Carbamazepine-induced Stevens-Johnson syndrome in Chinese with negative HLA-A*3101 and HLA-B*1502 genes: A case report

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Abstract. Stevens-Johnson syndrome (SJS) is a severe, potentially life-threatening disease primarily induced by medications, which often involves lesions of the oral mucosa, eyes and skin. Its pathogenesis is partially associated with genetic factors. The present study reported a case of SJS following oral administration of carbamazepine (CBZ). A total of ~2 weeks after initiating the medication, the patient developed generalized skin rashes with pruritus, which progressively evolved into patchy erythema on the face, neck, chest, back, oral cavity and lips; oral herpes also emerged. As the condition advanced, the patient experienced blurred vision, along with rupture and exudation of the oral and labial herpes. The patient was diagnosed with SJS. However, genotyping results revealed a negative status for both major histocompatibility complex, class I, A (HLA-A)*3101 and HLA-B*1502 genes, indicating that the risk of SJS induced by CBZ cannot be excluded, even if the genetic test yields negative results. After confirmation of the diagnosis, CBZ was discontinued and the patient received treatment including intravenous dexamethasone. Significant clinical improvement was observed within ~10 days. The present study emphasized that the potential risk of CBZ-associated SJS cannot be ignored, despite negative results for both HLA-A*3101 and HLA-B*1502.

Introduction

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are acute drug-induced hypersensitivity adverse reactions that predominantly involve the skin and mucous membranes. Although rare, with an incidence of ~5.76 cases per million person-years (1), these conditions are life-threatening. Subsequent to its onset, the diseases progress rapidly, follow a severe clinical course and are associated with a relatively high mortality rate (2). Carbamazepine (CBZ) is a widely used broad-spectrum antiepileptic drug in clinical practice and it can also be used for the treatment of other diseases, including trigeminal neuralgia, glossopharyngeal neuralgia, bipolar disorder and manic episodes. Due to its definite efficacy and good tolerability, it is widely applied in clinical practice. However, with the increase in clinical application, cases of carbamazepine-induced SJS have been frequently reported in the literature (3-5). The role of genetic factors in the pathogenesis of CBZ-related SJS/TEN has attracted widespread attention, among which the polymorphism of human leukocyte antigen (HLA) genes is closely related to the occurrence of the disease. Studies have shown that major histocompatibility complex, class I, A (HLA-A)*3101 and HLA-B*1502 genotypes are important genetic risk factors for CBZ-induced SJS/TEN (6). The HLA-B*1502 genotype has the strongest correlation with CBZ-related SJS/TEN in Asian populations (7,8), while the HLA-A*3101 genotype is significantly associated with CBZ-related SJS/TEN worldwide, especially in Europe (9). At present, clinical guidelines recommend that before using CBZ, HLA-B*1502 genotype screening should be performed for Asian populations and HLA-A*3101 genotype screening should be performed for Caucasian populations to reduce the risk of CBZ-related SJS/TEN (10). However, clinical practice has found that certain patients with CBZ-related SJS/TEN have negative screening results for both HLA-A*3101 and HLA-B*1502 genotypes. Such cases suggest that the pathogenesis of CBZ-induced SJS is not only determined by the above two HLA genotypes, but may also involve the synergistic effect of various factors, including other genetic factors, environmental factors (such as combined medication, infection) and individual metabolic differences (11). The present study reported a case of CBZ-induced SJS in a Chinese patient with negative HLA-A*3101 and HLA-B*1502 genotypes. The innovation and uniqueness of the present study are mainly reflected in three aspects: Case specificity, research perspective and clinical value. In terms of case specificity, this study supplemented a rare case type and enriching the clinical spectrum of the disease. From the research and clinical perspective, this study analyzed a rare

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HLA double-negative case, and summarized the clinical features and core management strategies. It is helpful to deepen the clinical understanding of CBZ-induced SJS and offer a certain reference for rational clinical medication.

