Diagnosis of renal cell carcinoma (RCC) frequently occurs at advanced stages, severely limiting the success of treatment, and median survival is barely more than a year. Previously, treatment of renal cancer was limited to nephrectomy or immunotherapy (interleukin or interferon-α), which was effective in a small subset of patients but often was accompanied by severe side effects. New orally administered targeted therapies have become available, offering broader benefits to patients with advanced RCC. Sorafenib is an oral, multikinase inhibitor recently approved by the U.S. Food and Drug Administration as treatment for advanced RCC based on its extension of median progression-free survival from 12–24 weeks. Oncology nurses must ensure patient adherence and manage side effects of emerging treatments. This article reviews the management of skin rash, hand-foot skin reaction, hypertension, diarrhea, and fatigue in patients receiving sorafenib. In addition, a case study of a patient receiving sorafenib is presented.

Renal cell carcinoma (RCC) accounts for 3% of all malignant tumors and is the sixth leading cause of cancer deaths in the United States. In 2007, an estimated 51,190 new cases of RCC will be diagnosed and 31,590 deaths will be attributed to renal cancer (Jemal et al., 2007). The incidence of renal cancer at all stages has increased steadily since 1973; unfortunately, increased detection of earlier-stage disease has not coincided with a decrease in the number of patients diagnosed with advanced renal cancer (Hock, Lynch, & Balaji, 2002). For patients with localized disease, nephrectomy offered five-year survival rates from 90%–95% (Bui et al., 2001), but median survival among patients with metastatic disease was only 13 months (Cohen & McGovern, 2005).

Most renal cancers are sporadic: Risk factors include smoking (associated with 24%–30% of all cases of RCC), obesity, sedentary lifestyle, environmental and occupational exposure (e.g., asbestos, cadmium, polycyclic hydrocarbons, solvents), and long-term use of diuretics or phenacetin-containing analgesics (Linehan et al., 2004). Patients with end-stage renal disease undergoing dialysis, particularly those with cystic disease, also are at higher risk for RCC (Denton et al., 2002). A small number of cases are hereditary, associated either with the von Hippel-Lindau (VHL) gene in clear cell renal cancer or the c-met gene on chromosome 7 in type 1 papillary renal cancer (Linehan et al.). Individuals with VHL syndrome are at risk to develop tumors in multiple organs, including several hundred clear cell tumors per kidney.

Pathogenesis of Renal Cancer

Loss of VHL gene function leads to increased expression of genes associated with tumor growth and angiogenesis, especially...
vascular endothelial growth factor (VEGF) and platelet-derived growth factor-α (PDGF-α) (George & Kaelin, 2003), and may be responsible for approximately 60% of cases of sporadic clear cell renal carcinomas (Cohen & McGovern, 2005). In those cases, the maternal and paternal VHL alleles appear to have been inactivated by acquired mutations (George & Kaelin).

Figure 1 illustrates how the product of the VHL gene normally functions as a tumor suppressor by down-regulating the activity of the hypoxia-inducible factor–1α (HIF-1α) protein. By binding to HIF-1α, VHL tags HIF-1α for ubiquitination (i.e., the process of being tagged with ubiquitin, which marks HIF-α for destruction), leading to HIF-1α degradation in the proteasome (Cohen & McGovern, 2005; George & Kaelin, 2003), thus preventing over-production of hypoxia-induced growth factors such as VEGF, PDGF-β, transforming growth factor α, and erythropoietin. In hypoxic conditions (i.e., the absence of VHL regulation), growth factors can promote angiogenesis and tumor growth, and evidence suggests that reintroducing VHL protein can inhibit RCC growth in vitro (Cohen & McGovern).

Clinical Presentation and Diagnosis

Renal cancer most often is diagnosed in individuals older than age 40, with a male-to-female ratio of 1.6:1 (Jemal et al., 2007). A classic triad of symptoms typically exists at presentation: flank pain, hematuria, and a palpable abdominal mass. Symptoms leading to the diagnosis may be associated with the primary tumor, metastatic disease, or paraneoplastic syndromes (Kim, Lam, & Belldegrun, 2006). Approximately 50% of renal tumors are discovered on radiographic examination for another medical condition (Cohen & McGovern, 2005; Kim et al., 2003).

Metastasis may be present at diagnosis (20%–30% incidence) or develop following surgical intervention for localized disease (20%–40%) (Janzon, Kim, Figlin, & Belldegrun, 2003). Renal cancer metastasizes to various sites (e.g., lung, lymph nodes, liver, bone, brain), resulting in metabolic or clinical complications that affect treatment selection and quality of life (Kim et al., 2006).

