Hypertension and Retinal Vascular Insufficiency Associated With Platelet Microthrombi

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Abstract

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A 45-year-old woman is described who, during the two-year course of her illness, had abdominal pain, gastrointestinal damage, and hypertension. Platelet “white bodies” also were seen to appear in her arterial circulation.

Additional Key Words

white bodies emboli gastrointestinal infarction retinal infarction

A patient is described who, during a two-year course of hypertension, had continual epigastric pain with the passage of undigested food and melena. During the illness, platelet white bodies were photographed circulating in the patient’s arterial system, while at autopsy changes of nonocclusive mesenteric ischemia and hypertension were found.

Case Report

A 45-year-old housewife entered the hospital because of abdominal pain and increasingly severe hypertension moderately well controlled by methyldopa. Both complaints had been present for only two years. The patient stated that for many months a dull epigastric pain frequently occurred after meals. The pain was aggravated by eating, but vomiting eased it. She had lost 10 kg of weight in the preceding four months. She also complained of tiredness, depression, occipital headaches, dysmenorrhea, dizziness, and seeing black spots before her eyes. No history was obtained of renal injury, pre-eclamptic toxemia, urinary tract infection, familial hypertension, or of recent or past loin pain. Except for the last two years, she had enjoyed good health.

On examination the patient was moderately built (55 kg; 155 cm), febrile (37.2°C), and pale. Radial pulse was regular at 90 and the blood pressure was 230/130 mm Hg. The optic fundus showed irregularity of arterial caliber. Slight epigastric tenderness was present. An electrocardiogram showed left ventricular hypertrophy. A chest radiograph showed moderate enlargement of the heart, particularly the left ventricle. The descending aorta was moderately prominent. Increased density of the pulmonary vessels was noticeable in the hila of both lungs. Fast sequen-

tial urography showed the right kidney to be 11.5 cm in length, both renal outlines appearing normal. Excretion on both sides was equal at four minutes, none occurring in the two-minute film. The pericaliceal systems appeared normal. The white cell count was 12,000 per cubic millimeter and ESR (Westergren) dropped 48 mm in one hour. Urinary output of catecholamines, BUN levels, and microbiological examination of urine were within normal limits. After a moderate response to an increased dosage of methyldopa, the patient was discharged from the hospital.

Two months later the patient came into the hospital for four weeks. This followed the onset of prolonged vomiting and diarrhea (seven to eight bowel actions daily) which had started several days previously. Her epigastric pain was worse. Upon examination, the patient was hyperpigmented, with diffuse moderate tenderness present in the upper abdomen. Pathogenic intestinal bacteria could not be isolated.

While in the hospital, the patient experienced a sudden loss of visual field in her right eye, not immediately progressive. The deficit corresponded to an occlusion of the superficial temporal branch of the right central retinal artery. The left eye showed edema at the posterior pole, hemorrhages, “soft exudates,” and early macula fanning, relative vision in the right eye being 6/36 and in the left eye 6/9. Two days later “white bodies” composed of platelet emboli1 were photographed moving along the arterial system of the patient’s right eye (fig. 1). On the temporal side of the eye, small multiple related areas of gray discoloration were present, representing the early degenerative changes of ischemic retinal tissue (fig. 2). This episode was not accompanied by any localizing neurological signs, although the patient complained bitterly of occipital headache and dizziness. Blood pressure readings ranged from 160/105 to 250/150 mm Hg. Skull radiographs did not show any raised intracranial pressure or abnormal calcification. The patient was then discharged.

One month later she again entered the hospital, her condition deteriorating until her death seven weeks later. She had congestive cardiac failure. Her vision deteriorated still further (right eye 6/60 and left eye 6/24), with edema of the macula and “exudate” becoming more marked.

Finally, during the last week of the patient’s life,
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oliguria, left-sided hemiparesis, frank nonocclusive mesenteric infarction, and increasing azotemia developed. A postmortem examination was performed and the findings were consistent with the above clinical diagnoses.

Discussion

Few documented reports exist concerning the pathological processes involved in damage to the small bowel in patients with hypertension. As pathological and biochemical correlations are yet to be made in this field, this patient is interesting because of her continual epigastric pain, the passage of melena and undigested food, the radiographical illustration of severe small bowel damage, the appearance of platelet “white bodies” in her blood stream, and the finding of changes consistent with mesenteric vascular insufficiency at postmortem.

The photographing of white bodies actually moving in a patient’s arterial system is extremely rare. It suggests that similar emboli brought about the arterial occlusion and sudden vision loss in this patient. As such, they should also be considered a cause of the loss of sight and cerebral damage sometimes associated with severe increases in systemic blood pressure. The cause of the bowel damage may be the effect of hypertension. Moritz and Oldt found, in patients with marked hypertension, only two organs (the kidney and the gastrointestinal tract) showed frequent severe arteriolar lesions, while no patients in a similarly examined control group showed any severe changes in these organs.

These observations have been amply confirmed by experimental work. Wilson and Pickering, producing hypertension in rabbits, noticed well-marked acute arterial lesions in the intestine, while Wilson and Byrom found in hypertensive rats that, apart from the kidney, the most severe acute lesions were present in the mesenteric arteries. The classic work of Goldblatt, who found that the first clinical sign of the development of a markedly severe increase in systemic blood pressure was the appearance of blood at the anal verge, suggests that in future studies the contribution of gastrointestinal tract damage to the pathophysiology of sustained hypertension should be further investigated.

![Platelet “white bodies” photographed each two minutes moving in the arterial system of the right eye of a patient who had hypertension and superior mesenteric vascular insufficiency.](image)

Platelet “white bodies” photographed each two minutes moving in the arterial system of the right eye of a patient who had hypertension and superior mesenteric vascular insufficiency. (a) Emboli are seen in the artery just above the major vein occupying most of the bottom of the field. The emboli are seen as white bodies (see arrows). (b) More extensive emboli are now seen approaching a bifurcation in the same artery. (c) Having passed the bifurcation, emboli are seen present in the two arterial branches previously empty of them.

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On the temporal side of the eye, small multiple related areas of gray discoloration show the early degenerative changes of ischemic damage to the retina.

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References

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