Systematic Review

**Conclusion**

Considering the lack of good quality studies, we could not determine an optimal fixation technique regarding the prevention of complications to the SRN. However, based on our data, a trend towards pin fixation being the safest technique for fixing complex wrist fractures is present. Pin fixation did not only induce the lowest incidence of complications in absolute numbers, but also showed the highest rate of resolving complications of the SRN. More research has to be done to determine whether pin fixation can be considered best and if pin fixation is best for all types of distal radial fractures. High quality RCTs are needed.

**Appendix A**

Used search string: ((("Wrist Joint"[Mesh]) OR "Radius"[Mesh]) OR "Radius Fractures"[Mesh]) OR "Wrist Injuries"[Mesh]) AND ((("Fracture Fixation"[Mesh]) OR "Fracture Fixation, Internal"[Mesh]) OR “Bone Nails”[Mesh]) OR “Bone Wires”[Mesh]) OR “External Fixators”[Mesh]) OR “Bone Plates”[Mesh]) AND (((("Radial Nerve"[Mesh]) OR "Radial Neuropathy"[Mesh]) OR “Postoperative Complications”[Mesh]) OR “Nerve Compression Syndromes”[Mesh]) OR “superficial radial nerve”) AND (((complication*) AND “radial nerve”) OR “superficial radial nerve”) NOT (("Elbow Joint"[Mesh]) OR “Elbow”[Mesh]) OR “Humerus”[Mesh]) ] AND English [lang]

**References**

Glomerular diseases for dummies

A Clinical Lesson

Maaike C.L. Hama, Marco C. van Maurika, Rosan L. Lechnera, Stefan P. Bergerb
a Medical student, Erasmus MC University Medical Center Rotterdam, the Netherlands
b Supervisor, Department of Nephrology, Erasmus MC University Medical Center Rotterdam, the Netherlands
Correspondence: Marco van Maurik, e-mail: 353417mm@student.eur.nl

Box 1. Overview A literature overview answering the following questions:

- When to consider the involvement of glomerular disease?
- Are there different types of Glomerular disease?
- Which glomerular diseases should be considered when there is isolated haematuria?
- Which glomerular disease should be considered when there is isolated proteinuria?
- Does the amount proteinuria help my diagnoses?
- Does the combination of haematuria and proteinuria help my diagnosis?
- Does the patient’s age help you differentiate between glomerular diseases?
- Do extra renal symptoms help us in the diagnosis?
- How can serology help us in our diagnosis?
- When do we see hypocomplementemia?
- Does the progression of the disease and the accompanying creatinine levels help us in our diagnoses?
- When is a kidney biopsy indicated?
- Are there contra-indications for a biopsy?

Introduction

Students and beginning doctors often find it difficult to navigate through the vast amount of glomerular diseases and their diverse causes and presentations. This makes it difficult for them to make a differential diagnosis. It can, however, be of great importance to quickly be able to differentiate between several groups of glomerular disease, especially in diseases with an acute onset and progression. Delay of treatment can cause permanent damage to the kidneys and higher treatment cost.[1]

When considering a glomerular disease, specific renal symptoms can be a sign of glomerular damage. Symptoms such as haematuria, foamy urine, oedema, anaemia, changes in the colour and smell of urine are signs of a renal component in the patient’s possible disease.[2] Glomerular diseases are often discovered in general health screenings because the proteinuria or haematuria accompanying the disease are usually asymptomatic in onset.[3] However, glomerular disease can have an acute presentation.[2] To further confirm renal involvement, the presence of haematuria and serum creatinine can be determined. One should note that these values do not always deviate from a normal range in glomerular diseases.[2]

Another aspect that could be observed is the amount of erythrocytes and protein in the urine. Haematuria is often a symptom of renal and glomerular diseases, but haematuria can also be a symptom of damage in the lower urinary tract. However, if more than 40% of the erythrocytes in the urine are dysmorphic in shape, mainly acanthocytes, or if erythrocyte casts can be found in the urine, a glomerular disease is likely.[4,5] Loss of albumin or larger serum proteins in the urine is a typical symptom of glomerular disease.[4]

One should keep in mind that there are many systemic diseases that have a secondary focus in the kidney and that not all of these patients will have kidney problems. It is however possible that systemic (autoimmune) disease can lead to, or has already caused, kidney damage.[2] Systemic disease may also present primarily with kidney involvement. It is very important to recognize these systemic syndromes because it may influence the diagnosis and prognosis of the patient.

