Blood Counts in Portal Hypertension of Non-Cirrhotic Origin: Correlation with Splenic Size and Splenic Pulp Pressure

S R NAIK, P BAMBERY, S K TYAGI, D V DATTA, A KOSHY

Department of Internal Medicine, Experimental Medicine and Hepatology, Postgraduate Institute of Medical Education and Research, Chandigarh 160 012

Abstract
Sixty-two patients with portal hypertension—37 with extra-hepatic venous obstruction and 25 with non-cirrhotic portal fibrosis—were studied. Splenic size was found to have a significant inverse correlation with total leucocyte and platelet counts, but not with hemoglobin. No correlation was found between splenic size and splenic pulp pressure or between splenic pulp pressure and blood counts. There was no significant difference in the frequency of blood count depression between the two groups studied. The exact mechanism for depression of counts associated with increased splenic size remains obscure.

Key words: Portal hypertension, extra-hepatic venous obstruction, non-cirrhotic portal fibrosis, hypersplenism, splenic pulp pressure, splenic size.

Introduction
Reports on the relationship between splenic size and hematological abnormalities in patients with portal hypertension are sparse. This may be partly due to the difficulties in reliable assessment of splenic size. Splenic size has been recently assessed clearly by radio-isotopic1,2 ultrasonographic3 and computerised tomographic4 techniques. We have previously shown that splenic size can be reliably assessed by measuring splenic cross-sectional area from conventional splenoprtovenography films.5 There is no study correlating common hematological parameters to splenic size and splenic pulp pressure in patients with portal hypertension due to non-cirrhotic causes. We therefore present a retrospective analysis of such data in our patients.

Material and Methods
Thirty-seven patients (28 males, 9 females) with evidence of extra-hepatic portal vein obstruction (EHO) and 25 (7 males, 18 females) with non-cirrhotic portal fibrosis (NCPF) were studied. Patients were included in the study after ascertaining that they had adequate clinical and investigative data—satisfactory splenoprtovenographic pictures suggesting either EHO or NCPF, liver biopsy excluding the possibility of cirrhosis of the liver, and complete blood counts. Splenic cross-sectional area (SCA in cm²) was estimated as described earlier5 by us. Splenic pulp pressures were available in 24 cases (10 EHO, 14 NCPF). Blood counts taken for analysis were either the first readings or subsequent lowest readings in each individual as suggested by Felix et al.6 whose criteria for ‘hypersplenism’ were also adopted: leucocyte counts below 4·0 × 10⁹/mm³ and platelet counts below 1·0 × 10⁹/mm³.

Data were analysed by Student’s t test, chi squared test and regression analysis. SCA, blood counts and splenic pulp pressures were compared within the two groups and correlation was drawn between the different parameters within each group and when the groups were combined.

Results
Patients with EHO were predominantly males (M::F = 3:1) as compared to NCPF patients (M:F = 0:39), the difference being highly significant (X² = 16·8, p < 0·001). The age range and mean ± SD for EHO and NCPF patients respectively were as follows: 10 to 60 years, 17·62 ± 11·49 and 16 to 50 years, 30·45 ± 11·27. EHO patients were thus significantly younger than NCPF patients (t = 5·23, p < 0·001).

Blood count data for EHO and NCPF was as follows: hemoglobin (g/dl) 9·8 ± 2·4, 9·4 ± 2·2; total leucocyte counts (× 10⁹/mm³) 5·2 ± 2·5, 4·6 ± 2·3; and platelet counts (× 10⁹/mm³) 1·3 ± 0·7, 1·3 ± 0·9. No differences were observed between the two groups (P > 0·05 for all). Mean splenic pulp pressures (cm of normal saline) for EHO (39·2 ± 8·5) and NCPF (36·9 ± 9·0) were also similar (P > 0·05). Mean SCA (cm²) was significantly higher in EHO (207·7 ± 47·9) than in NCPF (155·9 ± 47·9) (t = 5·07, P < 0·001). Using the criteria for ‘hypersplenism’ as described earlier,7 12 of 36 EHO and 10 of 24 NCPF cases had low leucocyte counts (X² = 0·43, P > 0·05); 18 of 31 EHO and 8 of 19 NCPF cases had low platelet counts (X² = 1·4, P > 0·05), and 4 of 18 EHO and 4 of 26 NCPF cases had low values of both the elements (X² = 0·05, P > 0·05). These differences were not significant.

