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SYSTEMIC CONTACT DERMATITIS

Ayu Yusriani Nasution*, Cut Putri Hazlianda
[1]Department of Dermatology and Venereology, Faculty of Medicine, Universitas Sumatera Utara, Jl. Dr. Mansyur No.5, Padang Bulan, Medan Baru, Medan, Sumatera Utara 20155, Indonesia
[2]Prof. Chairuddin Panusunan Lubis USU Hospital, Jl. Dr. Mansyur No.66, Merdeka, Medan Baru, Medan, Sumatera Utara 20154, Indonesia
*ayu.yusriani@gmail.com

ABSTRACT
Systemic Contact Dermatitis (SCD) is a delayed hypersensitivity reaction occurring in previously sensitized individuals following systemic re-exposure to specific allergen. Despite its clinical significance, SCD remains under-recognized, leading to diagnostic delays and higher disease burden. This review aims to summarize the current understanding of SCD and emphasizing its impact on patient outcomes and quality of life (QoL). This narrative review searched PubMed/MEDLINE, Scopus, ScienceDirect, and Google Scholar for articles published in 2019-2025. Eligible publications were reviews, observational studies, case reports or series, and textbook chapter. Selection disagreements were resolved through discussion. A total of 23 studies were included. SCD arises from type IV hypersensitivity mechanism, though mixed mechanism may occur in specific cases. The condition may manifest as widespread eczematous eruptions accompanied by systemic symptoms following oral, parenteral, or implant allergen exposures. Diagnosis primarily relies on clinical assessment and patch testing. Management focuses on allergen avoidance, corticosteroids, and immunosuppressive agents, while biologic and JAK inhibitor are emerging options for refractory cases. SCD constitutes a complex and often underdiagnosed form of contact dermatitis with significant clinical implications. Early recognition, precise allergen identification, and individualized management are critical for improving prognosis and QoL

Keywords: allergic contact dermatitis; contact dermatitis; hypersensitivity; systemic contact dermatitis

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INTRODUCTION

Systemic Contact Dermatitis (SCD) is a dermatologic condition in which it was characterized by a cutaneous and systemic hypersensitivity reaction in individuals who previously sensitized to a specific allergen via cutaneous portal of entry. Upon subsequent exposure to the same allergen or a cross-reactive compound, a secondary immune-mediated response may occur. The allergen exposure was obtained through systemic route, such as oral, intravenous, intramuscular, inhalation, transmucosa, or transcutaneous route (Aquino & Rosner, 2019; Zhang et al., 2025).

SCD is recognized as a potential cause of chronic or recurrent contact dermatitis, particularly in patients who are unresponsive to conventional topical or systemic therapies. This is especially relevant in cases involving dietary allergens, where individuals may inadvertently ingest their sensitizing allergen without awareness of what they are allergic to. SCD may also has clinical manifestation with both cutaneous eruptions and systemic symptoms. The systemic symptoms including nausea, vomiting, diarrhea, fever, malaise, headache, and arthralgia. The condition has been associated with a range of allergens such as metals (mercury, nickel, etc), pharmaceuticals, and food-derived substances like Balsam of Peru, as well as various botanical and herbal products.

Symptom onset typically occurs within 24 to 48 hours following systemic exposure to the allergen (Ahuja et al., 2024; Aquino & Rosner, 2019).

While SCD prevalence has no clear epidemiologic data, it is estimated that 20% of general population has hypersensitivity when they are in contact with associated allergen. Contact dermatitis has 2 large subtypes, such as irritant and allergic contact dermatitis. SCD has the same pathway and the same as allergic contact dermatitis, with the prevalence as much as 20% of all contact dermatitis cases. In all cases, 5% of the contact dermatitis patient tend to be chronic and disabling (Sedeh et al., 2022; Sedó-Mejía et al., 2020). The high DALY in SCD make the proper management of SCD important to improve patient quality of life (QoL). The best management for SCD is to avoiding contact with subsequent allergen. However, if the patient has been exposed to the allergen, the patient can be given anti-inflammatory drug, such as corticosteroid, anti-histamines, and calcineurin inhibitors. Monoclonal antibody and immunosuppressive drug can also be given, such as cyclosporine, mycophenolate mofetil, and azathioprine (Johansen et al., 2022; Tramontana et al., 2023). The disabling nature of SCD and the high tendency of patient unawareness to the diseases, while the main treatment is to avoiding contact with subsequent allergen make SCD important to be discussed. This study aims to broaden reader knowledge about SCD and how to improve SCD patients QoL.

