We describe the hypermetabolic skin lesions on interim (after two cycles) 2-deoxy-2-[18F] fluoro-D-glucose positron emission tomography/computed tomography (18F-FDG PET/CT) scan of a man with lung cancer who developed severe cutaneous hypersensitivity to paclitaxel.

CASE REPORT

Here, we present a case of a 49-year-old man with a history of right lung mass proven by biopsy to be a nonsmall cell lung cancer (squamous cell carcinoma) who developed HSR during therapy. In addition to the hypermetabolic primary malignancy, a positron emission tomography/computed tomography (PET/CT) scan showed multiple hypermetabolic skin lesions at several parts of the body. These cutaneous lesions were resolved in the restaging PET/CT scan performed after completion of the six cycles of chemotherapy. This is the first documented case of comparative PET/CT findings of a paclitaxel-induced hypersensitivity.

KEY WORDS: 2-deoxy-2-[18F]fluoro-D-glucose positron emission tomography/computed tomography, hypersensitivity reaction, lung cancer, paclitaxel
premedications. An $^{18}$F-FDG PET/CT scan performed 3 weeks after the second cycle of chemotherapy revealed multiple hypermetabolic skin lesions at several parts of the body including scalp, neck, chest, abdomen, and back, right inguinal region with maximum standardized uptake value of 4.31 [Figure 1, arrows] mimicking probable skin metastases in addition to hypermetabolic mass in the right lower lobe and hypermetabolic mediastinal lymph nodes. On physical examination, severe painful and pruritic rash on his scalp, neck, chest, abdomen, and back were inspected. It was retrospectively learned that he developed a widespread paclitaxel-induced drug eruption during the therapy within 2 days after the administration of the second cycle. Radiologic and clinical examination by a dermatologist indicated first the drug HSR as the underlying cause of these hypermetabolic skin lesions. Biologic tests revealed no autoimmunity. He reported not to have any skin lesion after the prior cycle of chemotherapy, and there were no skin lesions at the first $^{18}$F-FDG PET/CT performed for staging, as well. Based on the clinical course and physical examination, skin biopsy did not need to be performed. Successful symptomatic management with oral (dexamethasone 4 mg/day) and topical treatment produced prominent regression of the lesions. The patient was re-exposed to taxanes through desensitization protocol, and these allowed him to complete the six courses of therapy that includes paclitaxel. Subsequently, $^{18}$F-FDG PET/CT performed after 3 weeks after completion of six cycles of chemotherapy showed resolution of the skin lesions in addition to the regression in primary malignancy [Figure 2, arrows], which correlated with clinical resolution of the skin lesions without any scarring.

**DISCUSSION**

In view of the extensive use of paclitaxel in lung cancers, it is important to be aware of the possibility of drug hypersensitivity in these patients. The patients can have the reaction after the first or second course of therapy. HSR was developed within 2 days after the second paclitaxel infusion in our patient. $^{18}$F-FDG PET has been shown to be useful in the evaluation of many tumors including lung cancers. However, abnormal FDG uptake is not specific to malignancy, and inflammatory or infectious processes must be considered as well. Inflammatory cells (neutrophils and activated macrophages) at the sites of inflammation or infection will show increased FDG accumulation. Cutaneous $^{18}$F-FDG uptakes may be an incidental finding at PET/CT scans. Some benign skin lesions that show high $^{18}$F-FDG accumulations may mimic malignancies and lead to false positive interpretations. Abnormal skin uptake can be easily misinterpreted as skin metastases such as our case. As demonstrated in this case, benign skin lesions showing FDG uptake such as drug eruptions must be ruled out in patients with carcinoma.

Correlation with clinical findings can avoid false positive interpretation. In our patient, the additional finding of abnormal multiple skin uptakes of FDG in association with clinically evident rashes resulted in the correct diagnosis of drug hypersensitivity. He also had a dramatic response to the symptomatic therapy with oral and topical corticosteroids. Restaging $^{18}$F-FDG PET/CT scan performed
after completion of six cycles of chemotherapy showed resolution of these lesions.

Clinical and imaging findings together may aid the distinction of benign from malignant lesions, which is highly relevant to prognosis.

Adverse skin reactions associated with the use of paclitaxel have been reported in the literature. To our knowledge, comparative 18F-FDG PET/CT findings of paclitaxel-induced cutaneous hypersensitivity lesions have not previously been described.

CONCLUSIONS

The possibility of false positive inflammatory processes must be considered when abnormal skin FDG uptake is examined in 18F-FDG PET/CT scans. The presence of these hypersensitivity lesions should be taken into account during paclitaxel treatment. We would like to highlight the importance of correlation with clinical findings that can avoid false positive interpretation of these lesions.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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