This overview of recent literature aids the inexperienced in making a differential diagnosis when suspecting glomerular disease in a patient. We have attempted to categorize the glomerular diseases by utilizing clinical symptoms and results of diagnostic tests based on recent literature. This overview will quickly give the reader insight in which diseases could fit a patient’s clinical presentation.

In this review we discuss the most common glomerular diseases such as membranous nephropathy, minimal change disease, rapidly progressive glomerulonephritis, focal segmental glomerulosclerosis, IgA nephropathy and diabetic nephropathy and the glomerular diseases caused by hereditary predisposition, infections, vasculitides, systemic diseases and auto-immune diseases.
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Which glomerular diseases should be considered when there is isolated haematuria?

Haematuria can occur in some cases of glomerular disease. Haematuria is determined by using a dipstick. When speaking of isolated haematuria one can differentiate between macroscopic and the more common microscopic haematuria.[2] Microscopic haematuria can only be determined using a microscope or a dipstick and has a prevalence of 2.5% in the overall population.[3] The most common glomerular causes of isolated haematuria among patients younger than 40 are IgA nephropathy and thin basement membrane disease. [3] Alport’s disease can also present with haematuria and progressive proteinuria at a young age.[7] The haematuria in IgA nephropathy and Alport’s disease can be both microscopic as well as macroscopic.[7,8,9] Other important glomerular diseases presenting with haematuria are the diseases in the group of rapidly progressive glomerulonephritis, but usually these diseases also have proteinuria.[10]

Which glomerular disease should be considered when there is isolated proteinuria?

Nephrotic diseases often present with proteinuria but without haematuria.[2] The most common glomerular diseases without haematuria are minimal change disease, focal segmental glomerulosclerosis and diabetic nephropathy. [2,10,11,12] Amyloidosis can present without haematuria as well.[11,12] The absence of isolated haematuria allows us to exclude several diseases: IgA nephropathy always presents with haematuria and if boys below the age of 10 do not develop haematuria, the diagnosis of Alport’s syndrome is highly unlikely.[7,8]

The presence of proteinuria is to be determined by using a dipstick. When the dipstick is positive, there is an indication to determine the protein concentration in a single urine sample or a 24-hours urine collection. Less than 150 mg of protein in the 24-hours urine is considered normal.[2]

Does the amount of proteinuria help reach a diagnosis?

Proteins in the urine are often a sign of glomerular damage and the amount of proteinuria can help differentiate between glomerular diseases. Proteinuria of more than 3.5 grams a day is called nephrotic proteinuria.[2] Nephrotic proteinuria can lead to the nephrotic syndrome which involves oedema and hypoalbuminemia.[2] According to Koenig and Bolton the nephrotic syndrome in adults is caused by membranous nephropathy (MN) in 33% of the cases, focal segmental glomerulosclerosis (FSGS) in 33%, IgA nephropathy in 10%, minimal change disease (MCD) in 15% and membrano-proliferative glomerulonephritis (MPGN) in 2-5%.[1] Although the amount of protein narrows the differential diagnosis, it might not help to differentiate between these underlying diseases. MN for example, can also be caused by many underlying diseases.[1] The absence of symptoms of the nephritic syndrome, including edema and hypoalbuminemia, in a patient with nephrotic range proteinuria may point toward an secondary FSGS as a feature of chronic renal damage and hyperfiltration.

Does the combination of haematuria and proteinuria help in finding a diagnosis?

Most glomerular diseases present with a combination of both haematuria and proteinuria.[10] Therefore it is difficult to exclude certain diseases from the differential diagnosis based on the combination haematuria and proteinuria exclusively. The combination of proteinuria and haematuria is often seen when (extraenal) infection is involved.[10] We can however differentiate between the nephrotic syndrome with or without haematuria. The previous question showed that MN, FSGS, MCD, IgA Nephropathy and MPGN are the most common causes of the nephrotic syndrome.[10] FSGS and MCD commonly present without haematuria but MN, IgA and MPGN can present with haematuria, which makes these diseases more likely.[10] Clinical suspicion based on these two symptoms is often not enough to confirm a diagnosis.[10]

Does the patient’s age help differentiate between glomerular diseases?

