

# Clinical Approaches to Minimize Rash Associated With EGFR Inhibitors

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**Purpose/Objectives:** To present a systematic approach for managing the skin rash associated with epidermal growth factor receptor (EGFR)–targeted therapies.

**Data Sources:** Clinical research literature, published abstracts, and clinical experience. The approach presented in this article is based on a combination of clinical experience and consultations with dermatologists, oncologists, and pharmacists familiar with EGFR inhibitor–associated rash.

**Data Synthesis:** A proactive approach that includes patient education and the use of a grade-based treatment algorithm. The goal of the approach is to minimize the effects of the rash on patients' quality of life and the course of cancer treatment.

**Conclusions:** Using the approach described in this article to treat the rash associated with the use of EGFR inhibitors, nurses can lessen patient discomfort and help ensure that patients will continue cancer treatment for as long as necessary.

**Implications for Nursing:** The approach described in this article should help nurses to recognize, grade, and treat the skin rash associated with EGFR inhibitors.

## Key Points . . .

- ▶ The clinical use of epidermal growth factor receptor (EGFR) inhibitors will continue to grow as more indications are approved, additional agents enter clinical trials, and new combinations of agents are studied.
- ▶ One of the most common adverse events associated with EGFR inhibitors is a skin rash that, although usually mild to moderate in severity, can negatively affect patients' quality of life and interfere with cancer treatment.
- ▶ The grading of rash can be subjective. Clinicians and patients should collaborate to determine how to treat rashes.
- ▶ Although no evidence-based treatment guidelines have been established for the treatment of the skin rash, this adverse event is manageable using the approach outlined in this article.

Clinical oncology recently has shifted from the use of traditional cytotoxic chemotherapeutic agents that target rapidly dividing cells to the use of therapies that target proteins implicated in the development and progression of cancer. Those proteins include Bcr-Abl fusion protein found in patients with chronic myelogenous leukemia, the vascular endothelial growth factor involved in the development of several solid tumors, and the epidermal growth factor receptor (EGFR) implicated in the development and progression of many different cancers. In contrast to chemotherapeutic agents, which can cause anemia, neutropenia, severe nausea and vomiting, neuropathy, and total alopecia, targeted therapies generally are well tolerated and have less severe systemic adverse events (Herbst & Bunn, 2003; Silvestri & Rivera, 2005).

Targeted agents are not, however, without adverse events. Agents targeted against EGFR have a distinct toxicity profile that includes diarrhea and various cutaneous toxicities, the most common of which is a rash that often is accompanied by dryness and pruritus. Although usually mild to moderate in severity, skin rash can have a significant negative effect on patients' quality of life. In addition to dryness and itching, which can be very uncomfortable, people often are self-conscious about the rash, which is frequently in highly visible areas such as the face, neck, and chest.

This article will focus on effective management of EGFR inhibitor–related adverse events, specifically rash, with an em-

phasis on maintaining patients' quality of life during treatment and limiting the effect of rash on the course of cancer treatment so that patients remain on it for as long as necessary.

## EGFR as a Target for Cancer Therapy

EGFR, also known as human epidermal receptor (HER) 1, is a member of the HER family of receptor tyrosine kinases, which also includes HER2, HER3, and HER4 receptors (Yarden, 2001). After binding their respective ligands (extracellular proteins that specifically bind to them), the receptors pair with each other as homodimers (e.g., EGFR-EGFR) or heterodimers (e.g., EGFR-HER2) and initiate a cascade of signals that direct a cell's growth, proliferation, response to other signals, and ability to move within tissue (Yarden & Sliwkowski, 2001). Consequently, the HER family receptor tyrosine kinases play important roles in the regulation of growth and differentiation in normal and neoplastic

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