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Pancytopenia and Stevens-Johnson syndrome induced by oxcarbazepine: A case report

[1]Jialei Chen *MD*, [1]Wenguang Hu *MD*, [1]Mingping Lan *MD*, [2]Qun Yang *MD*

[1]Pediatric Neurology Department, Chengdu Women's and Children's Central Hospital, School of Medicine, University of Electronic Science and Technology of China; [2]Pharmacy Department, Chengdu Women's and Children's Central Hospital, School of Medicine, University of Electronic Science and Technology of China

Abstract

Oxcarbazepine (OXC), a commonly prescribed medication for focal and secondarily generalized seizure, has been associated with the development of pancytopenia in approximately 1% of patients. The incidence of Stevens-Johnson syndrome (SJS) in Han Chinese patients receiving OXC has been reported as 8 cases per 100,000 individuals annually. However, the simultaneous occurrence of both conditions is relatively rare. We report a case of pancytopenia and SJS induced by OXC. An 8-year-old Chinese female developed SJS after 9 days of initiating OXC therapy. On the 13th day of treatment, a complete blood count revealed pancytopenia. Prompt withdrawal of OXC, along with the administration of immunoglobulin, methylprednisolone, and supportive care, resulted in a favorable outcome with full recovery and restoration of normal hematological parameters. This case highlights the rare coexistence of pancytopenia and SJS induced by OXC in a single patient. Early diagnosis through heightened vigilance is crucial for achieving favorable outcomes. Prompt drug withdrawal and initiation of immunosuppressive treatments during the early phase can contribute to a good prognosis. Additionally, pre-exposure genetic testing and regular evaluation of the hematologic profile should be considered for patients undergoing OXC therapy.

Keywords: Oxcarbazepine, pancytopenia, Stevens-Johnson syndrome

INTRODUCTION

Oxcarbazepine (OXC) is widely used as an alternative to Carbamazepine (CBZ) for treating patients with focal and secondarily generalized seizure. In terms of reducing seizures, OXC has shown similar efficacy to CBZ but with fewer hematologic side effects and a lower incidence of Stevens-Johnson syndrome (SJS). Hematologic side effects, such as leukopenia, neutropenia, and thrombocytopenia, have been observed in patients prescribed OXC.[1] Two reports exist regarding pancytopenia induced by OXC.[1,2] SJS is a severe and life-threatening mucocutaneous reaction characterized by fever, maculopapular rashes, mucosal and variable internal organ involvement. Certain antiseizure medications (ASMs), including CBZ, lamotrigine, phenobarbital, phenytoin, and valproic acid, have been associated with the potential to cause SJS.[3] The variant allele *HLA-B*1502* is strongly associated with greater risk of SJS in patients treated with CBZ and OXC.[4] The annual incidence of SJS with CBZ is ten in every 100,000 newly exposed individuals.[5] Compared with CBZ, OXC has been rarely shown to induce SJS. In Han Chinese patients receiving OXC, the yearly incidence of SJS has been reported as only 8 cases per 100,000 individuals.[6] The co-occurrence of pancytopenia and SJS induced by OXC in a single patient is relatively rare. Here we present a pediatric patient of pancytopenia and SJS induced by OXC.

CASE REPORT

A 8-year-old Chinese female patient was admitted to our hospital presenting with a high fever and a 3-day history of multiple maculopapular eruptions. Previously diagnosed with self-limited epilepsy with centrotemporal spikes, she had been prescribed OXC with the initial dosage of 17.3 mg/kg/day 12 days ago. The dosage was increased to 28.8 mg/kg/day 5 days ago. After using this dosage for 2 days she developed spiking fever and

Address correspondence to: Jialei Chen, Pediatric Neurology Department, Chengdu Women's and Children's Central Hospital, School of Medicine, University of Electronic Science and Technology of China, No.1617, riyueroad, Qing Yang District, Chengdu City, Sichuan Province, China. Email: cjlxzz@sina.com

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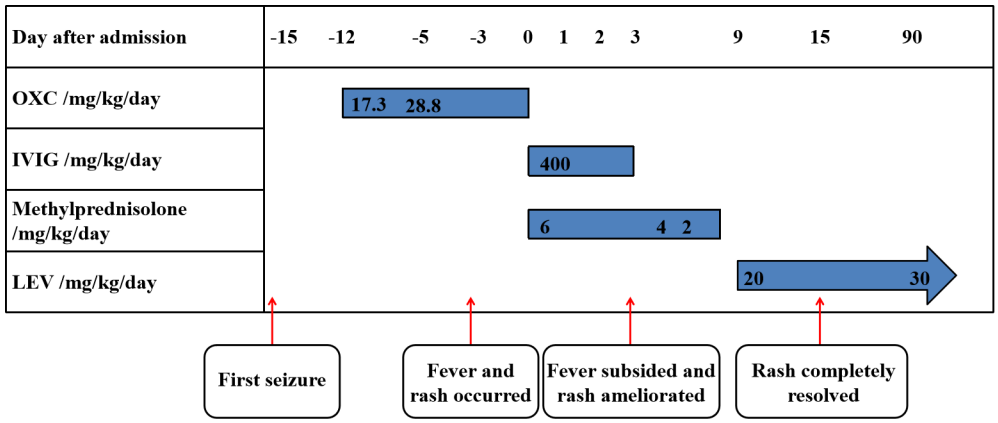

