Osanto et al. [1983] reviewed the literature on plasma cell leukaemia and reported an additional case. This was of a patient with IgDA type who went into complete remission but relapsed 1 year later with numerous subcutaneous nodules. We would like to report a patient with an IgG plasma cell leukaemia who had a similar outcome.

A 53-year-old man presented in October 1981 with recent weight loss, aching in legs and arms and a chest infection. He had felt vaguely unwell for the past year but had continued at work and had not presented to any medical practitioner before. He had slight axillary lymphadenopathy and a spleen and liver that were just palpable. Laboratory studies at presentation revealed Hb 9.6 g/dl, WBC 49 × 10^9/l with 84% plasma cells and 12% neutrophils, platelet count 30 × 10^9/l, ESR 145 mm, IgG 96 g/l, IgA 0.54 g/l, IgM 0.34 g/l. The marrow was densely infiltrated with plasma cells but the skeletal survey was normal. Proteinuria was 8.7 g/24 h and consisted of Bence Jones kappa protein. The serum paraprotein was typed as IgG3 kappa. The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.

The patient was treated with cyclical melphalan and prednisolone with rapid response. Plasma cells in the peripheral blood disappeared, the spleen and liver became impalpable and the raised calcium and mild renal impairment reverted to normal. The paraprotein and Bence Jones protein disappeared within 3 months. He continued in remission on melphalan and prednisolone until June 1982 when he developed subcutaneous nodules on arms, legs and trunk. Biopsy of these confirmed infiltration of the dermis by large lymphoma cells with plasmacytoid features. The bone marrow was normal and there was no detectable paraprotein.