METHOD

This study is a narrative review of the literature on systemic contact dermatitis (SCD). Literature was searched in [PubMed/MEDLINE, Scopus, ScienceDirect, and Google Scholar] for articles published from January 2019 to December 2025. Controlled vocabulary (MeSH) and free-text terms were combined with Boolean operators and truncation. The search query was ("systemic contact dermatitis" OR "allergic contact dermatitis" OR "contact dermatitis" OR "allergic contact dermatitis syndrome"), combined with theme-specific terms for prevalence, etiology (metals, foods, drugs), risk factors, pathophysiology, classification, diagnosis (patch testing), management, and prognosis. Because SCD-specific data on epidemiology, immunology, and treatment are limited, supplementary searches used "contact dermatitis" and "allergic contact dermatitis" as indirect evidence, including a search for Indonesian data. The study eligibility criteria was peer-reviewed articles in English or Indonesia, published within 2019-2025, narrative or systematic reviews, observational or epidemiological studies, case reports or case series, or standard textbook chapters addressing SCD or contact hypersensitivity related to metals, foods, or pharmaceutical agents, and if the full text was available. Conference abstracts, editorials or letters without original data, duplicates, and studies unrelated to contact hypersensitivity were excluded. Study that is earlier than 2019 was also excluded. The study screening was performed by AYN and checked by CPH. If there are any disagreements, it will be resolved by discussion. For each article, the year, journal, study design, allergen or trigger, and key findings were extracted by AYN. Findings were synthesized narratively and arranged thematically under prevalence, etiology, risk factors, pathophysiology, classification, diagnosis, management, and prognosis. A total of 23 studies was included.

RESULT

Prevalence

SCD has no clear epidemiologic data, but it is estimated that 20% of general population has hypersensitivity with some associated allergen (Sedó-Mejía et al., 2020). In contact dermatitis, as much as 15% cases reported, with medically confirmed about 8.3%. Irritant contact dermatitis (ICD) has larger prevalence compared to SCD, with ICD prevalence as much as 80% of all CD cases, and SCD with prevalence as much as 20% of all CD cases. In all CD cases, 5% CD patient can develop to chronic condition and disabling the patient (Sedó-Mejía et al., 2020; Tang et al., 2025). While in Indonesia there are no documented prevalence on SCD data, it is known that CD has high burden of disease. Skin disease is known to be one of the leading global burden of disease in population age group 5–19 years. While the highest DALY of skin diseases in Indonesia is atopic

dermatitis (100–200 per 100,000), CD also has high DALY, such as 30–40 per 100,000 (Damayanti et al., 2025; Prameswari et al., 2023; Suryani & Shofwati, 2023).

Etiology

The main group of allergens involved in SCD are metal, food, and pharmaceutical drugs. In metal allergen group, the most common allergen is nickel, mercury, and zinc. In the food allergen group, the most common allergen is balsam of Peru (BOP), aspartame, sorbic acid, and propolis. In pharmaceutical drugs, both topical and systemic route of drug can induce SCD. The most frequent topical drug to cause SCD is budesonide, bufexamac, dibucaine, chloramphenicol, diltiazem, tetracaine, acetarsone, and neomycin. While for systemic drug, the most frequent drug include mitomycin C, methylprednisolone, and hydrocortisone (Aquino & Rosner, 2019; de Groot, 2022). In the metallic derived SCD, in nickel, sensitization occurs through skin contact (jewelry, coins, piercings, orthodontic devices). Systemic exposure from foods (canned foods, chocolate, legumes, shellfish) or implants triggers dermatitis. In mercury, prior sensitization via topical antiseptics (mercurochrome, thimerosal) leads to SCD after systemic exposure (inhalation, ingestion from fillings, seafood). In zinc, exposure through dental fillings or industrial sources may trigger generalized dermatitis in sensitized individuals (Aquino & Rosner, 2019; Chamani et al., 2023).