Figure 1. The patient's clinical events and treatment regimen (dosage of each drug indicated in the blue box).

maculopapular rashes around her mouth and lips (Figure 1). The maculopapular rashes gradually spread to her face, trunk, and limbs. There were bullous lesions and hemorrhagic crusts on her lips. The blisters broke, leaving painful sores that made it difficult for her to eat and drink. It is important to note that she had not taken any other medications besides OXC and had no prior history of adverse drug reactions. OXC treatment was immediately discontinued on admission.

During the physical examination, it was observed that the patient had diffuse red maculopapular rashes affecting various areas, including the lips, mucosa of the mouth and genitals, trunk, and extremities (Figure 2). Additionally, redness, blisters, and erosions were present on the lips and inside of the mouth. The positive Nikolsky's sign was also observed. Swelling was noted in the eyelids, face, and lower limb. Furthermore, some pinpoint-size petechiae were seen on the upper limb.

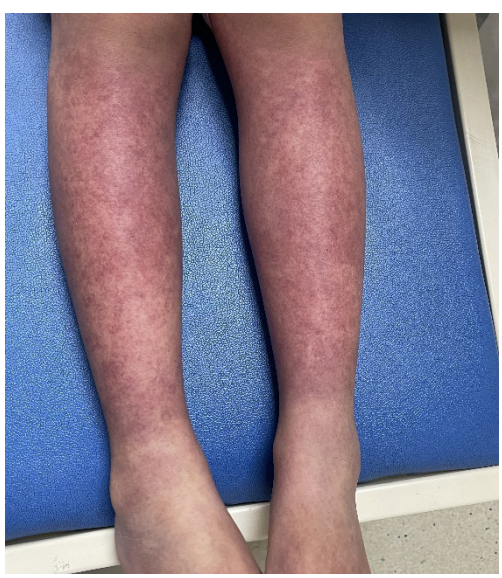

Figure 2. Maculopapular rashes over lower limb on admission.

On laboratory examination, it was found that the patient had an elevated level of C-reactive protein at 40 mg/L (reference range 0-10 mg/L) on admission. Mild elevations in serum alanine aminotransferase (48.4 U/L; reference range 7.0-40.0 U/L) and aspartate aminotransferase (53.1 U/L; reference range 13.0-35.0 U/L) were detected. The blood coagulation profiles were within normal limits. Serum levels of urea, bicarbonate, glucose, complement proteins C3 and C4 were all within the normal range. Serological tests for *Mycoplasma pneumoniae* antibodies, Epstein Barr virus antibodies, *Streptococcus pneumoniae* antibodies, and the human immunodeficiency virus antibody all yielded negative results. Blood culture did not reveal any positive findings. Prior to treatment with OXC, the complete blood count was normal. However, it revealed thrombocytopenia and anemia on admission (the 12th day of OXC treatment). On the following day, the patient progressed to pancytopenia, with a decrease in the white blood cell (WBC) count to 2.36×10^9 /L, neutrophil granulocyte count to 1.11×10^9 /L, platelet count to 50×10^9 /L, and hemoglobin level to 100 g/L (Table 1). The bone marrow examination revealed a reduction in thrombocytogenic megakaryocytes. Genotyping showed the presence of a *HLA-B*1502* allele.

The diagnosis of SJS was established based on the presence of skin lesions affecting less than 10 % of body surface area and involvement of two mucous membranes. Since no other medications were introduced during

the course of the illness, we concluded that both SJS and pancytopenia were induced by OXC. The patient received comprehensive supportive care, including skin and oral care, fluid compensation, and nutritional support. Additionally, intravenous immunoglobulins (IVIG) therapy (400 mg/kg/day) was given for 3 days. Subsequently, methylprednisolone (6 mg/kg/day) was intravenously administered for 4 consecutive days along with antihistamines. After discontinuation of OXC, the PLT and HB gradually increased, and the complete blood count revealed normal values rapidly on the second day after drug withdrawal. Her condition started to improve with the fever subsided and ameliorated maculopapular rashes three days after hospitalization. No swelling or petechiae were observed. Methylprednisolone treatment was continued followed with appropriate tapering by 2 mg/kg/day every 2 days. On the 9th day of hospitalization, significant improvement was observed and she was recommended levetiracetam (LEV) before discharge. During follow-up, she exhibited complete recovery (Figure 1), and the repeated complete blood count revealed normal values (Table 1).

