

their inhibition is of clinical relevance. As shown by Schade, inhibition of T-cells by dasatinib is achievable in murine models, but the doses required were higher than those required to inhibit CML models.<sup>9</sup> Thus, if the *in vivo* effects of dasatinib against NK cells are similar to those against T-cells, once-daily dosing with dasatinib may result in only minor suppression of NK cell function and greater suppression might occur when dasatinib is taken at higher doses or with greater frequency. Further *in vivo* studies expanding on our findings will assist in discerning any effects of dasatinib on NK cell function in patients.

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## To the editor:

### Single agent lenalidomide induces a response in refractory T-cell posttransplantation lymphoproliferative disorder

Patients undergoing solid organ transplantations carry a significant risk of developing posttransplantation lymphoproliferative disorders (PTLDs).<sup>1</sup> Only a minority of PTLD cases are of T-cell origin, accounting for 6% to 8% of cases.<sup>2,3</sup> T-cell PTLDs tend to occur later in the posttransplantation course and are generally resistant to therapy. In one series, the median survival was 5 weeks.<sup>4</sup> Treatment of T-cell PTLDs includes reduction of immunosuppression and various chemotherapy regimens, neither of which have been successful.<sup>2,4</sup>

We describe a 62-year-old female patient who developed T-cell PTLD 7 years after receiving a cadaveric renal transplant for autosomal dominant polycystic kidney disease. Her immunosuppressants included cyclosporine and prednisone. She presented with excessive fatigue and lymphocytosis. Her initial complete blood count (CBC) showed white blood count (WBC) count  $18.4 \times 10^9/L$  with 59% lymphocytes, hemoglobin 125 g/L, and platelet count  $233 \times 10^9/L$ . There was no lymphadenopathy or hepatosplenomegaly. Flow cytometry of blood lymphocytes revealed a clonal T-cell population expressing CD3, CD5, CD 45, and CD7, with coexpression of CD4 and CD8. These cells did not express CD34 or terminal deoxynucleotidyl transferase (tdt). T-cell receptor gene rearrangement study was positive. Studies for Epstein Barr virus were negative. Bone marrow was involved with disease. Initially, immunosuppressive therapy was reduced by 50%. Over the next 4 weeks, her fatigue worsened, she developed night sweats, and her lymphocyte count increased. Over the next eleven months, multiple therapies were tried without success (Figure 1). Approximately one year into her course she developed marked lymphocytosis and hepatosplenomegally.