Case report

A 34-year-old female patient with blepharospasm started taking CBZ upon the recommendation of a neurologist to relieve the symptoms. The initial oral dosage was 100 mg twice daily, which was increased to 200 mg twice daily after 3 days. Following 2 weeks of CBZ treatment, the patient developed a generalized rash with pruritus, which was followed by the gradual emergence of macular erythema on the chest, back, oral cavity and lips, along with oral herpes lesions (Fig. 1). After 7 days, as the condition had progressed, the herpes vesicles ruptured and exuded fluid, leading to difficulty in opening the mouth and subsequent inability to eat. During the onset of symptoms, the patient did not present with any fever or other systemic manifestations, and prior to this episode, the patient had not taken any other medications or consumed any special foods. Upon admission to Wufeng Tujia Autonomous County People's Hospital in November 2025, based on the clinical manifestations, the patient was diagnosed with SJS. The Severity of Illness Score for TEN (SCORTEN) (12) was adopted to evaluate the patient, who obtained a score of 1, indicating a low risk of mortality. Genetic testing revealed a negative status for both the HLA-A*3101 and HLA-B*1502 genes. The genetic testing was performed using a real-time PCR with sequence-specific primer method. Genomic DNA was extracted from the patient's peripheral blood sample using a commercial DNA extraction kit (HiPure blood DNA mini kit; cat. no. D3111; Guangzhou Magen Biotechnology Co., Ltd.). The kit used for amplification and detection was TargetSeq® Target Probes (T216V1), manufactured by iGeneTech Biotech (Beijing) Co., Ltd. (cat. no. PT1001832; batch no. 69136402); the procedures were carried out on a real-time fluorescent quantitative PCR instrument (ABI 9700; Thermo Fisher Scientific, Inc.) following the manufacturer's standard protocol. Genotypes were determined based on the presence or absence of specific amplification signals for the target alleles. All the genetic testing work was performed by CRED Diagnostics in Wuhan (China). CBZ was immediately discontinued and the patient was administered 10 mg of dexamethasone via intravenous infusion. After 3 days, the patient's pain symptoms had improved. The dose of dexamethasone was then tapered to 5 mg for a further 3 days, after which the herpes lesions began to resolve and no new rashes or erythematous changes appeared across the patient's body. The dose of dexamethasone was then tapered to 3 mg for another 3 days before complete discontinuation. During the treatment, in addition to dexamethasone, analgesic drugs (oral ibuprofen 300 mg three times daily) were also intermittently administrated, the patient was supplemented with vitamin C, energy supplements, fluids and electrolytes and the patient was instructed to use mouthwash to maintain oral hygiene. After 10 days of treatment, the patient was discharged with significant improvement. The patient was advised to have adequate rest and follow a light diet within two weeks after discharge. Daily normal saline gargling was performed to keep the oral cavity clean. The skin and mucous membranes were maintained clean and dry to prevent wound damage and secondary infection. Regular hospital follow-up was arranged once a week.

Discussion

SJS, also known as erythema multiforme major drug eruption, is a rare type IV delayed hypersensitivity reaction primarily induced by drug factors (95%). In addition to characteristic bullous skin reactions and epidermal detachment, it often involves mucosal tissues, such as the oral cavity, eyes and genitourinary tract, and may also be accompanied by fever (13,14). The disease has an acute onset, rapid progression and potential for recurrence. It is highly prone to infections and systemic complications, and severe cases may even develop multiple organ failure leading to death. There are significant ethnic differences in the incidence of SJS, with Asians having twice the risk of developing the disease compared to Caucasians (15). Currently, the pathogenesis of SJS is not fully understood. Studies have confirmed that SJS is mainly caused by drugs (14,15), such as sulfonamides, antiepileptic drugs, non-steroidal anti-inflammatory drugs, allopurinol and nevirapine. Additionally, immune factors and genetic factors of the body also have a certain impact on the onset of the disease.