In food derived SCD, BOP, the common fragrance and food allergen can cause flare-ups in sensitized patients. In aspartame, its metabolite (formaldehyde) may trigger SCD in formaldehyde-sensitive patients after high intake from diet drinks or chewable meds. In sorbic acid, in which it is food preservatives found in cheese, fruits, and toothpaste, can cause vesicular eczema or perioral dermatitis. In propolis, there is caffeate derivatives that can cause widespread dermatitis (Aquino & Rosner, 2019). In pharmaceutical derived SCD, SCD usually happened because of the absence of stratum corneum. Hence, there will be facilitated penetration of the drug and absorption from mucosae that can cause dermatitis and will be followed by skin eruption. There also seem to be fierce localized contact allergic reaction or extensive allergic contact dermatitis can stimulate the production of drug-specific T cells. The increased production of this cell will lead to its spreading to other skin location. This will lead to the eruption of SCD clinical manifestation (de Groot, 2022; Tramontana et al., 2023).

Risk Factor

The major risk factors for SCD include prior cutaneous sensitization, systemic re-exposure to known allergens (especially metals), presence of medical or orthopedic implants, dietary intake of allergenic substances, and environmental or occupational exposure to sensitizing agents. Clinicians should maintain a high index of suspicion for SCD in patients who presents with unexplained, recurrent, or widespread dermatitis, particularly when standard topical treatments fail or when a history of prior contact allergy or metal exposure exists (Iskandar et al., 2024; Jeong et al., 2025; Wu & Chang, 2023; Yang et al., 2022). The most established risk factor for developing SCD is prior cutaneous sensitization to a specific allergen. Once sensitization occurs, subsequent systemic exposure through oral, parenteral, or implanted routes can elicit widespread cutaneous reactions. In a 2022 case study (Yang et al., 2022), a patient developed diffuse dermatitis following total knee arthroplasty, with evidence suggesting metal ion release from the prosthesis as the immunologic trigger. This case show that exposure to metal implants, particularly those containing nickel, cobalt, chromium, and titanium, as systemic exposure, are capable to induce delayed hypersensitivity reactions in sensitized individuals (Yang et al., 2022). Systemic or generalized eczematous eruptions may also occur in patients with metallic implants even in the absence of local inflammation. It is thought that diffusion of metal ions or corrosion products from the implant can act as systemic haptens, initiating a delayed-type hypersensitivity reaction (Wu & Chang, 2023).

Dietary exposure also represents a significant risk factor for SCD, particularly in patients sensitized to dietary metals such as nickel. Ingestion of nickel-containing foods, such as cocoa, nuts, and

certain vegetables, can precipitate widespread eczematous eruptions in nickel-sensitive individuals. More recently, it is seen that occupational metal exposure, particularly to nickel, chromium, and cobalt, is a major cause of systemic allergic reactions. Persistent low-level exposure of these substance can sustain sensitization and contribute to systemic dermatitis manifestations (Iskandar et al., 2024). In addition to dietary and implant-related triggers, novel medical devices are emerging as risk factors for SCD. It is reported that case of systemic dermatitis following implantation of a spinal cord stimulation device, where systemic exposure to the device's metallic components, resulted in a generalized eczematous reaction. This shown that implantable technologies can be a potential trigger for systemic allergic responses, particularly in individuals with a known history of contact sensitization (Jeong et al., 2025).

Pathophysiology

There is still no clear understanding regarding SCD mechanism, the most accepted mechanism of SCD is thought to be mediated by type IV hypersensitivity reaction, in which it was similar to the pathomechanism of allergic contact dermatitis. After the allergen entry through non-invasive and invasive route, hapten-carrier complex is processed. Then it was presented to T helper cell by antigen presenting cell. It then led to the clonal expansion of CD4 T cell in local lymph nodes, which then will migrate to the skin and exacerbate the cytotoxic response. It's also seen that patient with SCD history had significantly higher fraction of CD3, CD4, and CD8 T cell, known as cutaneous associated antigen (CLA) than non SCD patient (Aquino & Rosner, 2019; Pan et al., 2021).