Staging and Prognostic Indicators

RCC is diverse, with distinct histologic characteristics that can differentiate treatment approaches and prognosis (Atkins et al., 2004; Motzer et al., 1999). Most cases of RCC (85%) are adenocarcinomas (National Cancer Institute, 2007). Table 1 describes the histologic subtypes of renal cancer as well as tumor location and genetic abnormalities (Cohen & McGovern, 2005; Linehan et al., 2004; Reuter & Presti, 2000).

Predictive models typically incorporate tumor-node-metastasis staging, which evaluates cancers on the basis of the primary tumor’s spread, the presence of metastatic disease, and prior radiation therapy (Lam, Shvarts, Leppert, Figlin, & Belldegrun, 2005; Mekhail et al., 2005; Zisman et al., 2002) (see Table 2). The National Comprehensive Cancer Network (2006) published revised practice guidelines for kidney cancer based on tumor-node-metastasis staging. Patients also can be classified using the Memorial Sloan-Kettering Cancer Center criteria (known as the Motzer score), which assigns patients to favorable-, intermediate-, and poor-risk groups based on performance status, lactate dehydrogenase levels, corrected serum calcium level, hemoglobin level, and nephrectomy status (Motzer et al., 1999). The Fuhrman nuclear grading system evaluates tumor cells by nuclear size, contour, and characteristics of nucleoli; increasing

Figure 1. Regulation of Hypoxia-Inducible Factor-Driven Gene Expression by Oxygen Levels and Von Hippel-Lindau

LOW-OXYGEN (HYPOXIA) OR INACTIVE VHL
In low-oxygen conditions or when pVHL is inactive, HIF-α is able to bind to HIF-β and activate transcription hypoxia-inducible genes.

Trigger transcription of hypoxia-inducible factors to angiogenesis

OXYGEN PRESENT OR FUNCTIONING VHL
When oxygen is available and the pVHL is active, HIF-α is tagged with Ub, which marks it for destruction.

EPO—erythropoietin; HIF—hypoxia-inducible factor; PDGF—platelet-derived growth factor; pVHL—von Hippel-Lindau protein; TGF—transforming growth factor; Ub—ubiquitin; VEGF—vascular endothelial growth factor; VHL—von Hippel-Lindau

Note. In hypoxic conditions or when VHL is inactivated, the transcription of HIFs (VEGF, PDGF-β, TGF-α, EPO) promotes tumor growth and angiogenesis. This process results in the high vascularity of renal tumors and the increased potential for tumor invasion of adjacent structures and metastasis.
abnormalities are associated with poorer prognoses (Fuhrman, Lasky, & Limas, 1982; Reuter & Presti, 2000). In a multivariate analysis, only Fuhrman grade, Eastern Cooperative Oncology Group performance status, and tumor-node-metastasis staging were independent prognostic variables (Patard et al., 2005). Many clinical trials now restrict enrollment based on prognostic risk factors so they may evaluate patient response to treatment and overall survival more consistently (McDermott, 2005; Zisman et al.).

### Treatment Options and Nursing Support

Current treatment options for RCC are listed in Table 3, as recommended by NCCN (2006). Radical nephrectomy has been the gold standard for treating local disease, although nephron-sparing techniques and laparoscopic nephrectomies are considered effective approaches for select patients (Atkins et al., 2004). Surgery may be useful for some patients with metastatic disease (Flanigan, 2004). As recently as 2005, cytokine therapy with interleukin-2 or interferon-α was viewed as the most useful treatment for metastatic disease, with strong consideration given to treatment in a clinical trial. However, the U.S. Food and Drug Administration (FDA) approved only interleukin-2 for the treatment of metastatic RCC (Chiron Corporation, 2000). The agent’s short duration of response and potentially severe side effects are concerns; trials are ongoing to assess the optimal uses for those therapies (McDermott, 2005). Nursing activities have included postsurgical support and support for the administration of interleukin-2 and interferon-α therapies and their side-effect management. Tumor histology guides treatment recommendations, including the use of cytokines in clear cell and chemotherapy in non-clear cell cancers (Motzer et al., 2002).

