Thrombocytosis and a Renal Abscess

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Dear Sir,

Following the report of Gafter et al. [1] concerning platelet counts and thrombopoietic activity in patients with chronic renal failure, we would like to present further evidence that thrombopoietin is possibly produced in the kidney and that certain kidney diseases may cause abnormalities in thrombopoietin activity.

A 35-year-old woman was admitted with nausea and vomiting. On examination the patient was pale and afebrile. A large, nontender mass was palpated in the left hypochondrium. The left loin was also nontender. Uri-nalysis revealed a normal sediment with negative cultures. Blood test results included: hematocrit 30%, MCV 71, WBC 15,000/mm³ and a platelet count of 700,000–950,000/mm³. Platelet functions (clot retraction, platelet factor III; platelet aggregation in the presence of ADP and collagen) were all normal. Bone marrow aspiration and biopsy showed normal regeneration in all 3 cell lines, with a normal number of megakaryocytes. Iron was seen in the reticulum.

Radiological examination demonstrated a large left renal mass, cystic, with smooth borders, and with calcified and hypovascular areas seen within the mass. No left hydronephrosis was seen. At explorative laparotomy, a left nephrectomy (weight 1,200 g) was performed. On cut section, kidney tissue had been replaced by a gigantic renal abscess (*Proteus mirabilis*). No malignant cells were found.

A platelet count performed 1 week postoperatively was 160,000/mm³, while 8 months postoperatively the patient remains asymptomatic, with repeated platelet counts of 250,000–350,000/mm³.

Thrombopoietin is believed to play an important role in platelet production via finalizing the maturation process of the young megakaryocytes [2]. Furthermore, studies have shown that thrombopoietin can be produced from cultured human embryonic kidney cells [3] and that bilaterally nephrectomized rats do not become thrombocytotic, as do normal rats, after experimental induction of acute anemia [4]. It seems plausible that thrombopoietin is produced by renal parenchyma, much like erythropoietin. Gafter et al. [1] suggest that the decreased platelet counts in patients with chronic renal failure result from insufficient thrombopoiesis, as evidenced by a normal megakaryocyte count and reduced thrombopoietic activity. Therefore could it not be that renal disease, especially renal masses, may lead to an unregulated and increased thrombopoietin production with subsequent thrombocytosis.

A situation analogous to the above-mentioned one may be the erythrocytosis found in renal tumors or cysts. Urinary tract studies are advisable in patients in whom the cause for thrombocytosis is not known.

References

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