Age is a helpful characteristic when differentiating between glomerular diseases.[10] Infection related glomerulonephritis is a disease that often presents itself shortly after a throat or skin infection in children between the age of 5 and 12 years old.[13] Another example is the classic children’s disease: the haemolytic uremic syndrome (HUS). Diarrhoea associated HUS mainly presents in young children who have had an Escherichia coli which produces Shiga toxin or streptococcus pneumonia infection. It is one of the most important causes of acute kidney disease in children and is important to be recognised promptly.[14] The most common cause of glomerular disease and nephrotic syndrome among children is the minimal change disease (MCD).[2]

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Nephrotic syndrome</th>
<th>Mild glomerulonephritis</th>
<th>Moderate-severe glomerulonephritis.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal Change Disease (MCD), Focal segmental glomerulosclerosis</td>
<td>IgA nephropathy, thin basement membrane</td>
<td>membranoproliferative glomerulonephritis</td>
<td></td>
</tr>
<tr>
<td>&lt; 15</td>
<td>FSGS, Mesangial proliferative glomerulonephritis (MPGN) Membranous Nephropathy (MN), preecclampsia, post-infectious disease, hereditary nephritis, Henoch-Schönlein purpura (mesangial proliferative glomerulonephritis)</td>
<td>IgA nephropathy, thin basement membrane</td>
<td>Post-infectious glomerulonephritis, lupus nephritis, rapidly progressive (crescentic) glomerulonephritis,</td>
</tr>
<tr>
<td>15-40</td>
<td>FSGS, MCD, diabetic nephropathy, IgA nephropathy, thin basement membrane</td>
<td>IgA nephropathy, thin basement membrane</td>
<td>Post-infectious glomerulonephritis, lupus nephritis, rapidly progressive (crescentic) glomerulonephritis,</td>
</tr>
<tr>
<td>&gt;40</td>
<td>FSGS, MN, diabetic nephropathy, MCD (age &gt;60 years), IgA proliferative glomerulonephritis, nephropathy, primary amyloidosis</td>
<td>IgA nephropathy, mixed cryoglobulinemia</td>
<td>fibribrally glomerulonephritis, membranoproliferative glomerulonephritis</td>
</tr>
<tr>
<td>(or the related disorder light chain deposition disease).</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: differential diagnosis of glomerular disease, Uptodate [8]
In adults however, membranous and diabetic nephropathy are the most common underlying causes of the nephrotic syndrome.[24,25] At older ages it is essential to consider malignancies as cause of glomerular disease. In patients, older than 60, light-chain deposition disease is the cause of the nephrotic syndrome in 15-20% of cases. Light chain deposition disease is common among patients with multiple myeloma or lymphoma.[27] Table 1 summarizes how the differential diagnosis of the nephrotic syndrome, mild glomerular nephritis (in which kidney function is normal) and moderate to severe glomerular nephritis (in which kidney function is impaired) differs between several age groups.[10,26]

**What is the importance of recognising extrarenal symptoms?**
It is important to conclude whether the glomerular disease is associated with a systemic disease before considering a primary glomerular disease. Extrarenal symptoms, signs of illness in other organ systems, in combination with a kidney problem often indicate a systemic disease and therefore serve as an important diagnostic tool. The secondary causes of glomerular disease are divided into the following categories: Systemic autoimmune diseases, infections, vasculitides, microangiopathy, diabetes mellitus and paraproteinaemia or amyloidosis. [6] Patients are usually diagnosed with these diseases before signs of renal involvement, but the disease can sometimes reveal itself as a renal disease.[6] Specific extrarenal symptoms will be discussed further on.

**Which autoimmune diseases can cause glomerular disease?**
The systemic autoimmune diseases that are associated with glomerular diseases are Systemic Lupus Erythematoses (SLE), several rheumatic diseases and cryoglobulinemia.[4] Patients with these disorders often exhibit typical symptoms in other organ systems such as joint pain, problems with mucous membranes, Raynaud’s phenomenon, butterfly rash (SLE) and sclerosis of the skin and other organs [6] Especially SLE is a common cause of glomerulopathies and can present with different kinds of glomerulopathies.[5] Seventy percent of SLE patients develop glomerulopathy and forty percent presents with renal symptoms.[6] Autoimmuneological phenomena can give a clue that there is an underlying systemic autoimmune disease (Box 2).[5]

**Box 2. Autoimunological phenomena**
- (mouth) ulcers
- UV sensitivity
- Raynaud
- Hair loss
- Skin abnormalities
- Sicca symptoms
- Uveitis

Goodpasture’s disease is a primary renal disease but may also present systemically causing haemoptysis, called Goodpasture’s Syndrome, a pulmonary-renal syndrome.[20]

