Medical Memoranda

Embolism from Mural Thrombus in the Thoracic Aorta

Mural thrombus in the internal carotid artery is a source of emboli to the retinal and cerebral arteries, causing transient or permanent blindness and hemiplegia (Gunning et al., 1946b); thrombi in subclavian arteries damaged by cervical ribs form emboli which lodge in the digital arteries, inducing ischaemia of the fingers and thickened intima (Gunning et al., 1964a). Emboli from mural thrombi in the popliteal artery have been implicated in gangrene of the great toe (Ross, 1936). Mural thrombi on plaques of nodular arteriosclerosis are common in the abdominal aorta, but the frequency with which they form emboli to the abdominal or leg arteries is unknown, the paucity of published cases suggesting the event is either uncommon or unrecognized.

Gross examples of a phenomenon are important because they are easily recognized and may be the extreme variants of events which are commonly unspectacular or undetectable. It is with this possibility in mind that a case in which mural thrombus in the thoracic aorta formed emboli to the abdominal and leg arteries is reported.

Case Report

An unmarried woman (U.O.H. 363216) aged 67 had for many years noticed slight breathlessness and constricting pain in the centre of the chest when walking. She was admitted to a cottage hospital complaining of pain in the right loin, vomiting, and weakness for nine days. Her temperature was 99.6° F. (37.6° C) and blood-pressure 190/90 mm. Hg. No abnormalities were found in the abdomen or chest. The urine contained moderate quantities of protein, but the centrifuged deposit showed neither red nor white cells. The blood urea was 53 mg./100 ml. Her pain continued for five days and several examinations failed to establish its cause.

She was transferred to the Radcliffe Infirmary, where examination showed a sinus rhythm of 96/minute. The only pulses in the legs were the femoral; her blood-pressure was 125/80 mm. Hg. Crepitations were present in both lungs, but there were no cardiac murmurs and no abnormal abdominal movements. The urine had a specific gravity of 1018, it contained between 300 and 1,000 mg. of protein per litre, and the centrifuged deposit had 8 white cells and several hyaline and granular casts per high-power field. The blood urea was 48 mg./100 ml.; plasma sodium 130, potassium 4.2, chloride 90, and bicarbonate 20 mEq/l. The plasma amylase was 7 units. The haemoglobin was 99%; white cells 20,000/cu. mm.—a neutrophil leukocytosis; the sedimentation rate was 97 mm./hour.

The next day her loin pain increased and she complained of pain in the legs. Examination revealed tenderness in the right hypochondrium. The following day her legs were cold and white, and bowel sounds were absent. On the third day she sweated profusely, the pulse was 120/minute and the systolic blood-pressure 50 mm. Hg, despite metaraminol and hydrocortisone. Faeculent material was aspirated from the stomach. She died on the seventeenth day of her illness.

Necropsy three days after death showed fresh infarcts of the same age in the spleen, intestines, and kidneys, the right kidney being completely infarcted. The corresponding arteries contained fresh thrombus, the right renal artery being occluded by thrombus which projected into the aortic lumen. The femoral arteries below the inguinal ligament did not contain thrombus; the leg arteries were not examined. A mural thrombus 3 cm. long occluded the first two intercostal arteries on the right side. The larger upper end was 1 cm. wide and 0.5 cm. deep; its surface was ragged and necrotic. The rest of the aorta showed moderate nodular arteriosclerosis, particularly below the renal arteries, where atheromatous ulcers were filled with thrombus. The coronary arteries showed moderately thickened intima, but they were patent. The heart valves were normal, neither the left atrium nor ventricle contained thrombus, and the foramen ovale was closed. The pulmonary veins were normal, the lungs were congested, and there were small pleural effusions.

The aortic mural thrombus was cut in half longitudinally and embedded in two blocks, from which sections were taken at 200-μ intervals. The ragged surface from the upper end was fresh fibrin thrombus surrounding granular platelet areas; its superficial irregularities were filled by a loose fibrin reticulum, leucocytes, and macrophages, which suggested either lysis of thrombus or thrombus lost as emboli. The centre of this fresh thrombus was necrotic. The underlying thrombus was hyaline fibrin on an intimal plaque 3 mm. thick. The large thrombus occluding the right renal artery was identical with the platelet fibrin thrombus from the thoracic aorta; in the remainder of this and the other occluded arteries there were small platelet fibrin thrombi and clotted blood. The infarcts in the spleen, kidneys, and intestines were the same age. There was no evidence of an arteritis.

