described in the Japanese population, several PP cases have also been reported in Western countries and, recently, in the Middle East.2,3

The pathogenesis of PP is not completely clear. In addition to being associated with several factors including exogenous (physical trauma, friction) and hormonal (pregnancy, menstruation), PP has classically been reported in association with metabolic derangements, especially ketogenic states (dieting, fasting, diabetes mellitus).2,3 Actually, several studies have detected elevated urine and/or blood ketone levels in patients with PP.2,3 In such circumstances, it is believed that ketone bodies may distribute around blood vessels leading to perivascular inflammation or enter into cells modifying their intracytoplasmic processes. The inflammation is believed to be mainly mediated by neutrophils: PP usually responds well to medications with antineutrophil effect, such as dapsone and tetracyclines, which would support this neutrophil-mediated theory. A role for decreased insulin levels, which is reported to occur after bariatric surgery,4 has also been hypothesized as cause of PP.2

In addition to its effect in changing the course of many skin diseases such as psoriasis, bariatric surgery has been associated with several dermatoses including bowel-associated dermatosis-arthritis syndrome, nutritional deficiency dermatoses, and alopecia.5 However, PP has never been reported after bariatric surgery. Given that such surgery may easily replicate the metabolic disturbance associated with other ketogenic states such as dieting or fasting,5,6 we believe that the association between PP and bariatric surgery may be underdiagnosed or underreported.

In conclusion, to our knowledge, this report is the first to describe PP developing after bariatric surgery, adding PP to the cutaneous complications of such procedures. Increased awareness of this rare entity and this association is important because bariatric surgery is a common procedure nowadays, and the metabolic abnormalities accompanying it mimic those that occur with other ketogenic states.

Mustafa Abbass, MS
Firass Abiad, MD
Ossama Abbas, MD

Author Affiliations: American University of Beirut Medical Center, Beirut, Lebanon.

Corresponding Author: Ossama Abbas, MD, Department of Dermatology, American University of Beirut Medical Center, Riad El Solh/Beirut 1107 2020, Beirut, Lebanon. PO Box 11-0236 (ossamaabbas2003@yahoo.com).


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Occurrence of Psoriasiform Eruption During Nivolumab Therapy for Primary Oral Mucosal Melanoma

The immunoinhibitory receptor programmed death 1 (PD-1) is expressed on antigen-stimulated T cells. The interaction between PD-1 and its ligands, which are expressed on dendritic cells, macrophages, and cancer cells, inhibits antitumor activity of cytotoxic T cells.1 A fully human anti–PD-1 antibody, nivolumab, has been approved in Japan for unresectable melanoma. We report a case of melanoma that responded well to nivolumab treatment, but the patient developed skin eruptions resembling psoriasis.

Report of a Case | An 80-year-old man had been receiving nivolumab therapy at 2 mg/kg every 3 weeks at another hospital to treat unresectable primary mucosal melanoma presenting on the upper lip, palate, and cheeks (Figure 1A). Prior to the therapy, he had no metastatic disease. He was previously healthy without personal or family history of psoriasis. The tumor on the lip enlarged over the course of the first 2 doses of nivolumab and then began to shrink after the third dose. Immediately following the fourth dose, the patient developed malaise, skin eruption, dysesthesia, and severe pain of the extremities. He was therefore referred to our hospital for evaluation of his systemic condition.

On admission, he had a low-grade fever, and the nodule on his lip was markedly reduced in size (Figure 1B). He had no gastrointestinal symptoms, and computed tomographic scans showed no metastatic lesions or interstitial pneumonia. Findings from neurological examination were unremarkable. Skin examination revealed asymptomatic, sharply bordered, scaly, erythematous plaques on the trunk and extremities, but eruptions having unclear borders or crusts were also seen (Figure 1C).

Routine laboratory test results were normal except for highly elevated C-reactive protein (CRP) (11.2 mg/dL; normal range <0.3 mg/dL). A skin biopsy performed on the day of admission revealed mild parakeratotic hyperkeratosis, irregular acanthosis, and moderate infiltration of mononuclear cells in the dermis (Figure 2). Some of the infiltrates tested positive for interleukin (IL)-17 or IL-23 by immunohistochemical analysis. The granular layer was absent in most areas.