The major histocompatibility complex (MHC) is a group of highly conserved cell surface proteins. In humans, MHC is also referred to as HLA. The primary functions of the HLA gene system include participating in self-recognition, regulating immune responses and mediating rejection of allogeneic transplants (16). It is generally recognized that both humoral immunity and cellular immunity are involved in allergic reactions. Humoral immunity is dominated by immunoglobulin E-mediated allergic reactions, while cellular immunity is initiated through the T cell receptor-antigen peptide-MHC complex. In rare cases, certain drugs can induce immune responses by interacting with HLA molecules, resulting in adverse drug reactions (ADRs). Polymorphism in HLA genes increases the likelihood of off-target binding between HLA molecules and small-molecule drugs. These potential off-target interactions are associated with T cell-mediated ADRs; however, the mechanisms underlying most HLA-related drug interactions remain poorly understood. Immune responses triggered by different HLA-B genes may lead to dose-independent ADRs (17), such as SJS and TEN. Numerous studies have confirmed an association between the HLA-B*1502 gene and CBZ-induced SJS/TEN (18,19). In a study involving 44 patients with CBZ-induced SJS/TEN, all participants were found to carry the HLA-B*1502 allele. Subsequent studies verified this association between CBZ-induced SJS/TEN and HLA-B*1502 across diverse Asian populations, including those in China, Thailand, Malaysia and India (19). A meta-analysis across multiple populations revealed a significant association between HLA-A*3101 and CBZ-induced drug-induced hypersensitivity syndrome, however no significant association with CBZ-induced SJS/TEN (20). Another study indicated that HLA-A*3101 is a predictive marker for CBZ-induced allergic reactions in Europeans (5).

