

SJS/TEN

A plain-English guide for you and your family

WHAT IS HAPPENING

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are two stages of the same dangerous reaction. A medicine you have been taking—often allopurinol (for gout), sulfa antibiotics, seizure medicines like carbamazepine or phenytoin, or certain HIV drugs—triggers your immune system to attack your own skin. It usually starts 1 to 3 weeks after you begin the drug, with fever, sore throat, and feeling like you have the flu. Then a painful red rash appears, quickly turning into blisters that burst and peel away in sheets, leaving raw skin underneath. Your mouth, eyes, and genital area often hurt and develop sores because the moist linings there are thinner and peel faster. This is a medical emergency. The team will stop the suspected drug right away, move you to a specialized unit (like a burn unit), carefully replace the fluids and salts you are losing through your damaged skin, keep you warm, protect your eyes, and watch closely for infection because bacteria can invade through the open wounds. Some people carry genes (like HLA-B*5801 or HLA-B*1502) that make this reaction more likely with certain drugs, so genetic testing before starting those medicines can prevent it. Recovery takes weeks, and scarring of the eyes or skin can happen, but stopping the drug early and getting expert care saves lives.

What you might feel

- Painful skin that feels like it's burning, followed by a red or purple rash that spreads quickly
- Large sheets of skin peel off, leaving raw, oozing areas (like a bad burn)
- Painful sores and blisters in the mouth, eyes, throat, and genital area
- High fever (often above 101°F) and feeling very sick—tired, achy, and weak
- Eyes that are red, burning, crusty, or stuck shut; light hurts to look at
- Trouble breathing or a harsh cough
- Peeing much less than usual, or dark urine
- SCORTEN score rises (a checklist that predicts how serious the case will be)

Call the doctor right away if...

- Skin peeling off in sheets when you barely touch it (Nikolsky sign)—this means the top layer of skin is dying and the condition is severe
- More than 10% of the body's skin is peeling or covered in raw, oozing areas (that size means TEN, the more dangerous form; under 10% is SJS)
- Trouble breathing, harsh cough, or hoarse voice—the airway lining may be peeling and swelling, which can block breathing
- Eyes crusted shut, severe pain with light, or vision getting blurry—permanent blindness can happen if the cornea scars
- Peeing much less, confusion, or very low blood pressure—the body is losing too much fluid and the kidneys and brain are in trouble

What the care team will do

- Stop the suspected trigger drug immediately and document the exact drug name, dose, and start date in the allergy list as a severe reaction
- Transfer patient to ICU or regional burn center and maintain strict temperature control with warmed room (86-90°F) and warming blankets because patient cannot regulate body heat
- Give intravenous fluids at 50-75% of Parkland burn formula (do not use full formula) and monitor urine output hourly to keep it above 0.5 mL per kg per hour
- Call ophthalmology (eye doctor) within 4 hours for eye exam and lubrication every 2 hours with preservative-free artificial tears, and avoid eye patches or anything touching the eye surface
- Perform gentle mouth care every 2 hours with saline rinses and apply petroleum jelly to lips; assess ability to swallow and coordinate speech therapy and nutrition consult for feeding tube if patient cannot eat
- Use non-stick dressings changed daily, avoid silver sulfadiazine cream if sulfa allergy suspected, and culture any areas with yellow drainage or foul odor before starting antibiotics
- Calculate SCORTEN score daily (checks age, cancer, heart rate, body surface area involved, blood sugar, bicarbonate, and urea) and notify physician if score increases
- Monitor temperature every 4 hours, draw blood cultures immediately if fever over 100.4°F (38°C) appears, and give broad-spectrum antibiotics within 1 hour if cultures are positive

Medicines you may be given — and why

Immediate withdrawal of the causative drug

The immune system is attacking the skin because it sees a medicine (or sometimes an infection trigger) as dangerous. When you remove that trigger drug right away, you cut off the signal that is telling the body to keep attacking. This stops new damage from piling up, though the reaction already started will take days to weeks to settle because the immune memory fades slowly.

Intravenous immunoglobulin (IVIG)

IVIG is a concentrate of antibodies collected from thousands of healthy blood donors. It works by blocking the death signals that activated T cells send to skin cells. Specifically, it covers up a molecule called Fas on the surface of keratinocytes so that Fas ligand (the 'kill' message from T cells) cannot dock and trigger cell suicide. It may also soak up some of the toxic proteins (granulysin, perforin) that punch holes in skin cells. This slows the wave of skin death and may shorten how long new blisters form.

Cyclosporine

Cyclosporine blocks calcineurin, an enzyme inside T cells that normally turns on genes for attack chemicals (like interleukin-2 and interferon-gamma). When calcineurin is blocked, T cells cannot multiply or send out the signals that tell them to kill skin cells. This dampens the immune attack at its source. Because SJS/TEN is driven by activated CD8+ T cells releasing granulysin and perforin, shutting down T-cell activation may slow the spread of new blisters and reduce the total area of skin loss.

Etanercept

Etanercept is a protein that acts like a sponge for tumor tissue death (necrosis) factor-alpha (TNF- α), a powerful inflammatory signal released by T cells and macrophages (scavenger immune cells) during SJS/TEN. TNF- α amplifies the immune attack and recruits more killer cells to the skin. Etanercept binds to TNF- α in the bloodstream and tissue fluid, preventing it from docking on cell surface receptors and triggering more inflammation. By soaking up TNF- α , etanercept may dampen the inflammatory storm and reduce the speed and extent of skin detachment. It is considered investigational — emerging case reports and small series suggest benefit, but it is not yet standard of care.

Systemic corticosteroids (NOT recommended as monotherapy)

Corticosteroids (such as methylprednisolone or prednisone) are powerful anti-inflammatory drugs that turn down many immune pathways at once. They enter cells and bind to glucocorticoid receptors, which then move into the nucleus and change gene expression —

reducing production of cytokines (inflammatory signals), shrinking the number of circulating immune cells, and dampening T-cell activation. In theory, this broad suppression could slow the immune attack in SJS/TEN. However, multiple retrospective studies and meta-analyses have found that using corticosteroids alone (without IVIG or cyclosporine) is associated with HIGHER mortality, longer hospital stays, and more infections, likely because steroids impair wound healing, raise blood sugar (which feeds bacteria), and broadly weaken the immune system without specifically blocking the key cell-death pathways (Fas-FasL, granulysin). For this reason, current consensus guidelines recommend AGAINST corticosteroid monotherapy. If steroids are used at all, they are given only in combination with IVIG or cyclosporine and only for a very short course to control the initial cytokine storm.

Where this information comes from

- British Association of Dermatologists (BAD) 2016 guideline
- EuroSCAR study (multinational SJS/TEN registry)
- American Academy of Dermatology
- Cochrane reviews on severe cutaneous adverse reactions

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