McGill, K. T., a 76-year-old woman, was diagnosed with stage IIA multiple myeloma in 2007 when she presented with severe back pain. K.T. underwent kyphoplasty, a minimally invasive surgery to relieve compression fractures, followed by eight cycles of lenalidomide with a good response. Peripheral blood stem cells were collected after mobilization with filgrastim and plerixafor. She was admitted to the hospital and conditioned for an autologous hematopoietic cell transplantation (HCT) with melphalan followed by reinfusion of peripheral blood stem cells, receiving a cell dose of $4.26 \times 10^6$ CD34 cells/kg. K.T. developed neutropenic fever on day 8 that responded to broad spectrum antimicrobials. She achieved a prompt hematologic recovery, reaching an absolute neutrophil count greater than 500 mm$^3$ by day 11 after transplantation. On day 9, however, K.T. developed diarrhea, negative for *Clostridium difficile*, which was managed with antibiotics. She was discharged 17 days following transplantation with blood counts within normal limits, except for mild anemia.

On day 19, K.T. presented for follow-up with a pruritic, erythematous skin rash covering 45% of her body surface area, involving her upper extremities, anterior, and posterior torso. She also experienced gastrointestinal (GI) symptoms of nausea, abdominal cramping, weight loss, and anorexia. Tapered methylprednisolone dosing was prescribed for a suspected diagnosis of autologous graft-versus-host disease (GVHD). Skin rash resolved in response to corticosteroids and pruritis was managed with topical triamcinolone. GI symptoms initially improved, but recurred following completion of the methylprednisolone taper.

On day 32, K.T. presented with progressive diarrhea and diffuse skin rash involving 100% of her body surface area. Medication review did not reveal a likely source for drug reaction. A skin biopsy was taken and showed foci of basal cell detachment of fibrinopurulent exudates and mucus covering the colonic mucosa and mucus. Biopsies did not reveal apoptosis, but viral inclusions were seen and immunohistochemical stain for cytomegalovirus was negative. With an excess of 10 loose bowel movements daily, K.T. required a short course of total parenteral nutrition for adequate caloric intake. GI symptoms failed to respond to treatment with vancomycin and metronidazole; therefore, prednisone was restarted for a two-week period followed by a successful taper. Total parenteral nutrition was discontinued and K.T. was discharged on day 77 receiving prednisone 10 mg every other day, antimicrobial prophylaxis, and vancomycin.

On day 103 (three days after discontinuation of steroids), K.T. was readmitted with complaints of progressive diarrhea while receiving vancomycin. Subsequent colonoscopy showed thick mucus covering the colonic mucosa and a severely inflamed colon. Pathology showed acute pseudomembranous colitis with foci of acute cryptitis and crypt dropout. Biopsies did not reveal apoptosis, but detachment of fibrinopurulent exudates was noted, consistent with a diagnosis of acute GVHD. Laboratory examination revealed mild anemia, thrombocytopenia, mild elevation in leukocyte count, normal liver function tests, and a negative cytomegalovirus test by polymerase chain reaction analysis. Stool assay tested positive for *C. difficile* on multiple occasions. High-dose prednisone was reinstituted and vancomycin was increased to 500 mg four times daily for 10 days. This was followed by a rapid steroid taper given the marked improvement in symptoms. On completion of taper, intestinal symptoms recurred, prompting re-admission for correction of fluid loss.

At the time of this writing, K.T. was being considered for alternative therapy in the treatment of recurrent acute GVHD and complicated *C. difficile* following autologous transplantation for multiple myeloma.

### Autologous Graft-Versus-Host Disease

Historically, GVHD has been a complication of allogeneic HCT and a major cause of morbidity and mortality (Drobyski, Hari, Keever-Taylor, Komoroski, & Grossman, 2009; Holmberg et al., 2006). An estimated 50%–70% of patients develop acute GVHD following allogeneic HCT (Kline, Van Besien, Nathanson, Noffsinger, & Artz, 2006). Holmberg et al. (2006) reported a similar syndrome that exists in the autologous setting after HCT, one that arises as a result of immunologic manipulation (immunosuppression administration and withdrawal to stimulate a graft-versus-tumor effect) or spontaneously without immunosuppressant stimulation.