The most promising agents now appear to be those that target the specific pathways implicated in RCC. Drugs targeting VEGF, PDGF, and other kinases have been hypothesized to be effective treatments for RCC and other cancers (George & Kaelin, 2003). Sorafenib (Nexavar®, Bayer Healthcare Pharmaceuticals and Onyx Pharmaceuticals Inc.) was approved by the FDA in December 2005 for advanced RCC, and sunitinib (Sutent®, Pfizer Pharmaceuticals) was approved in January 2006 for advanced RCC and gastrointestinal stromal tumors. The oral agents currently are used as monotherapy, although clinical trials investigating strategies combining the agents with immunotherapy—including interleukin-2, interferon, and other targeted agents—are ongoing. Additional targeted therapies are in various phases of clinical trials and may add to the therapeutic options for RCC.

### Sorafenib

The first oral agent approved for the treatment of metastatic RCC is sorafenib, a multitargeted tyrosine kinase inhibitor that acts on extracellular and intracellular pathways to interrupt tumor growth. Sorafenib interferes with a variety of cell surface kinases (e.g., VEGF receptor-1, 2, and 3; PDGF-β) in tumor vasculature, preventing the formation of new blood vessels that would feed tumor growth. Within cancer cells, sorafenib inhibits the c-Raf and b-Raf kinase pathways, which normally help regulate cell proliferation by interrupting the processes that may lead to

### Table 1. Histologic Subtypes of Renal Cancer

<table>
<thead>
<tr>
<th>TUMOR TYPE</th>
<th>APPROXIMATE %</th>
<th>TUMOR CHARACTERISTICS</th>
<th>CELL OF ORIGIN</th>
<th>GENETIC ABNORMALITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear cell renal cell carcinoma</td>
<td>74</td>
<td>Solitary, well-circumscribed lesion with rich capillary network; clear or granular cytoplasm</td>
<td>Proximal tubule</td>
<td>Von Hippel-Lindau gene</td>
</tr>
<tr>
<td>Papillary</td>
<td>5</td>
<td>Variable appearance; neoplastic cells line papillary fronds with a fibrovascular core. Foamy macrophages may be present in some papillary fronds.</td>
<td>Distal tubule + Distal tubule</td>
<td>c-met oncogene + Fumarate hydratase gene</td>
</tr>
<tr>
<td>Medullary carcinoma</td>
<td>1</td>
<td>Reticular or microcystic growth pattern; forms solid sheets of cells</td>
<td>Distal collecting ducts of the renal medulla</td>
<td>Associated with sickle cell disease</td>
</tr>
<tr>
<td>Chromophobe</td>
<td>5</td>
<td>Solitary, discrete, nonencapsulated irregular nuclear membranes with clear perinuclear halos; abundant eosinophilic cytoplasm. Forms large solid sheets of cells</td>
<td>Intercalated cells</td>
<td>Birt-Hogg-Dube</td>
</tr>
<tr>
<td>Oncocytoma</td>
<td>5</td>
<td>Solitary, well circumscribed; may have central stellate radiating scar</td>
<td>Intercalated cells of the collecting duct</td>
<td>Nonconsistent genetic abnormality</td>
</tr>
</tbody>
</table>

### Table 2. Tumor-Node-Metastasis Staging for Renal Cell Carcinoma

<table>
<thead>
<tr>
<th>STAGE</th>
<th>CHARACTERISTICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>The tumor is less than 7 cm and limited to the kidney.</td>
</tr>
<tr>
<td>2</td>
<td>The tumor is more than 7 cm and limited to the kidney.</td>
</tr>
<tr>
<td>3</td>
<td>The tumor extends into major veins, invades adrenal or perinephric tissues, or invades at least one regional lymph node.</td>
</tr>
<tr>
<td>4</td>
<td>The tumor extends beyond Gerota’s fascia and more than one regional lymph node or the patient presents with metastatic disease.</td>
</tr>
</tbody>
</table>

*Note: Based on information from Greene et al., 2002.*
tumor regression. The dual action increases the likelihood that treatment may change the course of the disease because sorafenib targets the conditions that give rise to the malignancy, rather than killing malignant cells. Sorafenib is considered cytostatic and cytoreductive (i.e., slowing or preventing tumor growth by targeting the tumor and vascular microenvironments), as opposed to purely cytotoxic. Responses to sorafenib treatment may be measured best by time to progression, progression-free survival, and overall survival rather than by complete or partial response rates (Gore & Escudier, 2006; Ratain & Eckhardt, 2004).