Besides that, mechanism there's also another proposed mechanism, such as the p-I concept, in which it describes the interaction between pharmacology drug with immunoreceptor. This concept hypothesized that the drug can bind directly to the T cell receptor, without being presented to major histocompatibility complex (MHC). This mechanism can also explain, why SCD reaction only observed in few hours till days, and why higher doses of the associated drug usually associated specifically with SCD (Aquino & Rosner, 2019; Rustemeyer, 2022). While most of the SCD mechanism represents type IV hypersensitivity reaction, there's also some evidence that suggest SCD has type III mechanism in unique cases. The rapid onset of cutaneous symptoms after systemic drug administration, combined with the detection of antibodies against hapten–albumin complexes, supports the possibility of mixed immune pathways in certain patients (Aquino & Rosner, 2019; Tanei & Hasegawa, 2022).

Classification

Prior to SCD, the term allergic contact dermatitis syndrome (ACDS) has been proposed to better describe the spectrum of reactions in patients who develop systemic or widespread skin manifestations following prior cutaneous sensitization to an allergen. This terminology provides a unified framework that encompasses both localized and systemic manifestations of allergic contact dermatitis (ACD), addressing the limitations of older terms such as SCD and Baboon Syndrome (BS). ACDS progresses through four clinical stages, depending on how far the allergic response has spread from the original sensitization site: Stage 1 (Localized ACDS): Typical localized allergic contact dermatitis, confined to the area of contact with the allergen, stage 2 (Regional Dissemination): The dermatitis spreads to nearby areas through lymphatic drainage, stage 3A (Hematogenous Dissemination): The allergen or immune mediators enter the bloodstream, leading to generalized skin lesions distant from the initial contact site, stage 3B (Systemic Reactivation): A previously sensitized individual develops a widespread eruption after systemic re-exposure to the allergen via ingestion, inhalation, or injection (Aquino & Rosner, 2019).

In this model, drug-related SCD occurring in patients with known sensitization represents either stage 3A or 3B of ACDS. However, drug-induced eruptions that occur without prior sensitization cannot be classified under ACDS, as they represent a different immunologic mechanism (Aquino &

Rosner, 2019). To describe disease without prior sensitization, it can be included in symmetrical drug-related intertriginous and flexural exanthema (SDRIFE). It can be used to describe a systemically induced drug reaction that clinically resembles SCD. There is five diagnostic criteria for SDRIFE:

1. Exposure to a systemically administered drug.
2. Sharply demarcated erythema in the gluteal or inguinal/perigenital areas (often V-shaped).
3. Involvement of at least one other intertriginous or flexural region.
4. Symmetrical distribution of lesions.
5. Absence of systemic symptoms (e.g., fever, malaise) (Aquino & Rosner, 2019).

DISCUSSION

Diagnosis

SCD presents with a wide clinical spectrum, and its diagnosis requires careful exclusion of other dermatologic conditions. The differential diagnosis is broad and includes infectious dermatoses, autoimmune bullous diseases, drug eruptions, other inflammatory skin disorders, viral exanthems, impetigo, perianal cellulitis, and staphylococcal scalded skin syndrome. Other differentials with similar localization include Hailey disease, pemphigus vegetans, inverse psoriasis, candidiasis, tinea cruris, AGEP, and SDRIFE. Clinicians should also differentiate SCD from allergic contact dermatitis (ACD) and irritant contact dermatitis (ICD) through a detailed clinical history, presentation, and assessment of exposure patterns, since these localized conditions can mimic early or localized forms of SCD. Specific allergy test can also help to differentiate between various differential diagnosis (Aquino & Rosner, 2019; Baruffi et al., 2020).

Patch testing (PT) remains a valuable diagnostic tool because of its adaptability to identify various potential allergens in SCD, although interpretation can be challenging. Several medications are commercially available as standardized patch test preparations, allowing for safe and reproducible testing. These include corticosteroids, antimicrobials, nonsteroidal anti-inflammatory drugs (NSAIDs), local anesthetics, and antihistamines. For other medications not commercially available, in-house preparations can be used following established guidelines. Pure drugs can be tested at a 10% dilution in petrolatum and 10% in alcohol. Commercial formulations should have any coating removed, then grounded into a fine powder, or mixed at 30% in white petrolatum or 30% in water. Besides drug, other allergen from food or metal can also be patch tested effectively (Aquino & Rosner, 2019; Baruffi et al., 2020).