DISCUSSION

ASMs have been associated with hematologic side effects, and several hypotheses have been proposed to explain how these drugs can lead to such side effects, including bone marrow suppression, destruction of immune system related cells, and direct marrow toxicity.[1] One of these mechanisms may be considered to be related to oxidative stress. The elevated levels of reactive oxygen species can cause DNA damages. ASMs may induce the hematological adverse reactions via oxidative stress on bone marrow hematopoietic progenitor cells. Valproic acid is a branched-chain carboxylic structurally similar to the fatty acid components of the cell membranes and may cause changes in the membrane's antigenic properties in a way that it becomes a foreign agent.[7] CBZ-dependent antiplatelet antibodies had been detected in a patient who developed thrombocytopenia ten days after starting CBZ.[8] Given the structural similarity of OXC to CBZ, it is conceivable that OXC might possess a potential for similar adverse effects on hematologic system.[2,9] The incidence of pancytopenia was reported to be 1 % in patients prescribed OXC, but only three cases evidenced OXC-associated pancytopenia in the literature.[1,2] In our case, the patient developed pancytopenia

Table 1: Complete blood count for a patient receiving OXC in treatment of self-limited epilepsy with centrotemporal spikes[a]

OXC treatment	WBC, $\times 10^9$/L (3.60-9.70)	Differential count, $\times 10^9$/L					Platelet count, $\times 10^9$/L (100-450)	Hemoglobin, g/L (120-146)
		Neutrophil (0.88-5.70)	Lymphocyte (0.68-6.21)	Monocyte (0.15-0.68)	Eosinophil (0.00-0.67)	Basophil (0.00-0.08)		
Before starting OXC								
Day 0	9.10	8.23	0.50	0.23	0.15	0.00	242	135
After starting OXC								
Day 12	4.56	2.69	0.94	0.55	0.36	0.01	55	107
Day 13	2.36	1.11	0.77	0.26	0.22	0.00	50	100
After stopping OXC								
Day 1	1.67	0.47	1.02	0.13	0.03	0.02	74	112
Day 2	3.79	1.31	2.10	0.35	0.01	0.02	143	124
Day 15	9.87	4.50	4.57	0.49	0.28	0.03	353	127
Month 3	5.76	2.20	2.98	0.44	0.12	0.02	291	126

[a] Reference ranges shown in parentheses

within 13 days after starting OXC at a dosage higher than recommended. After discontinuing OXC, her complete blood count gradually returned to normal. The temporal relationship between the onset of pancytopenia and the initiation of OXC, as well as the resolution of the abnormality upon discontinuation, suggests a cause-and-effect relationship between pancytopenia and OXC. Although the occurrence of pancytopenia in our case may be attributed to the inappropriate dosage of OXC, it is worth noting that pancytopenia has been reported to occur within 1 to 2 months after starting OXC with appropriate initial and titration dosages in other cases.[1] This highlights the importance of clinical vigilance for hematologic side effects and the regular monitoring of complete blood count during OXC treatment.

ASMs are known to be one of the main triggers of SJS, with CBZ being responsible for the majority of cases. OXC, although having a different metabolite pathway compared to CBZ, can also induce SJS due to the presence of the dibenzazepine ring in their molecular structure.[10] The incidence of SJS in Han Chinese patients receiving OXC therapy is rare, with a reported rate of 8 cases per 100,000 patients annually.[6] SJS typically develops within the first 4 to 40 days after initial exposure to OXC, mainly within 2 weeks.[4,11] The main clinical manifestations are rashes or maculopapules (100%), fever (83.3%), mucosal lesions (100%) and blisters (72.2%)[11](Table 2). The exact pathophysiological mechanisms of SJS are not fully understood[27], but there is evidence linking *HLA-B*1502* genotype with the risk of CBZ- and OXC-induced SJS.[4] Considering the consistent with the regional and ethnic distribution of the *HLA-B*1502* allele, pre-exposure *HLA-B*1502* testing has been implemented in Singapore and Thailand for use of CBZ. This is now also applicable to OXC. Pre-exposure genetic testing for *HLA-B*1502* has been recommended to reduce the incidence of SJS, especially in at higher risk populations in several Asian countries, including Vietnam, Cambodia, Reunion Islands, Thailand, some parts of India, Malaysia and China.[4] It has been postulated that metabolites, rather than the parent drug itself, are the causal agents. While there is evidence linking higher daily dosages of certain drugs to an increased risk of SJS[28], the relationship between OXC dosage and SJS has not been established.[15] Pancytopenia has been reported as a component of SJS in a few cases[29], but none of those cases were specifically attributed to OXC. The occurrence of pancytopenia in SJS patients may be related to direct antigen reaction with the cells or antigens affecting bone marrow functions.[30] After discontinuation of OXC, the PLT and HB gradually increased, and the complete blood count revealed normal values rapidly on the second day after drug withdrawal in our patient. However, within those 2 days after drug withdrawal, the symptoms of SJS were still obvious with repeated high fever and increasing rash. Therefore, in our patient, the pancytopenia is considered to be induced by OXC. Comorbidity of SJS may aggravate this process. Compared to CBZ, SJS induced by OXC tends to be less severe.[10] However, the co-occurrence of pancytopenia and SJS is particularly dangerous, and pancytopenia could be an early indicator for SJS complicated with other more lethal disorders.[31] Timely treatment is especially important for those patients. The management strategy still largely employed is a multidisciplinary approach starting from prompt drug withdrawal to supportive treatment. It was shown that IVIG (<2 g/kg) also had a beneficial effect in decreasing the mortality of SJS.[32] Although there is currently a lack of high-level evidence-based medical evidence to prove the efficacy of systemic immunotherapy in SJS, according to literature review and clinical experience, early and adequate glucocorticoids can alleviate the deterioration of the disease. And the combination of IVIG can reduce the dosage of glucocorticoids. Therefore, the expert consensus recommends IVIG can be used in dosage of 400 mg/kg/day for 3 to 5 days in SJS and prednisone can be given at 1.5 to 2 mg/kg/day for 7 to 10 days.[33] In our patient's case, the early administration of IVIG and short-term methylprednisolone led to a good prognosis.