Studies of Sorafenib

Promising results from phase II trials showed an increased median progression-free survival among patients treated with sorafenib versus placebo (24 weeks versus 6 weeks, p = 0.009) following an initial 12-week run-in treatment period in which all patients received sofafenib therapy. At the end of the run-in period, patients with stable disease were randomized to receive sorafenib or placebo (Ratain et al., 2006). The results prompted the phase III Treatment Approaches in Renal Cancer Global Evaluation Trial (TARGET): an international, multicenter, randomized, double-blind, placebo-controlled trial for patients with advanced RCC who had failed one prior systemic therapy. The primary efficacy end point was overall survival, and secondary end points included progression-free survival and tumor response rate as measured by Response Evaluation Criteria in Solid Tumors (Escudier, Szczylik, Eisen, Stadler, et al., 2005). Patients were randomized to receive sorafenib 400 mg twice daily or placebo with best supportive care. Both groups took their treatment while fasting or with a moderate-fat meal. Results from a planned, independently reviewed analysis of the first 769 patients showed median progression-free survival for the sorafenib arm of 5.5 months versus 2.8 months for placebo (p < 0.000001) (Bukowski et al., 2007). Because of the strength of the progression-free survival results, the protocol was amended in May 2005 to allow patients in the placebo arm to cross over to the treatment arm of the study (as well as those randomized to placebo who had progressed and were in follow-up). Such cross-over designs may provide the best opportunity for patients in clinical trials to receive active treatment, but they can obscure treatment effect on overall survival. Three months after the final patient was enrolled, 74% of those taking sorafenib still had stable disease versus 53% in the placebo group (Escudier, Szczylik, Eisen, Oudard, et al., 2005). The median overall survival for patients treated with sorafenib (n = 451) was not reached compared to 14.7 months for those (n = 452) who received placebo (p = 0.015). Sixteen months after the cross-over, when censoring data for the placebo group, median survival was 17.8 months for the sorafenib group and 14.3 months for the placebo group (p = 0.029) (Bukowski et al.).

Quality-of-Life Assessments

On day 1 of each treatment cycle, the Functional Assessment of Cancer Therapy Kidney Symptom Index was given to each patient in the TARGET study (N = 903) (Dhanda et al., 2006). The index consists of 27 questions about the emotional, functional, physical, and social well-being of patients with cancer and 15 questions specific to RCC symptoms (Cella et al., 2006). Patients in the sorafenib arm had statistically significant improvements in quality of life, specifically reporting less frequent cough, fever, and shortness of breath, while benefiting from an increased ability to enjoy life (Dhanda et al.; Escudier, Szczylik, Eisen, Oudard, et al., 2005). Patients taking sorafenib also worried less about their condition worsening than did patients receiving the placebo (Dhanda et al.). In the case study (on the next page), the patient was pleased with his treatment: He had stable disease after a year of treatment with sorafenib and managed to complete building a log cabin, a life goal he might otherwise not have been able to achieve. As of July 2007, he continues on sorafenib therapy without significant change in his side-effect profile or quality of life.

Nursing Management

Oncology nurses play a pivotal role in providing patient education regarding drug administration, dosing, and side-effect management. Those activities become even more essential with oral drugs such as sorafenib, which are self-administered on a daily outpatient basis. As with any chemotherapy, effective use of targeted agents depends on proper adherence and reporting and management of side effects to achieve optimal outcomes.

The most common side effects for patients receiving sorafenib are reversible skin rashes, hand-foot skin reaction, diarrhea, hypertension, and sensory neuropathic changes (National Cancer Institute, 2005). Safety data for sorafenib in adults are depicted in Table 4. In earlier studies, approximately 40% of patients receiving sorafenib experienced some form of skin rash, mostly grades 1 and 2 (mild or moderate in severity) (Bayer Pharmaceuticals

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Table 3. Current Treatment Options for Renal Cell Carcinoma

<table>
<thead>
<tr>
<th>STAGE</th>
<th>MODE</th>
<th>OPTIONS</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Surgical</td>
<td>Nephrectomy or cytoreductive nephrectomy</td>
<td>Neophron-sparing and/or laparoscopic surgery for selected patients</td>
</tr>
<tr>
<td>II</td>
<td>Systemic</td>
<td>Sorafenib, sunitinib, high- or low-dose interleukin-2, or interferon with or without low-dose interleukin-2</td>
<td>Patients progressing on these regimens also may consider bevacizumab. Patients with non–clear cell histology should receive gemcitabine, capecitabine, flouxuridine, 5-fluourouracil, or doxorubicin (sarcomatoid cases only) rather than interleukin-2 or interferon.</td>
</tr>
<tr>
<td>III</td>
<td>Palliative</td>
<td>Radiotherapy or best supportive care</td>
<td></td>
</tr>
</tbody>
</table>