**Which vasculitides may cause glomerular disease?**
Vasculitides may also be associated with glomerular disease. The vasculitides associated with glomerular injury are granulomatosis with polyangiitis (GPA, also known as Wegener’s disease), Micropolyangiitis (MPA), Eosinophilic granulomatosis with polyangiitis (EGPA, also known as Churg-Strauss syndrome) and IgA vasculitis (IGAV, also known as Henoch-Schönlein disease).[4]

These disorders are small vessel vasculitides. A vasculitis in the kidney usually leads to damage of the wall of the capillary loop and extracapillary glomerulonephritis.[5] These vasculitides usually present with typical extra-renal symptoms.[29] There are often skin abnormalities but each type of vasculitides has his own typical symptoms. GPA for example also presents with extrarenal symptoms in the upper respiratory system and IgA vasculitides usually presents one week after an infection. Especially patients with GPA have a high percentage with renal involvement (90%). [29] When vasculitides are suspected one should specify the types according to their symptoms or perform further research, for example a biopsy.

**What are clinical clues for microangiopathies?**
The category microangiopathies as a secondary cause of glomerular diseases contains a variety of diseases such as haemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation, malignant hypertension, as well as pre-eclampsia.[5] In these diseases or conditions damage occurs in the vascular endothelium and results in intimal fibrosis and/or thrombus formation in the smaller arterioles and glomerular capillaries (thrombotic microangiopathy). HUS typically presents with petechiae, hemotema or signs of thrombotic microangiopathy and patients may have a positive family history. TTP can differentiate itself from HUS by neurologic symptoms.[5]

**What is the presentation of Diabetic Nephropathy?**
Diabetic nephropathy typically presents with a nephrotic syndrome with diffuse glomerulosclerosis in patients who have had DM for more than 15 years. The disorder often shows slow progression.[5,39] When considering the diagnosis of Diabetic Nephropathy (DN) it should be taken into account that other kidney diseases are not excluded in diabetic patients. In early onset of DN, the decline of renal function is masked by hyperfiltration.[5] Patients with DM are often screened for kidney damage, therefore DN is often found before it becomes symptomatic.

Patients with DM type 1 often already have retinopathy and high blood pressure when they present with diabetic nephropathy.[5] In patients with DM type 2 this may not be the case and will more often present with vague symptoms and abnormal course of disease. Sometimes DM type 2 may present itself with proteinuria.[5]
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Which glomerular disease can be caused by paraproteinaemia and amyloidosis?

Patients with amyloidosis or paraproteinaemia can present with a nephrotic syndrome. The diseases in this category which lead to glomerular problems are primary (AL, ALH, or rarely AH) amyloidosis, monoclonal immunoglobulin deposition diseases (MIDD; light chain deposition disease, heavy chain deposition disease, and light and heavy chain deposition disease) and miscellaneous glomerulopathies (monoclonal cryoglobulinemia, proliferative glomerulonephritis due to monoclonal IgG deposition).[27] One should note however, that these conditions can also cause tubular and interstitial diseases, such as cast nephropathy, that may lead to renal insufficiency as a result.[27]

A typical feature of multiple myeloma is the presence of monoclonal light chain immunoglobulins (Bence Jones protein) in the urine which are produced by the myeloma.[27] It is important to realise that Bence Jones proteins can give negative dipstick results even while there is proteinuria. This is because the dipstick only measures albumin. The 24-hours urine collection will however show pathologic amounts of proteinuria.[4]

In patients with proteinuria but with no protein found using a dipstick the presence of a paraprotein should be ruled out. Paraproteinemnic renal disease should be specifically considered in older patients because the higher incidence of multiple myeloma and lymphoma.[27]

What are the hereditary components and other predisposing conditions (chronic infections) of glomerular disease?

Alport’s disease is a genetic disorder and is inherited in a X-chromosomal fashion in 80% of the cases.[8] Patients with Alport’s disease often present with sensorineural hearing loss and ocular abnormalities.[8] Alport’s disease is a glomerular disease as a result of a change in the collagen structure of the glomerular basement membrane (GBM), which is caused by a defect in the gene encoding for one of the chains of collagen IV. Because of the gene defects the permeability of the GBM changes which results in haematuria and proteinuria.[6]

Other causes of glomerular problems are certain (chronic) infections such as hepatitis and HIV. Hepatitis B, for example, can cause polyarteritis nodosa and membranous glomerulopathy. HCV often presents with a membranoproliferative glomerulonephritis.[28] However, not all these patients develop glomerular problems.[28]

HIV associated nephropathy (HIVAN) is often expressed as a focal sclerosing glomerulosclerosis (FSGS) associated with tubular micro cysts and interstitial inflammation.[29] The classic presentation of HIVAN, almost exclusively occurring in blacks, is often accompanied by significant proteinuria and rapidly progressive kidney disease with normal blood pressure and normal enlarged kidneys.

