CONDITION

Acute tubular necrosis

Learn how acute tubular necrosis can cause sudden kidney injury, including its symptoms, causes, diagnosis, treatment, complications, and follow-up care.

WHAT IT IS
Sudden injury to kidney tubules

BODY SYSTEM
Kidneys and urinary system

MAIN SYMPTOMS
Reduced urine, swelling, shortness of breath, confusion

WHAT CAUSES IT
Low oxygen or tubule-damaging substances

HOW IT IS DIAGNOSED
Combined blood and urine tests, sometimes kidney biopsy

MAIN TREATMENT
Supportive care and cause correction

CAN IT BE CURED?
Sometimes - kidney recovery may be incomplete

IS IT AN EMERGENCY?
Sometimes - severe trouble breathing

LONG-TERM OUTLOOK
Often substantial recovery, sometimes lasting kidney loss

Overview

Acute tubular necrosis is a type of sudden kidney injury. It affects the tubules, which are tiny structures inside the kidneys that help filter the blood and balance water, salts, and waste products.

The condition is often called ATN. Many kidney specialists now use the term acute tubular injury , because kidney tissue may be injured without widespread tissue death, or necrosis.

ATN is one of several possible causes of acute kidney injury , also called AKI. Acute kidney injury means kidney function has declined over hours or days. ATN is an "intrinsic" kidney injury, meaning the problem is within the kidneys themselves. Other forms of AKI may result from reduced blood flow to otherwise healthy kidneys or from a blockage in the urinary tract.

ATN often develops during a serious illness, major operation, severe infection, or period of very low blood pressure. It can also occur after exposure to substances that damage kidney tubules. Some people develop it while already in the hospital, although it can begin outside the hospital after an illness, injury, or toxic exposure.

The kidneys may recover if the underlying problem is treated and further injury is prevented. Recovery may take days to weeks and is not always complete. Careful monitoring matters because severe AKI can cause dangerous changes in fluid levels, potassium, and body acids.

Symptoms

ATN does not always cause obvious symptoms. The first sign may be a change in blood tests, especially a rising level of creatinine. Creatinine is a waste product normally removed by the kidneys.

Possible symptoms include:

Some people with ATN continue to produce a normal amount of urine. Normal urine output therefore does not rule out kidney injury.

Symptoms may also come from the illness that caused the kidney damage. For example, a person with a severe infection may have fever, low blood pressure, rapid breathing, or confusion. Someone with major muscle injury may have muscle pain, weakness, and dark urine.

- Producing less urine than usual
- Swelling in the feet, ankles, legs, hands, or face
- Shortness of breath from excess fluid
- Nausea, vomiting, or reduced appetite
- Unusual tiredness or weakness
- Difficulty concentrating, drowsiness, or confusion
- Dark, brown, or reddish urine when muscle breakdown or blood pigments are involved

Causes and risk factors

ATN usually develops when kidney tubules do not receive enough oxygen or are exposed to a damaging substance. More than one factor may be present.

Reduced oxygen delivery to the kidneys can occur during prolonged or severe low blood pressure. Possible situations include major blood loss, severe dehydration, septic shock, heart

failure, cardiac arrest, extensive burns, or complications of major surgery. A short, mild reduction in blood flow does not always cause ATN, but longer or more severe disruption can injure tubular cells.

Some medicines and chemicals can directly or indirectly injure the kidney tubules. Examples include certain antibiotics like amoxicillin or doxycycline , chemotherapy medicines, antiviral medicines, and toxic alcohols. The risk depends on the specific substance, the amount of exposure, kidney function, hydration, other medicines, and the person's overall health.

Iodinated contrast used for some CT scans and procedures has historically been linked with AKI. Current evidence suggests the risk of kidney injury directly caused by modern intravenous contrast is lower than once believed, especially in people without severe kidney disease. The expected benefit and kidney risk should be considered together rather than automatically avoiding a needed scan.

Pigments released into the blood can also damage tubules. Rhabdomyolysis is the rapid breakdown of muscle that releases a protein called myoglobin. It can follow a crush injury, prolonged immobility, extreme exertion, seizures, heat illness, or certain medicines and drugs. Extensive destruction of red blood cells can release hemoglobin and cause a similar problem.

Factors that may increase vulnerability include existing chronic kidney disease, older age, diabetes, heart or liver disease, severe infection, dehydration, and exposure to several kidney-stressing medicines at the same time. These factors do not mean a person will develop ATN.

Diagnosis

There is no single routine test that proves someone has ATN. Clinicians combine the medical history, physical examination, blood tests, urine tests, and imaging findings. They also look for other causes of acute kidney injury.

Blood testing tracks creatinine and measures potassium, bicarbonate, urea, and other substances that can change when the kidneys are not working normally. Creatinine may take time to rise after an injury, so repeat tests are often needed.

AKI is generally identified by a meaningful rise in creatinine over a short period or by low urine output. Clinicians interpret these changes in context because a person's baseline kidney function, muscle mass, fluid balance, and recent treatments can affect the results.

A urine sample may be checked for blood, protein, infection, and abnormal cells or particles. Under a microscope, ATN may produce tubular cells and dark, grainy structures commonly called "muddy brown" casts. Their presence can support the diagnosis, but their absence does

not rule it out.

Urine sodium measurements sometimes help distinguish reduced kidney blood flow from tubular injury. These results have important limits. Diuretics like furosemide or hydrochlorothiazide , chronic kidney disease, sepsis, contrast exposure, and other factors can make them difficult to interpret.

