Postgraduate Medical Journal (April 1977) **53**, 222–223

CASE REPORTS

Co-existent chronic lymphatic leukaemia with polycythaemia vera

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Summary

A patient presenting with chronic lymphocytic leukaemia and iron deficiency anaemia who, following oral iron therapy, developed the clinical and pathological features of polycythaemia vera is reported. The relationship between these two diseases when co-existing is discussed.

Introduction

The association of polycythaemia (rubra) vera (PRV) with various lymphoproliferative disorders has been reported occasionally in the literature (Bethard, Block and Robson, 1953; De Baker and Lawrence, 1951; Heinle *et al.*, 1966; Vianna and Essman, 1971). The association between PRV and chronic lymphocytic leukaemia (CLL) would appear to be rare. Hansen (1973), analysing 189 cases of CLL, mentions no cases of PRV and the authors have found during an analysis of 228 cases of CLL and 293 cases of PRV at Christie and Withington Hospitals over the past 12 years only one case in which the diseases co-existed. This case is now described.

Case report

A 75-year-old widow presented to hospital with a bruised and swollen right leg. Preliminary examination revealed a spontaneous haemorrhage into the thigh, so she was referred to the haematology department. She had a past history of mild congestive cardiac failure and osteoarthrosis of both knees. Her medication on presentation was amiloride and paracetamol, there being no history of recent aspirin ingestion. Direct questioning revealed no symptoms suggestive of further haemorrhagic episodes. On examination there was a large ecchymosis of the right thigh and moderately severe bilateral varicose veins. Small lymph nodes were palpable in the left posterior triangle of the neck and the left axilla. The liver and spleen were just palpable. Investigations revealed: Hb 14·3 g/dl; WBC $27{\cdot}8 \times 10^9$/l (71% mature lymphocytes, 27% neutrophils, 1% monocytes, 1% eosinophils; many smear cells present); platelets 482×10^9/l; MCV 75 fl; MCH 22·5 pg. Prothrombin time 15 sec (N12 sec). Cephalin-kaolin time, thrombin time and fibrinogen were all normal. IgG 1·5 g/l, IgA 1·9 g/l, IgM 0·7 g/l; direct Coombs test, negative. A diagnosis of benign CLL was made and her spontaneous haemorrhage was considered to be due to a ruptured internal varicosity. No treatment other than local support was instituted. At follow-up 1 month later the swelling had subsided. Hb 12·6 g/dl; WBC $27{\cdot}8 \times 10^9$/l; platelets 482×10^9/l; serum iron 5·4 μmol/l; TIBC 79·5 μmol/l. Faecal occult blood examination was negative and no history of gastrointestinal symptoms was elicited. Oral iron supplements were instituted. Three months later she complained of lethargy, headaches, but no pruritus. Hb was 21·6 g/dl. The oral iron was discontinued. At that time other results were as follows: WBC $26{\cdot}2 \times 10^9$/l (neutrophils 33%; lymphocytes 61%; monocytes 4%; eosinophils 2%), platelets 380×10^9/l. Red cell mass 60 ml/kg; plasma volume 47·6 ml/kg. Arterial blood gases: P_{O_2} 94 mmHg, P_{CO_2} 32 mmHg; pH 7·39. Urea and urate were normal. Serum lysosyme 10·4 μg/ml, unsaturated B_{12} binding capacity 2617 mg/l, leucocyte alkaline phosphatase (LAP) score 108. Marrow aspiration and trephine revealed infiltration with mature lymphocytes. Stainable iron was present. Typing of her lymphocytes using rosettes showed B cells 60% and T cells 6·7%.

A diagnosis of PRV co-existing with CLL was made and she was treated initially by venesection but subsequently received ^{32}P. Since that time she has remained well with Hb 13–14 g/dl, WBC 13·8–$20{\cdot}9 \times 10^9$/l (70–84% mature lymphocytes) and platelets 150×10^9/l.

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Discussion

The relationship between PRV and CLL when co-existing is not understood. Heinle *et al.* (1966) in describing three cases of PRV associated with CLL lymphoma and multiple myeloma, respectively, referred to the possibility of a unified histogenesis involving these disorders while Bethard *et al.* (1953) suggested that these two disorders are probably unrelated. Vianna and Essman (1971) noted a suppression of CLL by PRV in one patient in whom the two diseases co-existed. The present case does not show this phenomenon. Certainly the co-existence of two uncommon diseases would be exceedingly rare by chance alone and points to an association. If this is so, then a more likely explanation than unified histogenesis would be that of impaired T cell immunity in CLL which might predispose to the emergence of a second neoplastic clone. Hansen (1973) has reviewed the evidence for predisposition in CLL for a second neoplasia, and quotes eighteen series of CLL each showing a high incidence of solid tumours. While this is suggestive that predisposition occurs such series are highly selective and hence conclusions based on them are uncertain. Benign CLL which may be entirely asymptomatic may be more frequent than generally realized. Until this incidence is accurately known, the degree of predisposition of CLL for a second neoplasia must remain in doubt.

Acknowledgment

Gratitude is expressed to Mr M. Palmer, Statistical Department, Christie Hospital, for reviewing patient records and to Dr I. Todd, Christie, for permission to report a patient under his care.

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Postgraduate Medical Journal (April 1977) **53**, 223–224

Symptomatic hyponatraemia associated with tolbutamide therapy

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Summary

A case of hyponatraemic coma occurring in a patient with diabetes mellitus treated with tolbutamide is described. Although chlorpropamide is known to cause water retention in some circumstances, this is a less well recognized complication of tolbutamide therapy.

Introduction

The antidiuretic action of chlorpropamide in diabetes insipidus is well described and it is probable that chlorpropamide acts by potentiating the renal response to small amounts of endogenous vasopressin (Miller and Moses, 1970). Patients with diabetes insipidus treated with chlorpropamide may develop water retention with symptomatic hyponatraemia (Webster and Bain, 1970). Patients with diabetes mellitus treated with chlorpropamide may develop a similar complication, though only if fluid balance is already compromised by, for example, the co-existence of incipient or overt heart failure (Weissman, Shenkman and Gregerman, 1971). Not all sulphonylureas have a similar effect on water excretion and glibenclamide has been found to have a diuretic effect (Moses, Howanitz and Miller, 1973). The effects of tolbutamide are less clear. A case is now reported of symptomatic hyponatraemia associated with tolbutamide therapy in a patient with diabetes mellitus.

Case report

A 53-year-old lorry driver was admitted to hospital having been found unconscious at home. He had been a diabetic since a drainage operation for