For testing protocol and guideline, The European Society of Contact Dermatitis (ESCD) and The European Network on Drug Allergy (ENDA) has provide the guidance for drug patch testing. ESCD recommends performing patch testing 6 weeks to 6 months after complete resolution of the adverse reaction. The result should be taken at 20 minutes, day 4, and, if still negative, an additional reading at day 7. Meanwhile ENDA, suggests a slightly shorter interval of 3 weeks to 3 months post-reaction, with readings on day 2 and day 3 or 4 (Aquino & Rosner, 2019).

The gold standard diagnostic for ACD is patch testing, but in SCD, while there are already established standardized testing protocols, patch testing can present with negative and positive test. It can be caused by the lower absorption of the allergen from the skin. Therefore, patch testing has variable sensitivity and specificity in SCD patients. Some patients may have negative patch test results yet develop a positive reaction upon oral drug challenge, indicating that not all systemic hypersensitivity reactions can be reproduced via cutaneous exposure. Therefore, negative patch tests do not completely exclude drug hypersensitivity. Patch testing should always be interpreted in the context of the patient's clinical history, timing of reaction, and type of exposure (Aquino & Rosner, 2019; Baruffi et al., 2020).

Management

The most important therapeutic intervention in SCD is identification and elimination of the systemic hapten source. This can be conducted by stopping or avoiding the culprit drug, removing or replacing an allergenic implant or device when feasible, or dietary modification if a food or metal (nickel, cobalt) is implicated. If disease remains severe or recurrent despite avoidance, escalate to steroid-sparing systemic immunosuppressants (cyclosporine for short-term rapid control, methotrexate or azathioprine or MMF for longer steroid-sparing management) or consider referral for biologic or JAK therapy in refractory cases (de Groot, 2022).

For an acute SCD flare the immediate goal is rapid suppression of the T-cell driven skin inflammation and symptom relief. High-potency topical corticosteroids remain first-line for localized or moderately extensive disease (>20% body surface). Duration of the corticosteroid given can be short courses by using systemic oral corticosteroids to gain quick control. Sedating or non-sedating antihistamines are commonly prescribed for pruritus. Supportive measures, such as emollients, gentle skin care and avoidance of irritants should be implemented concurrently. These acute measures are standard in clinical reviews and practical guidance for SCD and ACD (Jacob et al., 2023).

When avoidance and topical therapy are insufficient, systemic immunosuppressants can be given in severe eczematous disease. Cyclosporine is commonly used for rapid control of severe eczematous flares, while methotrexate, azathioprine, and mycophenolate mofetil are used as steroid-sparing options for chronic disease. Biologics and JAK inhibitors have proven efficacy in atopic dermatitis and there are increasing case reports/series and reviews suggesting benefit in refractory contact dermatitis with systemic presentations. However, high-quality data specific to SCD are sparse, so these agents are considered off-label for SCD. It is usually used for refractory cases after failed conventional therapies attempt and after multidisciplinary discussion. When used, these agents require specialist oversight and the usual safety monitoring (Drucker et al., 2020; Tramontana et al., 2023).

Prognosis

The prognosis of SCD depends importantly on three main factors, identification and elimination of the systemic allergen source, the extent or severity of the skin reaction, and the patient's ability to avoid future exposures. If the systemic exposure is recognized and discontinued, many patients achieve marked improvement or resolution of dermatitis. Conversely, if the systemic source remains unidentified or exposures cannot be removed, the dermatitis can persist, relapse frequently, and becoming chronic or recalcitrant. Thus timely identification of the allergen and strict avoidance are key prognostic factors (Baruffi et al., 2020; de Groot, 2022; Pan et al., 2021).

CONCLUSION

SCD is a delayed-type hypersensitivity disorder that caused by systemic re-exposure to a sensitizing allergen. SCD contributes significantly to chronic dermatitis and impaired quality of life. It can be induced by diverse agents such as metals, pharmaceuticals, and dietary allergens, reflecting its complex pathophysiology. Early recognition and comprehensive management remain essential to prevent chronicity and enhance patient outcomes. Diagnosis relies on clinical evaluation supported by patch testing, though variable sensitivity and specificity requires careful interpretation. The main treatment for SCD is allergen avoidance, with corticosteroids, antihistamines, or immunosuppressive agents used to control inflammation. In refractory cases, biologic and JAK inhibitor therapies may offer emerging alternatives.

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