An ultrasound may be used to look for a urinary blockage and assess kidney size. It does not usually confirm ATN, but it can identify other explanations for AKI.

Clinicians also review recent illnesses, blood pressure changes, operations, imaging studies, supplements, nonprescription products, and medicines. Blood tests may check for muscle breakdown, infection, immune disease, or destruction of red blood cells when these are possible causes.

A kidney biopsy removes a small tissue sample for examination. It is not usually needed when the history and test results clearly fit ATN. It may be considered when kidney function does not improve as expected or when another kidney disease could require different treatment.

Treatment and management

Treatment focuses on correcting the cause, supporting the body while the kidneys recover, and preventing additional injury. ATN often requires hospital care, especially when kidney function is changing quickly or the underlying illness is serious.

Fluids may be given when low circulating blood volume is contributing to the injury. Fluid treatment must be individualized. Too little fluid can worsen kidney blood flow, while too much can cause swelling and breathing problems. People with shock may need medicines that support blood pressure as well as treatment for bleeding, infection, heart problems, or another cause.

The care team reviews all medicines, supplements, and recent exposures. A suspected kidney-damaging medicine may be stopped, replaced, or adjusted when medically appropriate. Other medicines may require lower doses because injured kidneys remove them more slowly. Patients should not stop an important prescription on their own unless a clinician directs them to do so.

Diuretics, sometimes called water pills, can help the body remove excess fluid when urine production is still possible. They do not repair injured tubules or reliably shorten AKI. Their role is mainly to manage fluid overload.

Blood tests and urine output are monitored closely. Treatment may also be needed for high potassium, excess acid in the blood, or complications of the underlying illness. Nutrition and

fluid intake may be adjusted with help from the medical team or a dietitian. Restrictions should be based on current laboratory results and fluid balance rather than followed without guidance.

Some people need temporary dialysis. Dialysis removes waste, potassium, acids, and extra fluid from the blood. It may be used when complications cannot be controlled with other treatment, such as:

The decision to start dialysis is based on the whole clinical situation, not on one creatinine result. Needing dialysis during ATN does not always mean it will be permanent.

- Severe or persistent high potassium
- Fluid buildup causing serious breathing problems
- Severe acid buildup
- Certain poisonings or medicine overdoses
- Symptoms caused by accumulated waste, such as inflammation around the heart or significant brain dysfunction

Complications

ATN can disrupt the kidneys' control of fluid, minerals, and waste. Fluid overload may cause widespread swelling, high blood pressure, or fluid in the lungs.

A high potassium level can affect heart rhythm. Acid may accumulate in the blood, and medicines cleared by the kidneys may build up unless their doses are adjusted. Severe waste accumulation, called uremia, can contribute to nausea, confusion, bleeding problems, or inflammation around the heart.

During recovery, urine output can increase before the tubules fully regain their ability to balance water and salts. This recovery phase may lead to dehydration or low levels of potassium, magnesium, or other minerals if losses are not monitored.

Many people regain much or most of their kidney function. However, ATN can increase the chance of later chronic kidney disease, another episode of AKI, or long-term loss of kidney function. The risk is greater after severe or repeated injury and when kidney disease was already present.

Follow up

Follow-up depends on the severity of the injury, whether dialysis was needed, and how close

kidney function is to its previous level. A clinician may repeat blood and urine tests, check blood pressure and fluid status, and review all medicines after discharge.

Ask before restarting medicines that were held during the illness. This may include certain blood pressure medicines, diuretics, or medicines affected by kidney function. Avoid starting pain relievers, supplements, herbal products, or high-dose vitamins without checking whether they are safe for the kidneys.

Routine follow-up may include monitoring creatinine, potassium, urine protein, and blood pressure. A kidney specialist may be involved when recovery is incomplete, the cause remains uncertain, dialysis was required, or there is significant underlying kidney disease.

Contact a clinician promptly for a clear decrease in urine, increasing swelling, repeated vomiting, worsening weakness, or difficulty keeping fluids down.

Seek emergency care for severe trouble breathing, chest pain, fainting, a seizure, new severe confusion, or an inability to wake normally. These symptoms can occur with serious fluid, electrolyte, or underlying medical problems and should not wait for a routine appointment.

Sources

- Kidney Disease: Improving Global Outcomes. "KDIGO Clinical Practice Guideline for Acute Kidney Injury." Kidney International Supplements . 2012;2(1):1-138.
- National Institute for Health and Care Excellence. Acute Kidney Injury: Prevention, Detection and Management . NICE Guideline NG148. Published December 2019; updated October 2024.
- Kellum JA, Romagnani P, Ashuntantang G, Ronco C, Zarbock A, Anders HJ. "Acute Kidney Injury." Nature Reviews Disease Primers . 2021;7:52.
- Davenport MS, Perazella MA, Yee J, et al. "Use of Intravenous Iodinated Contrast Media in Patients With Kidney Disease: Consensus Statements From the American College of Radiology and the National Kidney Foundation." Radiology . 2020;294(3):660-668.
- Perazella MA, Rosner MH. "Drug-Induced Acute Kidney Injury." Clinical Journal of the American Society of Nephrology . 2022;17(8):1220-1233.
- National Kidney Foundation. "Acute Kidney Injury." Patient education resource. Accessed March 8, 2025.