Discussion

The diagnosis of embolism requires a source for the embolus, distal arteries occluded by this material, and absence of multiple arterial thromboses or arteritis, all of which were found after death but not detected during life in this patient.

Mural thrombus in the internal carotid artery is suggested by reduced pulsation and bruits; it is confirmed by arteriograms, which in arteries of this calibre show irregularities of the arterial wall. In contrast, mural thrombus in the aorta is undetected, either clinically or radiologically, unless it is massive, because the remaining lumen is still large.

Transient occlusion of retinal and cerebral arteries by emboli is usually associated with striking symptoms and signs (Russell, 1963; Gunning et al., 1964b); however, Skovborg and Lauritzen (1965) observed three symptomless retinal emboli in a diabetic patient. In a detailed clinicopathological study of aorto-iliac thrombosis Gunning et al. (in preparation) found extensive mural thrombus in these arteries, and on histological grounds suggested that emboli of mural thrombus and atheromatous material were to be expected. The case histories, however, did not reveal progress of ischaemic symptoms corresponding to the progressive thrombosis observed anatomically, nor did they suggest episodes of embolism. They concluded that the legs, unlike the retina, brain, and fingers, were not sensitive indices of arterial occlusion unless the blockages were substantial and lasting. Furthermore, Gunning et al.'s study of internal carotid endarterectomy specimens showed fresh mural thrombus if symptoms of retinal and cerebral ischaemia occurred in the seven weeks before endarterectomy, but fresh thrombus was not found in cases with a greater interval between symptoms and surgery. By analogy, fresh thrombus on aortic plaques is a potential source of emboli to the abdominal organs and legs; but to demonstrate such a relationship between mural thrombus and embolism in the lower limb requires extensive histological examination.

Deposits of mural thrombus on the intima of coronary arteries (Duguid, 1946) and aorta (Crawford and Lawrie, 1952) are organized to plaques identical with nodular arteriosclerosis, and homologous pulmonary thrombo-emboli in animals undergo similar changes (McLetchie, 1952). It is probable that emboli of mural thrombus impacted at arterial bifurcations or occluding small arteries are also incorporated into the arterial wall.

In this patient emboli from mural thrombus in the thoracic aorta occluded many distal arteries and caused death. It is likely that the mural thrombi commonly found in the abdominal aorta also form emboli to the legs, which are undetected.
clinically. The organization of these emboli would contribute to the intimal thickening universally found in the leg arteries of the elderly.

It is a pleasure to thank Dr. A. M. Cooke for permission to publish this case and Professor Sir George Pickering for his helpful criticism.

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Chronic Milk-alkali Syndrome after Prolonged Excessive Intake of Antacid Tablets


Rifkind et al. (1960) reported the case of a woman who developed the clinical features of chronic milk-alkali syndrome following prolonged excessive consumption of Rennies tablets. We here report a second case, differing in the absence of generalized osteoporosis.

Case Report

A 40-year-old woman admitted to hospital for investigation on 13 May 1963 complained of excessive thirst for the previous eight years, and because of this her urine had been tested for sugar on several occasions, with negative results. She said that she had sustained rib fractures in 1960 and 1961, once when turning a mattress and once on sneezing. We were unable to obtain the x-ray films taken on these occasions. In July 1962 she first noted spontaneous twitching of the legs and itching of the skin of the trunk and arms. From March 1963 her ankles and feet felt stiff. She then became increasingly tired and found it necessary to give up her secretarial work. Two weeks before admission she had a solitary episode of dysuria, urethral pain, and frequency of micturition lasting for two days. Her appetite remained normal, there was no nausea or vomiting, and her weight was steady.

Further questioning revealed that she had consumed about 24 Rennies tablets daily since 1952. She first took this proprietary antacid to relieve heartburn in her second pregnancy. Heartburn did not recur after the baby was born, but she had acquired a taste for the tablets and continued to take them regularly. Each Rennies tablet contains mag. carb. pond. 78 mg, mag. hydrox. 2.9 mg, mag. oxal. lev. 3.4 mg, calc. carb. 671.6 mg, kaolin. lev. 3.4 mg, calc. phosph. 1.1 mg, and ol. menth. pip. 2.6 mg. The patient was therefore taking some 16 gm. of calcium carbonate daily, in addition to her normal dietary intake. She had also been given tablets containing calcium in 1961, after fracturing a rib, but had taken these for only about two weeks. There was no history of vitamin-D medication. Her average consumption of milk was less than 1 pint (570 ml) daily.