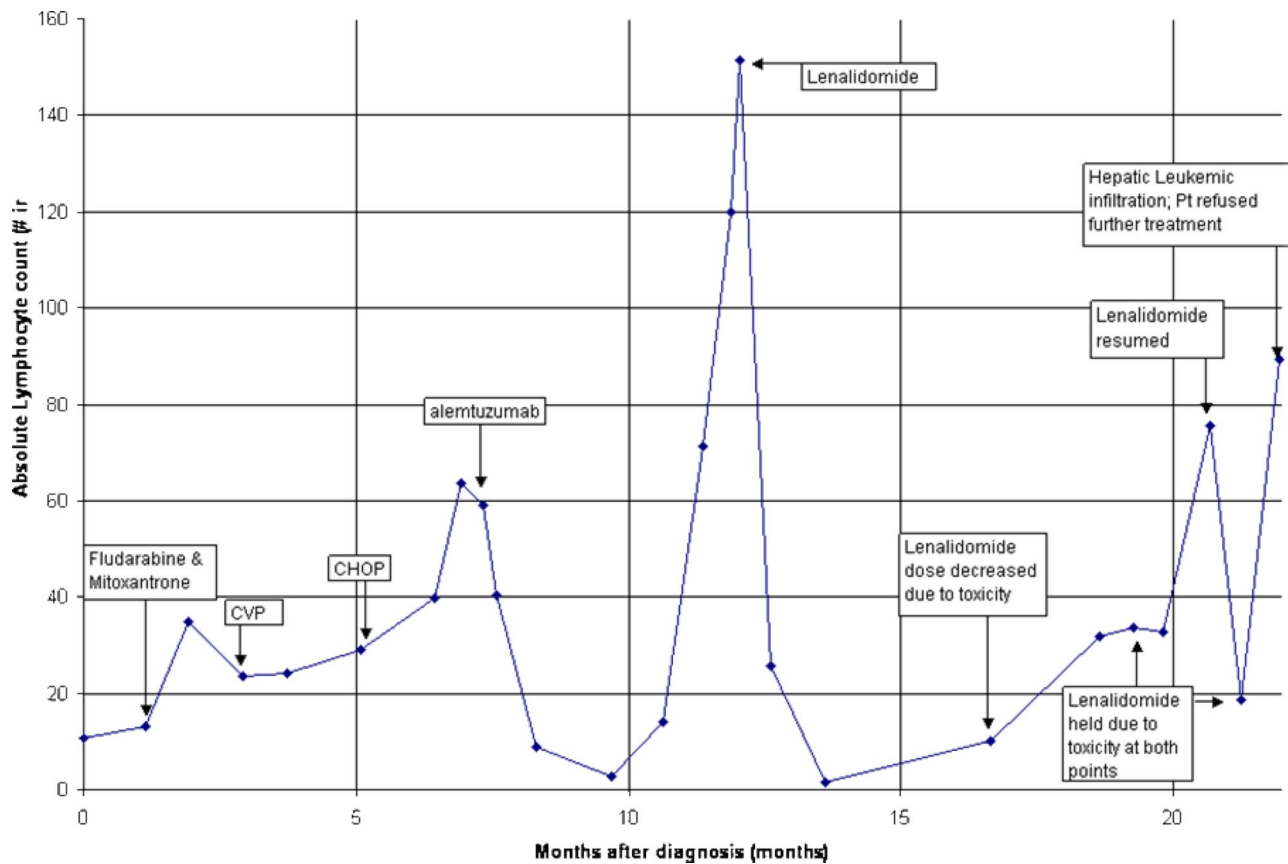
After discussion with the patient, she was started on lenalidomide, a thalidomide derivative at 25 mg/day. Lenalidomide is an immunomodulatory agent that augments T-cell response, increases secretion of tumor necrosis factor- $\alpha$  and interleukin-12, and suppresses angiogenesis.<sup>5</sup> Thalidomide and lenalidomide have been shown to have activity against T-cell lymphomas.<sup>6,7</sup> After 7 weeks of lenalidomide, our patient's lymphocyte count became normal (Figure 1). She reported subjective improvement and her hepatosplenomegally resolved. After 6 months, severe muscle cramps necessitated decrease of the lenalidomide dose to 10 mg every other day. Her cramps persisted, requiring discontinuation of treatment. This was followed by relapse of disease in 3 weeks (Figure 1). Resumption of lenalidomide at 10 mg twice a week again resulted in improvement of lymphocytosis. After 9 months, with patient unable to tolerate lenalidomide treatment, her disease progressed rapidly with heavy hepatic infiltration of leukemic cells. The patient declined further therapy and expired in 2 weeks.

Our patient achieved a complete hematologic response with lenalidomide therapy for 9 months, with normalization of her WBC count at optimal doses. We believe that this is the first report of a patient with T-cell PTLD obtaining response with lenalidomide therapy. This case shows a clear need for further investigation into the role of lenalidomide in T-cell PTLD.

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**Figure 1. Response of T-cell posttransplantation lymphoproliferative disorder to lenalidomide therapy.** The response to therapy is depicted as absolute lymphocyte count. Patient had treatment failures to fludarabine; cyclophosphamide, prednisone, and vincristine (CVP), and cyclophosphamide, doxorubicin, prednisone, and vincristine (CHOP). Alemtuzumab resulted in a partial hematologic response before relapse. Lenalidomide resulted in a complete hematologic response. She ultimately was unable to tolerate lenalidomide therapy, resulting in disease progression and death.

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## To the editor:

### The American Society of Hematology: a success at age 50; blood banking and sodium citrate

I enjoyed the excellent article by Drs Jaffe and Kaushansky, “The American Society of Hematology: a success at age 50.”<sup>1</sup>

There is some controversy concerning the introduction of sodium citrate for blood transfusion and the establishment of the first blood bank in the United States.

Dr Luis Agote used sodium citrate for transfusion of whole blood for a patient on November 9, 1914.<sup>2</sup> Instead of publishing his work in a medical journal, he gave the story to “LaPresna,” the leading newspaper of Buenos Aires. Two months later in the “New York Medical Record” of January 23, 1915, Dr Richard Lewisohn of Mt Sinai Hospital in New York reported on 2 cases in which he used citrate of soda for transfusion. Agote was

considerably displeased because Lewisohn did not mention Agote in this report. Actually, Albert Hustin, a Belgian surgeon, used sodium citrate and glucose as an anticoagulant for blood transfusion on March 17, 1914. He performed his transfusion at St Jans Hospital in Brussels.<sup>3</sup>

The first blood bank was established by Dr John S. Lundy, Head, Section of Anesthesia, at Mayo Clinic in 1935.<sup>4</sup> He had kept citrated blood in the “ice box” for as long as 14 days and found that it could be administered with the usual benefits to the patient and without reaction. The Cook County Hospital began such a service in 1936. Thirty-two percent of Mayo Clinic transfusions were administered to nonsurgical patients. Two years later, Lundy