How can serology help reach a diagnosis?

Serology is a useful tool that will help us differentiating between several glomerular diseases. With serology it is possible to identify antibodies in the serum. Examples of relevant antibodies are: ANCA (Anti neutrophil cytoplasmic antibody), ANA (antinuclear antibodies) and anti-double-stranded DNA antibodies and antibodies directed at certain infections such as HBV, HCV and HIV.[10,16,17] The serum complement levels can be measured as well, but we will discuss this further below.

A combination of ANA antibodies and hypocomplementemia is suspicious for SLE. But even in the case of SLE, because the subtype of glomerulonephritis can only poorly be predicted, it is necessary to perform renal biopsy, because each subtype requires different treatment.[18] ANCA antibodies are measurable in patients with one of the many vasculitides such as granulomatosis with polyangiitis (GPA, Wegener’s disease), microscopic polyangiitis, Churg-Strauss, renal-limited vasculitis and drug-induced vasculitides.[10,19]

When patients show signs of alveolar bleeding (haemoptysis) anti-GBM disease should be considered, but be aware that this disease can present without pulmonary involvement. The presence of Anti-GBM disease can be diagnosed utilizing serology alone. An ELISA is enough evidence to diagnose the disease.[20] The presence of anti-PLA2 receptor antibodies has a high specificity for primary membranous nephropathy in nephrotic patients and may render a renal biopsy unnecessary.[30]

It should be noted that serology usually cannot give a definite diagnosis and a renal biopsy is still the best way to reach a definite diagnosis in a patient with suspected glomerular disease.[10]

Complement levels as a new diagnostic tool?

Recent studies show that some glomerular diseases are associated with a shift in complement levels. The serum complement levels can be measured in the blood. Hypocomplementemia is common in several glomerulonephritic diseases such as: Lupus nephritis, post-infectious glomerulonephritis, membranoproliferative glomerulonephritis and combined cryoglobulinemia.[10,21-23] The cause of hypocomplementemia is the rapid activation of complement by immune depositions which cannot be matched with novo-synthesis of complement.[10,22,23]

IgA nephropathy, fibrillary glomerulonephritis and membranous Nephropathy show less activation of complement and therefore maintain normal complement values unless the patient has SLE or HBV.[21,23]

Does the progression of the disease and the accompanying creatinine levels help us reach a diagnosis?

Progression of kidney disease can be observed by looking at several factors such as the urine production, the amount of excreted proteins and the serum creatinine. The serum creatinine can be used as an estimation of the glomerular filtration rate (GFR) and therefore as an estimation of kidney function.[4] It is not always possible to use the serum creatinine levels because the serum level has a wide reference range and can be influenced by other factors, such as muscle mass.[2]

Renal biopsies that sometimes present with acute kidney failure are: concurrent acute tubular necrosis (especially in patient above 50 or with MCD), tubular injury in collapsing FSGS (idiopathic or HIV-associated), MCD with an acute interstitial nephritis (caused by NSAID use), crescentic
glomerulonephritis with membranous nephropathy and nephrotic syndrome due to immune complex depositions.[10]

Creatinine levels are more reliable in patients that have been monitored for a longer duration.[2] A fast (within days, weeks or months) increasing level of creatinine is a typical presentation of Rapidly progressive glomerulonephritis (RPGN). Because advanced RPGN leads to irreversible kidney damage, it is important to recognize RPGN in an early stage. [31] Rapidly progressive glomerulonephritis has a common presentation with acute macroscopic haematuria and oedema, but particularly presents itself with decreased kidney function which rapidly progresses in time[31,32]. It is necessary to identify whether a RPGN is possible considering the consequences, of failing to recognize the disease, are severe.[31,32]

There are diseases which present with mild symptoms and a normal kidney function. Especially IgA nephropathy, thin membrane disease and minimal change disease are diseases which can present very mildly.[10] Mild glomerulonephritis presents with a normal kidney function and normal creatinine levels. Mild glomerulonephritis can be caused (but not always) by IgA nephropathy, post-infectious glomerulonephritis, hereditary nephritis and lupus nephritis. However, these diseases do not always present with mild glomerulonephritis.[10] Table 1 shows the differential diagnoses of mild and moderate to severe glomerulonephritis.[10]

When is a kidney biopsy indicated?