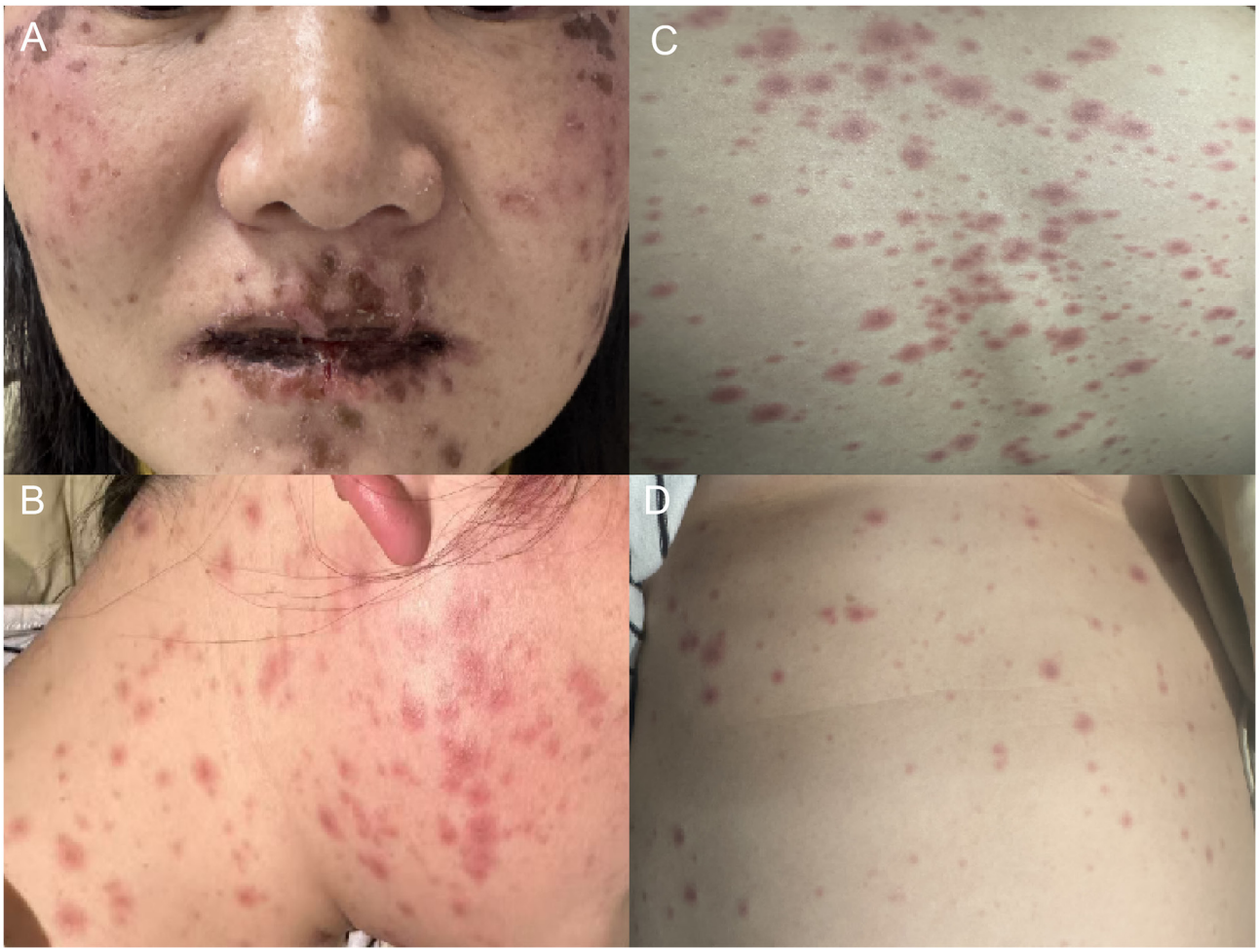

Figure 1. Presentation of the patient with mucocutaneous lesions. Multiple erythematous macular skin lesions were visible on (A) the face and lips, (B) neck, (C) back and (D) abdomen. Herpes with ulceration and crusting was noted on the lips.

These findings suggest that the type of cutaneous adverse drug reactions may be determined by genetic variations. Consequently, guidelines and drug labels recommend HLA-B*1502 genotyping for high-risk populations prior to CBZ administration. CBZ should not be used in individuals positive for this allele unless the potential benefits outweigh the risks (10). In the present case, the patient did not undergo genotyping before administration of CBZ. HLA-B*1502 and HLA-A*3101 genes were tested during the treatment and the results showed that both were negative. This result holds significant clinical significance, suggesting that the pathogenesis of CBZ-induced SJS is complex and diverse. In current clinical practice, positive HLA gene screening indicates a significantly increased risk of SJS following CBZ administration; however, negative gene screening cannot completely rule out the possibility of SJS occurrence. This case exemplified the following clinical scenario: Despite the absence of relevant risk gene carriers, the patient still developed SJS after CBZ treatment. This indicates that in addition to HLA-A*3101 and HLA-B*1502 genes, other genetic factors, environmental factors or individual metabolic differences may be involved in the pathogenesis of CBZ-related SJS. It also dispels the misconception that 'negative gene results ensure safe CBZ use' and provides important practical insights for clinical medication risk assessment.

The SCORTEN is widely used to predict the mortality of patients with SJS/TEN (21). SCORTEN should be assessed within 24 h of admission and re-evaluated on the 3rd day. This scoring system is based on seven independent risk factors: Age, malignant tumor, tachycardia, epidermal detachment rate, serum urea, serum glucose and bicarbonate levels (Table SI). The more risk factors present, the higher the mortality rate (Table SII).

Currently, there is no standard treatment protocol for SJS, and symptomatic and supportive care remains the mainstay of management. Intravenous immunoglobulin (IVIg) has been widely used in patients with SJS/TEN. Previously, the clinical benefit of IVIg in patients with SJS was controversial; however, recent studies have demonstrated that high-dose IVIg (<2 g/kg) exerts a positive effect on reducing the mortality of SJS/TEN (22). Similarly, the efficacy of systemic corticosteroids in SJS treatment is controversial. A major concern is that corticosteroid use in patients with SJS/TEN may increase the risk of infection, although some studies have also reported positive therapeutic effects (23,24). A growing body of evidence indicates that combined treatment with IVIg and corticosteroids, rather than IVIg monotherapy, can improve the prognosis of SJS/TEN (25-27). In addition, therapeutic plasma exchange (PE) and the double plasma molecular adsorption