Case Study

February 2003

Mr. E, a 54-year-old man, presents with intermittent hematuria that has existed for four months. The following was noted on examination:

- A large partially exophytic mass was found in the left kidney (8.9 cm).
- Metastatic disease exists in the lungs and retroperitoneal lymph nodes.
- Left renal vein tumor thrombus extends into the left renal vein and left adrenal vein.
- Tumor thrombus does not extend into the inferior vena cava.
- A left radical nephrectomy was performed.
- Staging: T3b N2 M1 (clear cell histology)

The tumor extends into major veins or its segmental branches or vena cava below the diaphragm. Metastases exist in more than one regional lymph node, and distant metastases are present (National Comprehensive Cancer Network, 2006).

March 2003

The patient and family met with medical oncologists at several institutions to discuss treatment options. Mr. E declines high-dose interleukin-2 (IL-2) therapy and enters a phase II clinical trial of IL-2 and thalidomide.

January 2004

Treatment was discontinued for disease progression.

February 2004

The patient was treated on a phase I clinical trial of interferon and an investigational biologic response modifier.

August 2004

Treatment was discontinued because of disease progression.

September 2004

The patient was treated on a phase I clinical trial of gemcitabine and an investigational targeted therapy.

January 2005

Mr. E presented with headaches; a magnetic resonance imaging scan of the brain revealed three metastatic lesions with edema. Systemic therapy was discontinued for disease progression. The patient received gamma knife therapy to one lesion in the frontal lobe and two lesions in the parietal lobe.

February 2005

Mr. E begins sorafenib on a clinical trial evaluating the effects of sorafenib on blood pressure. He takes sorafenib 400 mg twice daily without dose interruption or modification.

March 2006

- Computed tomography scans of the chest, abdomen, and pelvis reveal stable disease; a magnetic resonance imaging scan of the brain demonstrates no new lesions.
- Mr. E experienced the following toxicities: grade 2 hand-foot reaction, intermittent grade 1 fatigue, and intermittent grade 1 diarrhea.
- For skin care, he used Udderly Smooth® (Redex Industries Inc.) three times daily and wore cushioned shoes (he could not walk barefoot).
- His current medications were psyllium fiber, loperamide, vitamin C, and a multivitamin for older adults.

July 2007

- Mr. E continues on sorafenib 400 mg twice daily. He has tolerated therapy well, has not required dose interruption or dose reduction, and continues to have stable disease.

Mr. E retired at the time of his initial diagnosis. He subsequently completed building the log cabin he started in July 2002. He and his wife travel to visit family and friends who live out of state and travel throughout the United States for pleasure. He says that he is “slower than I’d like to be but way ahead of where everyone thought I’d be when I was initially diagnosed with stage IV renal cancer.”

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sorafenib treatment should be interrupted until the skin’s condition returns to baseline (Bayer Pharmaceuticals Corporation, 2005). When the drug is withdrawn, toxicity typically begins to resolve within a few days. Once baseline activities of daily living have been regained, sorafenib may be reintroduced at a reduced dose of 400 mg once a day (Bayer Pharmaceuticals Corporation). If a grade 3 hand-foot skin reaction recurs, patients may require additional treatment interruption and further dose reduction (400 mg every other day). For 3 of 451 patients, discontinuation of sorafenib was necessary (Bayer Pharmaceuticals Corporation).

Hypertension has been seen in studies of other antiangiogenic treatments and is another possible side effect of sorafenib (Veronese et al., 2006). A patient’s blood pressure at the start of therapy should be 140/90 mmHg or less, as was mandated during phase II and III clinical trials (Bayer Pharmaceuticals Corporation, 2005). For patients with a history of hypertension, blood pressure should be stable before starting treatment. The prescribing information suggests that, during active treatment, blood pressure should be monitored weekly for the first six weeks of therapy. Most events of hypertension in the TARGET study were diagnosed within the first month of administration. However, any elevation in diastolic blood pressure of 20 mmHg or a reading of more than 150/100 mmHg should warrant immediate medical intervention, and sorafenib doses should be held until blood pressure returns to baseline. Patients who are taking antihypertensive medications should be receiving the maximum dose of the first drug before adding a second antihypertensive agent.