A kidney biopsy is an important diagnostic tool in kidney disease and is often necessary to reach a final diagnosis. It is important to consider whether the result of the biopsy will influence treatment of the disease. It is only justified to perform a biopsy if the outcome will confirm diagnosis or influence the treatment.[9]

A biopsy should always be considered in context of the clinical symptoms and laboratory findings. This is important because a biopsy could incidentally be taken from a part of the kidney that is not affected by the disease, which could lead to a false negative biopsy (sample error).[33]

A biopsy is especially indicated in young adults with an idiopathic nephrotic syndrome. In this case, a biopsy can differentiate between MCD, FSGS or MN.[33] A biopsy should also be performed in patients with SLE and renal symptoms because SLE can lead to several different types of glomerular diseases.[18]

A renal biopsy can cause several serious complications. Bleeding is the primary complication of renal biopsy. Post-biopsy bleeding can occur at three sites: (1) into the collecting system, leading to microscopic or gross hematuria and possible ureteral obstruction; (2) underneath the renal capsule, leading to pressure tamponade and pain; or (3) into the perinephric space, leading to hematoctit formation and a possibly large fall in hematocrit.[34,35] These complications may result in silent hemotoma, hematuria (3.5%), erythrocyte transfusion(0.9%), angiographic intervention to control bleeding(0.9%), nephrectomy(0.02%) or even death (0.02%).

Risks factors for bleeding complications are hypertension (systolic pressure >130mmHg), age (>40 years) and of course coagulopathies.[35] Coagulopathies are therefore a contraindication for renal biopsy and the importance of the renal biopsy should be considered individually by case.[37]

Other complications of renal biopsy are pain lasting more than 12 hours (4%), arteriovenous fistulas (18%), chronic hypertension because of a large subcapsular hemotoma (rare), perirenal infection (0.2%) and rarely a function of the liver, pancreas or spleen may occur.[36] Because of these complications, a solitary native kidney has been considered an absolute contraindication to percutaneous biopsy. The concern is that marked bleeding may lead to nephrectomy and loss of all of the patient’s functioning renal mass.[37] If a renal biopsy is considered necessary when there is a solitary kidney, surgical biopsy can be considered.[36]

It is not necessary to perform a biopsy in patients with a very mild disease. A biopsy is also not indicated if proteinuria exists with less than 500 mg per day, if there is no haematuria, normal kidney function and no indications for systemic disease.[36,38]

| Clinical Lesson |

### Table 2 - Systemic autoimmune diseases

<table>
<thead>
<tr>
<th>SLE</th>
<th>Rheumatic diseases</th>
<th>Cryo–globulinemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Butterfly rash</td>
<td>- CREST-syndrome</td>
<td>- Purpura</td>
</tr>
<tr>
<td>- Chronic discoid lesions</td>
<td>- Raynaud’s phenomenon</td>
<td>- in cold environments</td>
</tr>
<tr>
<td>- Ulcers (oral &amp; nasopharyngeal)</td>
<td>- Symmetrical skin thickening</td>
<td></td>
</tr>
<tr>
<td>- UV sensitivity</td>
<td>- Skin abnormalities:</td>
<td></td>
</tr>
<tr>
<td>- Arthritis (without erosions)</td>
<td>- depigmentation, telangiectasia</td>
<td></td>
</tr>
<tr>
<td>- Serositis</td>
<td>- Myalgia</td>
<td></td>
</tr>
<tr>
<td>- Neurological disorders</td>
<td>(epilepsy, psychosis)</td>
<td></td>
</tr>
<tr>
<td>- Hematologic disorders</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>- Immunologic disorders</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>- Anti-nuclear antibodies</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>
Clinical Lesson

This overview has been based on textbooks, UptoDate, other individual disease prevalence as too low. Disorders as 'rheumatic disorders' because we considered the disease. Furthermore, we summarized several rheuma related disease that we decided not to include based on the rarity of the all existing glomerular diseases. Fibrillary glomerulopathy is a disease that we did not manage to include based on the rarity of the disease. It should be mentioned that we did not manage to include all existing glomerular diseases. Fibrillary glomerulopathy is a disease that we decided not to include based on the rarity of the disease. Furthermore, we summarized several rheuma related disorders as 'rheumatic disorders' because we considered the individual disease prevalence as too low.

This overview has been based on textbooks, UptoDate, other recent literature and clinical experiences. We emphasize that each patient should still be assessed individually.

**References**

Table 3 - Overview of vasculitides and their presentations

Clinical Lesson