On examination she did not appear acutely ill. Slight generalized brown pigmentation of the skin and numerous scratch marks on her back were present. There were calcium deposits on the palate and lateral sides of both cornae, but no subcutaneous soft-tissue swellings or other abnormal physical findings. Her blood-pressure was 125/90 mm. Hg.

Investigations.—Haemoglobin 10.6 g./100 ml.; M.C.V. 92 cubic microns; M.C.H.C. 30.5%; W.B.C. 6,500/c.mm.; E.S.R. (Westergren) 68 mm./hr.; blood urea 261 mg./100 ml.; plasma sodium 136 mEq/l.; potassium 5.8 mEq/l.; chloride 95 mEq/l.; bicarbonate 21 mEq/l.; serum calcium 11.4 mg./100 ml.; phosphate 5.7 mg./100 ml.; alkaline phosphatase 7.9 King-Armstrong units/100 ml.; serum albumin 3.3 g.; serum globulin 3.5 g. A 24-hour urine sample (on 23 May) contained 0.34 g. protein, 94 mg. calcium, and 410 mg. phosphate. Urinary cell excretion (measured over four hours): W.B.C. 8,600,000/hr., R.B.C. 1,000,000/hr. Two mid-stream specimens of urine were cultured; one grew micrococci and the other Escherichia coli and Streptococcus faecalis. Mantoux test was negative, with 1/1,000 old tuberculin. Chest x-ray picture was normal and no evidence of generalized bone disease was seen on radiographs of the lumbar and thoracic spine, hands, or teeth, the lamina dura being intact. Plain x-ray films of the abdomen and renal tomograms showed small kidneys (each was 9.5 cm. in length) with diffused parenchymal calcification. An attempt at renal biopsy was unsuccessful.

In hospital she accepted the advice to stop taking Rennies tablets and was given a low-protein diet with high oral fluid intake. By 24 May the blood urea had fallen to 150 mg./100 ml., and the serum calcium level was 9.6 mg./100 ml and phosphate 6.8 mg./100 ml. She left hospital on 30 May, and in view of the probable urinary infection was given tetracycline, 250 mg. four times daily, to take at home.

She was readmitted on 6 June complaining of vomiting, thirst, and abdominal pain. She was clinically dehydrated, with blood-pressure 90/70 mm. Hg. The blood urea was 396 mg./100 ml. and plasma bicarbonate 9 mEq/l. She was given intravenous fluids with marked clinical improvement, the blood urea falling to 217 mg./100 ml. The serum calcium was 7.4 mg./100 ml., phosphate 8.7 mg./100 ml. She was discharged home for the second time on 16 June, taking sodium bicarbonate, 2 g. three times daily, to correct acidosis. She was still on this treatment at her most recent review in February 1966. She then looked and felt apart from slight pruritus, which was less severe than formerly. Dysuria had not recurred. Skin pigmentation persisted, and the blood-pressure was 60/30 mm. Hg in each arm. Further investigations in February 1966 showed haemoglobin 12.4 g./100 ml.; E.S.R. (Westergren) 47 mm./hr.; blood urea 95 mg./100 ml.; plasma sodium 144 mEq/l.; potassium 3.4 mEq/l.; chloride 88 mEq/l.; bicarbonate 33 mEq/l.; serum calcium 9.3 mg./100 ml.; phosphate 4.6 mg./100 ml.; alkaline phosphatase 13 King-Armstrong units/100 ml.; serum albumin 4.5 g./100 ml.; globulin 2.8 g./100 ml. Mid-stream urine contained a trace of protein, a few R.B.C.s but no W.B.C.s, and was sterile on culture. Plain x-ray film of the abdomen showed no change in the size of the kidneys or in the degree of nephrocalcinosis compared with the 1963 films.

Comment

The term “milk-alkali syndrome” has been used to describe two separate conditions that may complicate treatment of peptic ulcer. Cope (1936) first reported hypercalcaemia, alkalosis, and temporary impairment of renal function, these abnormalities being corrected by stopping alkali therapy. Our case, however, is an example of the chronic form of milk-alkali syndrome in which renal function remains permanently impaired when alkali are withdrawn. This condition was first described by Burnett et al. (1949), who described the occurrence of the following syndrome in six patients with a history of prolonged and excessive intake of milk and absorbable alkali: hypercalcaemia without hypercalciiuria or hypophosphataemia, normal serum alkaline phosphatase, marked chronic renal insufficiency with azotaemia, mild alkalosis, calcinosis manifested especially by an ocular lesion resembling band keratitis, and improvement in clinical state on a lowered intake of milk and absorbable alkali. Two of these patients had renal

REFERENCES