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FOR PATIENTS

Understanding
IgA Nephropathy

What is **IgA Nephropathy?**

IgA Nephropathy (IgAN) is the most common form of **glomerulonephritis, an inflammation of the glomeruli,** the filtration units of our kidneys. The disease is triggered by processes in which the **antibody immunoglobulin A (IgA)** plays a central role.[1]

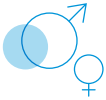
In the course of IgA Nephropathy, **so-called immune complexes are deposited** in the glomeruli. These deposits lead to inflammatory changes which can damage the kidneys and severely limit their function.[2,3]

Another name for IgA Nephropathy (from the Greek words "nephros", meaning "kidney", and "pathos", meaning "disease") is **Berger's disease.**[3,4]

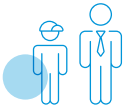
IgA Nephropathy **at a Glance**

IgA nephropathy (IgAN) is an orphan disease that **affects a total of 200,000 people** across the EU and UK combined[5,6]

IgAN is the **most common form** of glomerulonephritis[7,8]



It affects **twice as many men** as women[9]



Individuals **aged 16 to 35 years** develop IgAN most frequently[3]

What do our **kidneys do?**

Our kidneys perform different tasks:[10,11]

They **regulate the body's fluid balance**, affecting our **blood pressure.**

They **produce urine** and **excrete waste products** which were produced in our body or absorbed with food.

They **balance** the **body's acid-base metabolism.**

They **produce hormones** that our body needs to produce red blood cells, among other things.

They **regulate the concentration of various electrolytes** – substances such as sodium, potassium, and phosphate – which can be found disolved in body fluids, and serve important functions in metabolism.

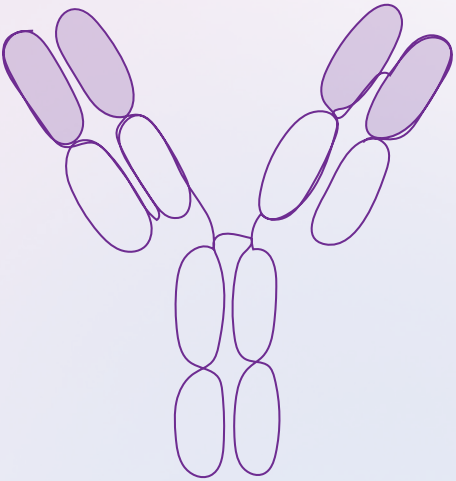
How does **IgA Nephropathy** develop?

The exact cause of the disease is still unknown. Experts assume that several factors come together to cause it, **including a genetic predisposition.**[1]

Immunoglobulin A (IgA) is also known to play a key role. This **protein is part of our immune system** and is responsible for fighting off pathogens, among other things. In people with IgA Nephropathy, structurally abnormal **IgA molecules enter the bloodstream** in large numbers. **The immune system recognises the abnormality and produces antibodies to bind and neutralise them.** These aggregates of antibodies are called **immune complexes.**[12,13]

Deposits of these **complexes can accumulate in the glomeruli,** potentially leading to inflammation of the kidneys, which can damage and scar the renal corpuscles. Kidney processes are disrupted, and as the disease progresses, the **kidneys lose the ability to fulfil their function** either partly or completely.

Possible consequences of IgA nephropathy include **permanent damage to the kidneys,** which can lead to **organ failure.** If this happens, **artificial filtration of the blood (haemodialysis)** may be necessary.[14]



What **signs and symptoms** can occur?

Symptoms of IgA Nephropathy may include:

visible or invisible blood in the urine (haematuria),

increased excretion of protein in the urine (proteinuria), detectable by foamy and/or cloudy urine,

high blood pressure (hypertension).

Patients do not always experience these or other symptoms of the disease. The disease often causes no symptoms, especially in its early stages.[1,3,14]

How is **IgA Nephropathy** diagnosed?

If IgA Nephropathy is suspected, the doctor will arrange for several tests. These include a **urine analysis in the laboratory.** If the urine contains **red blood cells** or if the **excreted amount of protein is increased,** this may be an indication for the disease. Some people also have **increased levels of IgA** in their blood.[1,2,15]

Often, IgA Nephropathy is detected by coincidence, for example if abnormal values are found in a routine urine test performed by the GP.[16]

However, abnormalities in the urine or blood are not sufficient for a definitive diagnosis of IgAN. This requires a **biopsy of the kidney, i.e. the examination of a tissue sample.** Based on the findings, the disease can be **divided into different severity levels.**[1,7,14]

What **measurements** are important?

During check-ups, your doctor will perform different tests. The results may help to better assess your individual risk of disease progression.

These measurements include, but are not limited to:

- **Blood pressure:** If protein excretion is less than 1 g/day, blood pressure should be less than 130/80 mmHg. If protein excretion is higher, levels should be less than 125/75 mmHg.[17]
- **The amount of protein excreted in the urine:** Proteinuria greater than 1 g/day, in addition to elevated blood pressure, is considered the most important risk factor for disease progression. Proteinuria suggests there might be damage to the kidneys. If the level is elevated compared to the previous examination, the damage is probably already more advanced.[7,18]
- **Glomerular filtration rate (GFR):** It describes the volume of blood filtered through the glomeruli per minute and is one of the most important parameters for assessing renal function. The normal value depends, among other things, on age.[17,19]

Other factors may increase the individual risk of disease progression, including, for example, **obesity, smoking, or lack of physical activity.**[20,21,22]

Is there a **typical progression?**

The course of IgA Nephropathy varies from person to person. **In some patients, it is stable for decades, while in others, loss of kidney function progresses steadily.** 25 % of patients develop end-stage renal failure within 20 years.[1,3]

Therefore, it is important to detect the disease as early as possible to initiate the appropriate therapy.

How do doctors treat **IgA Nephropathy?**

Recently, a new and targeted therapy has been made available in the UK for patients at risk of rapid disease progression under certain conditions.[1,3]

- For patients at **low risk, follow-up examinations** should take place **every 6 to 12 months.**[1]
- **Supportive measures** can be introduced to patients with persistent proteinuria of more than 0.5 g/day. The most important ones include taking **medication to lower blood pressure.** In addition, patients should aspire to a general **lifestyle change,** including more exercise and an adapted diet. More information on this can be found on the next page.[1,7]

If supportive therapies do not show the desired effect after three to six months, a **six-month therapy with glucocorticoids** can be initiated. In addition, it may be **useful to take fish oil.** The **omega-3 fatty acids** contained therein can help **to reduce inflammation.**[1,3,23]

If the disease has progressed to the point where the kidney function is severely impaired, **artificial blood filtration (haemodialysis) or a kidney transplant** may be necessary.[3]

Patients at risk of **rapid disease progression** may be eligible for a new and targeted therapy under certain conditions. To find out what suitable therapies are available to you according to your disease progression, please contact your treating physician.

What can you, **as a patient,** do?

Follow-up visits to the doctor and taking prescribed medications regularly are important components of living with IgAN. **Moreover, you can also contribute a lot to improving your health and maintaining your quality of life:**[7]

Try to **reduce any excess bodyweight** and exercise regularly.

A diet with **reduced salt** content can help lower high blood pressure.

If you smoke, you should **give up cigarettes completely, if possible.**

Protect yourself from infections. Vaccine protection also plays an important role. Make sure to have the **standard vaccinations refreshed regularly** and ask your doctor **which additional vaccinations may be useful for you.**

Connect with other people with kidney disease to **share your experiences.**

Ask your doctor for advice before starting a new exercise programme or changing your diet. They can also advise you on aids that can help you stop smoking.

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