SCA and leucocyte counts had significant inverse correlation with each other within the EHO group (r = −0·36, P < 0·05), within the NCPF group (r = −0·55, P < 0·01) and also when the groups were combined (r = −0·42, P < 0·01) (Fig). SCA had no correlation with hemoglobin (P > 0·05). Although SCA and platelet counts had no correlation within the individual groups (P > 0·05), a significant inverse correlation was observed between the two when the groups were combined (P > 0·05 for all).

Discussion
Our patients with non-cirrhotic causes of portal hypertension (EHO and NCPF) showed a good inverse
correlation between splenic size as assessed by estimation of splenic cross-sectional area and leucocyte and platelet counts, but not with hemoglobin. The degree of portal hypertension assessed by splenic pulp pressure records did not have any correlation either with blood counts or with splenic size. No comparable studies correlating splenic size, blood counts and the degree of portal hypertension are available. In a detailed study from Calcutta\(^5\) of radiological, hemodynamic, histological and hematological features of 100 patients with portal hypertension (25 EHO, 16 NCPF and 59 cirrhotics), somewhat lower counts were found in both EHO and NCPF cases as compared with cirrhotics; platelet counts were lowest in cirrhotics followed by NCPF, and normal in EHO. The study however did not compare the different groups nor did it attempt any correlation, since the thrust of the study was on pathogenesis of portal hypertension. In another study\(^6\) employing the technique of sequestration of \(^51\)Cr-labelled heat-damaged erythrocytes in 24 patients with splenomegaly (19 cirrhotics, 5 due to miscellaneous causes), splenic size was found to have an excellent correlation with the rate of removal of erythrocytes by the spleen, but not with splenic pulp pressures. Although these workers studied predominantly cirrhotics, they suggested that splenomegaly increases sequestration rates regardless of its cause. Using similar techniques, other workers\(^\text{a}\) showed that anemia in chronic liver disease was rarely due to excessive splenic pooling or splenic hemolysis. They however did observe that the splenic platelet pool was considerably increased and platelet life span was reduced. Their conclusion that splenic pooling was not necessarily associated with thrombocytopenia could be attributed to the low numbers of cases they studied.

Our study demonstrates a lack of correlation of splenic size with splenic pulp pressures in cases with non-cirrhotic causes of portal hypertension (EHO, NCPF or both together), similar to what has been shown earlier in cirrhotics.\(^a\) We also showed that although increased splenic size was associated with leucopenia and thrombocytopenia, the latter disturbances did not depend upon splenic pulp pressures per se. Our conclusions are however handicapped by low numbers of patients in whom splenic pulp pressures were available. Hypersplenism is a relatively imprecise term referring to: (a) splenomegaly, (b) any combination of anemia, leucopenia and/or thrombocytopenia, (c) compensatory bone marrow hyperplasia, and (d) improvement after splenectomy.\(^3\) It occurs in 10-15% of cirrhotics, but is rarely of clinical significance.\(^3\) No studies of hypersplenism in NCPF or EHO are yet reported, but we know of data suggesting that the higher frequency of hypersplenism in NCPF than in EHO could be used as a discriminating point (Matsuda V and colleagues, Japan—unpublished data). Hypersplenism is considered to be relatively uncommon in EHO.\(^3\) Although we did not observe any discriminatory value of blood counts in our patients, more observations in this area would be desirable, since such findings could be of practical value while planning therapy. In patients with tropical splenomegaly, thrombocytopenia\(^2\) and leucopenia\(^3\) have been reported, but these studies did not report any correlation with splenic size or pulp pressures.

Lack of correlation between the splenic size and the degree of hypersplenism as well as between the latter and the degree of reticuloendothelial hyperplasia may have relevance to the relationship between the splenic size and blood counts. Our results strongly indicate the need for prospective studies correlating splenic size, splenic sequestration and hematological changes in portal hypertension due to non-cirrhotic causes.

References


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