREN

Steroid-resistant NEPHROTIC CHILDREN

NEPHROTIC ADULTS

Minimal change disease

Focal segmental glomerulosclerosis

Other glomerular diseases
KDIGO Clinical Practice Guideline for GN

STEROID-SENSITIVE NEPHROTIC SYNDROME IN CHILDREN

MINIMAL CHANGE DISEASE IN ADULTS

STEROID-RESISTANT NEPHROTIC SYNDROME IN CHILDREN

IDIOPATHIC FSGS IN ADULTS
Examples of Rating Guideline Recommendations

QUALITY of Supporting Evidence is shown as A, B, C or D

<table>
<thead>
<tr>
<th>Level</th>
<th>Recommendation</th>
<th>Quality Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1</td>
<td>We recommend….</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Most patients should receive the recommended course of action</td>
<td>1A</td>
</tr>
<tr>
<td></td>
<td>Supported by evidence from high quality RCTs</td>
<td></td>
</tr>
<tr>
<td>Level 2</td>
<td>We suggest …</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Different choices will be appropriate for different patients. Each patient needs help to arrive at a management decision appropriate for them</td>
<td>2D</td>
</tr>
<tr>
<td></td>
<td>No RCTs</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Supported by limited observational data</td>
<td></td>
</tr>
</tbody>
</table>
**Examples of Rating Guideline Recommendations**

<table>
<thead>
<tr>
<th>Level</th>
<th>Recommendation</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1</td>
<td>We recommend…</td>
<td>1A, 1B</td>
</tr>
<tr>
<td></td>
<td>Most patients should receive the recommended course of action</td>
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<td></td>
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Of 42 recommendations or suggestions for steroid-sensitive & steroid-resistant NS - in children

Only 8 (19%) are 1A or 1B
Examples of Rating Guideline Recommendations

- Level 1: We recommend...
  - Most patients should receive the recommended course of action
  - Supported by evidence from high quality RCTs

- Level 2: We suggest...
  - Different choices will be appropriate for different patients.
  - Each patient needs help to arrive at a management decision appropriate for them
  - Supported by limited observational data

QUALITY of Supporting Evidence is shown as A, B, C or D

Of 20 recommendations or suggestions for MCD, FSGS – in adults

None are 1A or 1B

Of 42 recommendations or suggestions for steroid-sensitive & steroid-resistant NS – in children

Only 8 (19%) are 1A or 1B
Examples of Rating Guideline Recommendations

QUALITY of Supporting Evidence is shown as A, B, C or D

<table>
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<th>1A</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Evidence from high quality RCTs</td>
</tr>
</tbody>
</table>

The recommendations or suggestions

*only apply* to nephrotic syndrome

*not* to asymptomatic proteinuria

<table>
<thead>
<tr>
<th>Level 2</th>
<th>Different choices will be appropriate for different patients. Each patient needs help to arrive at a management decision appropriate for them</th>
<th>No RCTs</th>
<th>Supported by limited observational data</th>
</tr>
</thead>
</table>
Female, born 1955 – Primary FSGS
Female, born 1955 – Primary FSGS

Proteinuria (g/day):
- 1996: 4
- 1998: 3
- 2000: 2
- 2002: 1
- 2004: 0
- 2006: 5
- 2008: 4
- 2012: 3

Serum albumin (g/l):
- 1996: 40
- 1998: 30
- 2000: 20
- 2002: 10
- 2004: 30
- 2006: 40
- 2008: 30
- 2012: 20

GFR (ml/min):
- 1996: 100
- 1998: 100
- 2000: 100
- 2002: 100
- 2004: 100
- 2006: 100
- 2008: 100
- 2012: 100

Pred
- 1996: Present
- 1998: Present
- 2000: Present
- 2002: Present
- 2004: Present
- 2006: Present
- 2008: Present
- 2012: Present

CyA
- 1996: Present
- 1998: Present
- 2000: Present
- 2002: Present
- 2004: Present
- 2006: Present
- 2008: Present
- 2012: Present
Female, born 1955 – Primary FSGS