Research Article

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Tawam Hospital's Experience with Plasma Cell Leukemia.

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INTRODUCTION

There are various types of plasma cell dyscrasias, ranging from mild (MGUS) to very aggressive (PCL). Primary PCL is a condition that develops from scratch, while leukemic transformation of underlying relapsing or Myeloma recalcitrant to treatment. The disease's diagnostic standards have been thoroughly explained before [1]. The illness has an extremely bad prognosis and, even with vigorous therapy, typically progresses quickly and downward.

The first proteasome inhibitor authorized for the treatment of multiple myeloma (MM) was boratezamib. According to recent findings, treating PCL upfront with this drug has shown positive results [2]. We discuss our PCL experience. There were five patients found to have PCL. While the other patients had secondary PCL, one patient had primary PCL.

CASE REPORT

Case 1

A 48-year-old man of South Asian ancestry appeared lethargic and exhausted. After being admitted to a different hospital, leukocytosis and anemia were discovered in him. He was in circulation.

cells of plasma > 2000/mm3. The patient was moved to our facility so that we could provide more care. He underwent a thorough work-up that included an echocardiogram, skeletal survey, immunofixation, bone marrow biopsy, cytogenetic analysis, serum and urine electrophoresis, and liver, renal, and bone profile 93% of the plasma cells in the bone marrow had plasmablasts. CD 19-, CD 45-, CD 138 +, and CD 38 +. IgG lambda was detected by SPEP and IFE at 68.7 gm/liter. The Bence Jones protein had both free lambda light chain and IgG lambda. A normal male was shown by a cytogenetic investigation, and beta 2 microglobulin was raised at 4.5.

Doxil, Dexamethasone, Bortezomib, and Thalidomide (DVD-T) were used to induce him. His SPEP/IFE revealed a monoclonal increase of 4.3 gm/lit (>90%) after three cycles, however the bone 50% of the illness was still present in the marrow. The patient followed the same regimen for an additional three cycles before being reassessed. Remaining M protein was detected at 3.4 gm/liter by SPEP and IFE. The level of beta 2 microglobulin has dropped to 2.3. There were still 2% of plasma cells in the bone marrow.

After that, the patient was referred to an outside facility for HSCT.

Case 2

In January 2010, a 49-year-old man was diagnosed with chronic phase CML. Using imatinib 400, she experienced a significant molecular remission.

bone pain and exhaustion in her neighborhood hospital. It was discovered that she had acute renal failure and severe anemia (hemoglobin 6.4). She received supportive care before being referred for more testing. It was discovered that she had an increased levels of total protein, monoclonal-spiked IgG, and free lambda light chain exceeding 1000 mg/ml. Patient declined to get a bone marrow biopsy. Data from flow cytometry agreed with PCL. Renal function quickly improved after induction with RVd. After that, she traveled to Singapore, where she received a partial response after four more cycles of RVd. She then underwent a stem cell transplant using Melphalan 140 mg/m2, after which she had two cycles of VCd. Six weeks later, restaging revealed just PR. The patient began taking lenalidomide.She had pancytopenia when she was seen once more in December 2014. She had high monoclonal spike, free lambda light chains > 2300 mg per milliliter, and circulating plasma cells. Patient deteriorated quickly and passed away.

Case 3

A 59-year-old woman with severe renal failure, sepsis, and hypercalcemia was admitted to the intensive care unit. In addition to pancytopenia with circulating plasma cells, she exhibited an increased total protein level. Following her stabilization, she was identified as having salmon Durie stage 3 B IgA MM, normal female karyotype, circulating plasma cells (ISS staging not completed), and no evidence of (t 4;14), (t11;14). The patient experienced a partial response (PR) after three cycles of VMP treatment. Before the commencement of the fourth cycle of VMP, the patient changed into PCL. She had

urosepsis at the time of her admission, and her condition and expiry were gradually getting worse.

Case 4

Salmon Durie stage 3 A, ISS 2 MM, normal male karyotype, FISH negative for P53, t4;14, and t11;14 were diagnosed in a 60-year-old guy. Induction using pegylated Adriamycin and Bortezomib as well as low-dose dexamethasone. Within six months of the patient's therapy ending, their condition worsened, and they were prescribed salvage therapy consisting of dexamethasone and thalidomide as well as therapeutic warfarin anticoagulation. Six cycles later, he had a vGPR.

The therapeutic dose of thalidomide was lowered to 100 mg per day since he was unable to tolerate it. After using low dose thalidomide for six months, he eventually converted to CR. After three years of maintaining the remission rate, he started to experience anemia that was gradually getting worse and a growing M spike. He was admitted with sudden, severe dyspnea.

Case 5

A 77-year-old man with renal insufficiency was diagnosed with ISS stage 3 and IgG lambda MM Salmon Durie stage 3 B. Dexamethasone and Bortezomib were used to induce him. Patient success after six cycles, a PR. He was administered lenalidomide and dexamethasone (Rd) for two cycles after declining further bortezomib. He had proof.Bortezomib was added after the biochemical progression (RVd).

The patient underwent 16 cycles in all. After that, he spent eight months back on Road. After that, he received salvage using VMP without any advantage. The patient was not followed up with for six months. He traveled to Germany for a second opinion while waiting and was prescribed pomalidomide; nevertheless, he later developed PCL. After receiving palliative care, he passed away after eight weeks.

DISCUSSION

The plasma cell tumor's most aggressive manifestation is PCL. Melphalan or polychemotherapy had been the basis of treatment in the past (VAD). The median result was depressing 6-7 months of survival. According to recent findings, PCL patients may have longer life times if they use new medications such IMiDs in addition to bortezomib. Despite the fact that our experience is restricted to six cases, it generally seems to corroborate the idea that PCL must be treated with new medicines right away. Even with the new medicines, secondary PCL performed badly in our experience.

In a clinic environment, botezomib and IMiD-based regimens are simply administered with the right supporting care. Following that, eligible patients may be combined with autologous HSCT [3]. Consequently, individuals might necessitate maintenance after 2-4 cycles of consolidation. Because this disease is so uncommon, the only way to assess and validate this line of care is through a large-scale intergroup trial.

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