Hodgkin’s disease presenting as idiopathic thrombocytopenic purpura

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Summary

A case of Hodgkin’s disease presenting as idiopathic thrombocytopenic purpura in a 23-year-old male is reported. This is a rare presentation of Hodgkin’s disease having been previously described in only two cases.

KEY WORDS: Hodgkin’s disease, idiopathic thrombocytopenic purpura.

Introduction

Idiopathic thrombocytopenic purpura (ITP) may be the initial manifestation of systemic lupus erythematosus, lymphoproliferative disorder, or Evan’s syndrome. The possibility of an associated or underlying pathological process should be borne in mind in cases of ITP, although it may be months or years before the true nature of the primary disease is revealed.

A case is presented in which an unsuspected diagnosis of Hodgkin’s disease was made following splenectomy for ITP.

Case report

A 23-year-old engineer presented to his general practitioner in July 1982 with a 7-day history of a purpuric rash and gingival bleeding. Some 4 weeks earlier he had had an upper respiratory tract infection. He took little alcohol and had taken no drugs in recent months. On examination there was purpura on the legs, soft palate and gums but no other abnormality was detected. A full blood count showed a haemoglobin 14 g/dl, leucocyte count $8 \times 10^9$/litre, and platelets $6 \times 10^9$/litre. A bone marrow aspirate was consistent with idiopathic thrombocytopenic purpura with increased numbers of megakaryocytes and defective platelet budding. Viral titres showed no evidence of recent infection. Anti-DNA titres and rheumatoid factor were negative. The patient was treated with oral prednisolone. His platelet count returned to normal and steroid therapy was tailed off and stopped after 3 months.

Three months later he relapsed with platelets of $6 \times 10^9$/litre and recurrence of his symptoms. Prednisolone was restarted at 60 mg/day with a view to raising the platelets before elective splenectomy. One week later he was found to be unrousable with evidence of having vomited blood in his sleep. Subsequently he had a grand mal fit and respiratory arrest. Following resuscitation he had repeated haematemeses and melaena—he remained drowsy with brisk reflexes and bilateral extensor plantars. Computed axial tomography (CT scan) revealed a left frontal haematoma without evidence of mid-line shift. Emergency splenectomy was performed with platelet infusion following clamping of the splenic pedicle. The patient made a good recovery—platelet count rose rapidly to normal and has remained so since. There was no subsequent neurological abnormality and follow-up CT scan showed resolution of the haematoma.

Histological examination of the spleen showed evidence of mixed-cellularity Hodgkin’s disease, a diagnosis hitherto unsuspected. A CT scanning of the abdomen showed bilateral para-aortic disease and bipedal lymphography confirmed extensive para-aortic lymph node involvement. A bone marrow aspiration and trephine showed no evidence of lymphomatous infiltration but a bone scintogram demonstrated widespread disease. No evidence of Hodgkin’s disease above the diaphragm was found. The patient was staged as IVA according to the Ann Arbor classification (Carbone et al., 1971).

An alternating regime of chlorambucil, vinblastine, prednisolone procarbazine (Chl.V.P.P.) and adriamycin, bleomycin, vinblastine and DTIC (A.B.V.D.) was commenced (Bonnadonna et al.,
1982) and a complete remission obtained. The patient remains well to date.

Discussion

The concurrence of autoimmune disorders with lymphoproliferative disorders is well recognized (Jones, 1973), particularly in association with chronic lymphatic leukaemia. In Hodgkin's disease autoimmune haemolytic anaemia is uncommon and has been reported as occurring in 2-7% of cases in one large series (Eisner, Ley and Mayer, 1967). Thrombocytopenia appears to be a very unusual complication. Twenty-eight cases have been previously described (Cohen, 1978; Fink and Al-Mondhiry, 1976; Rudders, 1974; Paz, Bolo and Sanabria, 1980; Kirshner, Zamkoff and Gottlieb, 1980). In only two of these cases, however, (Rudders, Aisenberg and Schiller, 1972; Paz et al., 1980) did the diagnosis of ITP precede that of Hodgkin's disease.

It is not clear whether the association of ITP and Hodgkin’s disease has an adverse effect upon the outcome (Cohen, 1978; Fink and Al-Mondhiry, 1976). The association has been described in all histological types of Hodgkin's disease and in all clinical and pathological stages.

This case serves as a reminder that spleens removed in the treatment of ITP should always be submitted for careful histological analysis.

References


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