Fatigue was reported by 37% of patients taking sorafenib, 5% at grade 3 or more (Bayer Pharmaceuticals Corporation, 2005). The exact etiology of treatment-related fatigue is not known, but it may be related to the disease, anemia, poor nutritional intake, lack of sleep, or drug mechanisms of action. Nursing management is no different than management of any patient with cancer experiencing fatigue. Patients should be encouraged to balance rest and activity so that energy more likely will be available when patients wish to engage in activities.

Diarrhea occurred in 43% of patients taking sorafenib; only 2% had grade 3, and none had grade 4 (Bayer Pharmaceuticals Corporation, 2005). According to the National Institutes of Health (2003) Common Terminology Criteria version 3.0, grade 1 refers to mild toxicities, grade 2 refers to moderate toxicities, grade 3 refers to severe toxicities, and grade 4 refers to life-threatening toxicities.

### Drug Interactions

Sorafenib is metabolized primarily via CYP3A4 in the liver; therefore, substances that induce CYP3A4 (e.g., St. John’s wort, rifampin) may decrease sorafenib concentrations. Coadministration with rifampin, St. John’s wort, or dexamethasone is expected to increase metabolism of sorafenib and thus decrease sorafenib concentrations (Onyx Pharmaceuticals, Inc., & Bayer Pharmaceuticals Corporation, 2007). Nurses must learn whether patients are using herbal remedies and recognize the potential for drug interactions with rifampin, phenytoin, phenobarbital, and dexamethasone. However, use of the CYP3A4 inhibitor ketoconazole (400 mg) did not alter the sorafenib area under the concentration time curve. Sorafenib may increase international normalized ratio levels in patients who are taking warfarin (Bayer Pharmaceuticals Corporation, 2005). International normalized ratio levels and prothrombin time should be monitored closely to prevent complications when patients are on treatment (Bayer Pharmaceuticals Corporation).

### Table 4. Phase III Safety Data: Most Frequently Occurring Grade 3 or 4 Adverse Events

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Sorafenib (N = 451)</th>
<th>Placebo (N = 451)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Any Grade</td>
<td>Grade 3 or 4</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Fatigue</td>
<td>195</td>
<td>43</td>
</tr>
<tr>
<td>Hand-foot skin reaction</td>
<td>134</td>
<td>30</td>
</tr>
<tr>
<td>Hypertension</td>
<td>76</td>
<td>17</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>65</td>
<td>14</td>
</tr>
<tr>
<td>Bone pain</td>
<td>34</td>
<td>8</td>
</tr>
<tr>
<td>Tumor pain</td>
<td>29</td>
<td>6</td>
</tr>
</tbody>
</table>

Note. According to the National Institutes of Health (2003) Common Terminology Criteria version 3.0, grade 1 refers to mild toxicities, grade 2 refers to moderate toxicities, grade 3 refers to severe toxicities, and grade 4 refers to life-threatening toxicities.

Note. Based on information from Bayer Pharmaceuticals Corporation, 2005.
Conclusion

By targeting specific factors known to contribute to RCC, sorafenib offers improved progression-free survival (Escudier, Szczylik, Eisen, Stadler, 2005; Ratain et al., 2005) without requiring hospital or outpatient clinic administration of IV medications. Insurance coverage for sorafenib and other oral cancer therapies varies, and many pharmaceutical companies have resources to assist with insurance review and programs to help patients with the costs associated with therapy. Oral cancer therapies are among the fastest-growing categories of treatment (Bedell, 2003), and as they become more prevalent, oncology nurses will have increasing roles in patient education, supporting adherence and managing side effects. They may be required to educate other nursing staff who need to become familiar with new therapies for RCC, as patients may receive their treatment from community oncologists rather than from larger centers that specialize in high-dose interleukin-2, low-dose immunotherapy, or clinical trials.

Healthcare providers who are monitoring patients receiving sorafenib therapy need to educate and empower those patients regarding their therapy. Prior to dose initiation, healthcare providers should educate patients about the nuances of managing the early signs of adverse events. Doing so helps patients maintain their quality of life and continue treatment. Patients should be given instructions on appropriate times to contact the healthcare team of adverse events before they become too serious: Not everything can be managed at home by patients and their families or caregivers. Oncology nurses can provide assistance, and that message needs to be communicated to patients. Patients should be reassured that although oral tyrosine kinase inhibitors, such as sorafenib, can be self-administered at home, treatment management works best through a team effort. Nurses should work with patients as they learn more about the mechanisms of RCC and the new treatments that are extending quality and quantity of life.

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