On the third day after hospitalization, the patient developed a high fever, over 39.5°C. He was prescribed oral prednisolone (0.7 mg/kg), and the systemic symptoms and skin eruptions improved immediately. After termination of prednisolone treatment, the eruptions recurred along with increased CRP levels and a fever up to 37.8°C. Readministration of prednisolone (0.4 mg/kg) immediately resolved these symptoms, and at last follow-up he was taking 0.1 mg/kg of prednisolone. During the 3 months after the last dose of nivolumab, the lesions on the palate decreased in size. No melanoma cells were found in the biopsy from the upper lip.

Discussion | Previous clinical trials of anti–PD-1 antibody have demonstrated a potent antitumor activity for metastatic
melanoma. Approximately one-third of the patients showed regression of their lesions. Adverse events, most of which were mild to moderate, were observed in more than 80% of all patients. Cutaneous adverse events occurred in about half of the patients, which were categorized as rash, vitiligo, pruritus, and acneiform eruptions.

Our patient developed skin eruptions mimicking psoriasis, which has not been noted in previous reports to our knowledge. Psoriasis or psoriasiform eruption is well known to occur as a paradoxical reaction during biological therapies for severe psoriasis. This phenomenon is thought to be mediated by the increased production of interferons. Previous studies have demonstrated that blockade of the immune-checkpoint receptors, such as PD-1 and cytotoxic T-lymphocyte antigen-4, by its antibodies augmented the helper T cell type 1 (TH1) and TH17 cell activities, which might correlate with antitumor effect. The occurrence of the psoriasiform eruptions and systemic illness temporally coincided with the regression of melanoma lesions, suggesting strong correlation with nivolumab’s mechanism of action. Therefore, psoriasiform eruptions may be induced in cases of sufficient nivolumab antitumor efficacy. Further investigations are needed to clarify the relation between the cutaneous adverse events and antitumor activity of nivolumab.

Mikio Ohtsuka, MD
Takako Miura, MD
Tatsuhiko Mori, MD
Masato Ishikawa, MD
Toshiyuki Yamamoto, MD

Author Affiliations: Department of Dermatology, Fukushima Medical University School of Medicine, Fukushima, Japan.

Corresponding Author: Mikio Ohtsuka, MD, Department of Dermatology, Fukushima Medical University School of Medicine, Hikarigaoka-1, Fukushima, 960-1295, Japan (motsuka@fmu.ac.jp).


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Edema of the anterior neck, chin, and inferior cheeks (Sycosis vaccinatum) were present on the chin and jawline. Two weeks after initial presentation, pink depressed scars were present on the chin and jawline.

Discussion | We propose the term *sycosis vaccinatum* to describe the autoinoculation of vaccinia in the beard area with resulting viral folliculitis seen in the present case. To our knowledge, this reaction has not been discussed in the literature or reported to the vaccine adverse event reporting system (VAERS). In contrast to the term *vaccinia folliculitis*, *sycosis vaccinatum* describes the diagnosis and clinical presentation and avoids confusion with postvaccinial nonviral folliculitis (PVNVF).1,2

The course of sycosis vaccinatum parallels the progression at a primary vaccination site from vesiculopustule to crusted papule and ultimately to healing with a depressed scar. The timing of sycosis vaccinatum evolution is 7 to 10 days following vaccination, which is similar to that of PVNVF. However, sycosis vaccinatum is a localized form of autoinoculation, whereas PVNVF is a poorly understood idiosyncratic inflammatory or hypersensitivity response to vaccination.1,2

Findings of PCR, viral cultures, electron microscopy, immunohistochemical (IHC) analysis, and immunofluorescence (IF) studies are negative for the vaccinia virus in PVNVF but positive in sycosis vaccinatum.1,2 We confirmed sycosis vaccinatum by PCR, following the guidelines of the Centers for Disease Control and Prevention (CDC) for confirming a case of inadvertent autoinoculation.3 Histopathologic analysis in sycosis vaccinatum shows ballooning degeneration of keratinocytes within the epidermis with intracytoplasmic inclusions called Guarnieri bodies.2 An acute inflammatory cell infiltrate composed of neutrophils and lymphocytes extending into the epidermis is also characteristic. Use of IHC analysis and IF studies can also identify the vaccinia virus.3 The preferred diagnostic test is PCR because it is more sensitive than culture and does not require expertise in processing or interpreting electron microscopic specimens.

Treatment for sycosis vaccinatum is primarily supportive, involving local wound care and measures taken to prevent further inoculation (cessation of shaving, covering of the wounds, and avoidance of contact with other people). Sycosis vaccinatum requires VIG therapy if systemic symptoms are present, and

**Figure 1. Sycosis Vaccinatum, Clinical Findings**

Vesiculopustules with central umbilication in the beard area at time of presentation.