system (DPMAS) are also potential treatments in SJS/TEN. The mechanism of PE is removal of drug, drug metabolites and cytokines from the patient. The Japanese guidelines recommend systematic steroids, IVIg and PE as the three first-line treatments of choice. Although certain studies have shown PE was effective in the treatment of SJS/TEN (28-30), its efficacy has been found to be controversial in clinical practice (31,32). A recent study showed no benefit of PE for reducing in-hospital mortality or the length of hospital stay (33). The DPMAS is a blood purification technique that employs various adsorbents to non-selectively bind and eliminate toxins or inflammatory mediators in the blood, thereby alleviating inflammation and regulating immune responses. A study reported that the levels of TNF-α and IFN-γ were elevated in patients with SJS/TEN and decrease significantly following DPMAS treatment (34). It is speculated that the adsorption effect of DPMAS is similar to that of TNF inhibitors, as it can reduce pro-inflammatory factors and thus inhibit the progression of SJS/TEN. Cyclosporine, a calcineurin inhibitor, can regulate T lymphocyte-mediated cytotoxicity and inhibit key molecules such as Fas ligand, NF-κB and TNF-α. Multiple case reports and meta-analyses have confirmed that cyclosporine treatment can improve the survival rate of patients with SJS/TEN (35,36). The complex pathogenesis and rarity of SJS/TEN pose significant challenges to its treatment. Although the aforementioned therapeutic approaches have demonstrated certain efficacy, limitations such as small sample sizes and the lack of randomized controlled trials have compromised the quality of relevant studies. Consequently, a definitive standard treatment for this disease remains unavailable to date.

Therapeutic intervention for the patient in the present case was the discontinuation of CBZ, combined with stepwise administration of dexamethasone (10 mg intravenously for 3 consecutive days, followed by 5 mg for 3 days and 3 mg for 2 days). Other supportive measures included analgesia, energy support, maintenance of water-electrolyte balance, keeping skin wounds dry and using mouthwash to maintain oral hygiene. The treatment achieved a significant effect: The systemic symptoms improved markedly after 10 days of hospitalization and was the patient was discharged successfully. The patient was treated with intravenous dexamethasone alone, without IVIg, for two reasons: Symptoms had peaked at admission (SCORTEN score 1) with no subsequent progression, reflecting low mortality risk, and the patient opted for monotherapy due to financial difficulties. Nevertheless, it may be reasonable to assume that IVIg should be prioritized in combination therapy when clinically indicated to ensure therapeutic efficacy.

Since this present study is a retrospective case study, it is necessary to point out that it has certain limitations. First, the present study is a single-case report, which only reports one Chinese patient with CBZ-induced SJS who is negative for both HLA-A*3101 and HLA-B*1502 genotypes. The small sample size limits the generalizability and representativeness of the research conclusions, making it impossible to fully reflect the overall clinical characteristics and pathogenic rules for such cases. Second, the present study only detected two genotypes, HLA-A*3101 and HLA-B*1502, and did not further explore the specific roles of other potential genetic factors involved in the pathogenesis, such as other HLA subtypes, environmental factors or individual metabolic differences. In the future, more cases of CBZ-related SJS with negative HLA-A*3101 and HLA-B*1502 genotypes will be collected, and integrated basic and clinical research will be further carried out to screen and verify other genetic factors, environmental factors and metabolism-related factors that may be involved in the pathogenesis of CBZ-induced HLA-negative SJS, so as to improve the pathogenesis system of CBZ-related SJS.

In summary, SJS is a severe disease primarily induced by drugs, and can be life-threatening. Genotyping for HLA-A*3101 and HLA-B*1502 should be conducted before initiating CBZ therapy, so as to assess the risk of SJS. It is still necessary to keep close clinical monitoring during and after CBZ administration even if the patient tests negative for HLA-A*3101 and HLA-B*1502 genes. On the one hand, SJS has an acute onset and rapid progression, and early identification and timely intervention are key to improving prognosis and reducing mortality. On the other hand, as shown in the present case, negative gene screening cannot completely avoid the onset risk; clinicians should not lower their vigilance, but inform patients to closely observe symptoms during medication (especially within 2-8 weeks, the high-incidence period of SJS) and seek medical attention immediately with drug withdrawal if abnormal manifestations occur to avoid delayed treatment.

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Availability of data and materials

All data generated in the study are included in the article.

Authors' contributions

All authors were involved in the patient's care. ZY and JL contributed to the conception, design and data collection. FY drafted the manuscript. ZY and JL confirmed the authenticity of all raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The present study was conducted according to the principles of the Declaration of Helsinki. The patient agreed to receive relevant clinical examinations including genetic testing.

Patient consent for publication

Written consent was obtained from the patient for the publication of clinical data and images in this academic paper.

Competing interests

The authors declare that they have no competing interests.

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