In conclusion, the co-occurrence of pancytopenia and SJS induced by OXC can manifest in a single patient. This combination of conditions may indicate a more severe disease presentation. Early diagnosis through heightened vigilance is crucial for achieving favorable outcomes. Prompt drug withdrawal and initiation of immunosuppressive treatments during the early phase can contribute to a good prognosis. Additionally, pre-exposure genetic testing and regular evaluation of the hematologic profile should be considered for patients undergoing OXC therapy as preventive measures.

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Table 2: Clinical features of OXC-induced SJS

Patient#	Sex	Age (years)	Time of onset (days)	Clinical presentations				Genotype	Treatment	Prognosis
				Fever	Rashes	Mucosal lesions	Blisters			
1[12]	M	6	10	√	√	√	√	NA	topical methylprednisolone aseponat	symptoms improvement after 7 days
2[13]	M	10	9	√	√	√	NA	*HLA-B*1502*	methylprednisolone, azithromycin, cream urea, tobramycin-dexamethasone eyedrops	complete recovery after 15 days
3[6]	M	53	11	√	√	√	NA	*HLA-B*1502*	NA	NA
4[14]	M	9	14	√	√	√	√	*HLA-B*1518/B*4001*	steroid, antihistamine	symptoms improvement after 12 days
5[15]	M	21	14	√	√	√	√	NA	steroid, antihistamine	condition improved after 15 days
6[16]	F	10	22	√	√	√	√	NA	IVIG, clindamycin, fluid compensation	marked improvement after 8 days
7[17]	M	20	23	√	√	√	√	NA	IVIG	complete resolution in 1 week
8[18]	F	81	11	NA	√	√	√	NA	steroid, antibiotic, antihistamine	restore favorably
9[19]	F	18	7	NA	√	√	NA	NA	dexamethasone, pheniramine, ranitidine, paracetamol, clotrimazole local application	recovery after 12 days
10[19]	M	58	4	NA	√	√	NA	NA	dexamethasone, antihistamine, mupirocin 2%, gatifloxacin eye drops	recovery after 13 days
11[20]	F	38	10	√	√	√	√	*HLA-B*1518/B*4001*	dexamethasone, antihistamine	symptoms improvement after 10 days
12[21]	F	3	14	√	√	√	√	NA	IVIG, steroid, antihistamine	symptoms improvement after 10 days
13[22]	M	3	6	√	√	√	NA	NA	methylprednisolone, IVIG, antihistamine	complete recovery after 15 days
14[23]	M	8	8	√	√	√	√	*HLA-B*1502*	IVIG, methylprednisolone	complete recovery after 14 days
15[24]	F	6	14	√	√	√	√	NA	IVIG, steroid, antihistamine	symptoms improvement after 17 days
16[25]	M	29	40	√	√	√	√	*HLA-B*1502*	antihistamine	complete recovery after 1 month
17[26]	F	6	17	√	√	√	√	NA	steroid, antihistamine, antibiotic	complete recovery after 3 weeks
18 (this report)	F	8	9	√	√	√	√	*HLA-B*1502*	IVIG, methylprednisolone, antihistamine	complete recovery after 15 days

DISCLOSURE

Ethics: Written informed consent was obtained from the patient and her parents for publication of this case report and any accompanying images.

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