

## Case Reports

### A CASE OF ANGINA PECTORIS WITH TERMINAL URÆMIA

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This patient, Mrs. S., was admitted to hospital, December 15th, 1927, because of precordial pain. She was 75 years of age and had not been seriously ill since childhood, but had consulted me occasionally during the past five years because of digestive disturbances, and had had a barium series done, with normal findings, two years ago. On March 10th, 1927, at 4 a.m., she had a peculiar seizure with semi-consciousness lasting for thirty minutes, stertorous breathing, loss of sphincter-control, and dyspnoea. There was no irregularity of the pulse and the blood-pressure was not elevated. Precordial pain was present later, as she became normal, but was not a marked feature.

After three or four days in bed, she appeared to be normal again, and was soon up and about under restrictions. Between that date and December 11th she had two similar, but less severe, attacks, with no loss of consciousness and without marked precordial pain.

On December 11th, at 10.30 a.m., she dropped while walking to the bathroom and found herself on the floor. She dragged herself back to her bedroom before help arrived, and immediately complained of severe precordial pain and had marked dyspnoea. She was seen within a few minutes by a doctor, who called me at the hospital, reporting that the attack looked like an anginal one. When seen a little later, the dyspnoea was more marked than on previous occasions, and the precordial pain was the outstanding feature, not being relieved by 1/3 grain of morphine hypodermically.

For the next three days the precordial pain remained the outstanding feature; there was no fever, and no pericardial friction was detected.

On December 15th, she was admitted to hospital, and an electrocardiographic tracing was taken the next morning. To our surprise it was reported to be normal. Examination in the hospital showed similar findings to those recorded outside; a regular pulse of 76-84, with considerable

sclerosis of the vessel walls; a slightly enlarged heart (3 cm./11 cm. at the level of the third rib); normal heart sounds; no friction-rub; and a blood-pressure of 124/80. The first few days in hospital were uneventful, except for a slight daily febrile rise to about 100°, but on December 20th (five days after admission) she became more dyspnoeic and cyanosed, and the lungs filled with fine moist râles. On the evening of December 21st she developed an attack of acute dyspnoea, with all the appearances of uræmia, and a venesection was done at 10 a.m. Her blood-chemistry next morning showed a urea-nitrogen figure of 38, and a creatinine of 2.00. This had changed by December 27th to a urea-nitrogen figure of 79 and a creatinine of 2.14, and by December 31st to a urea-nitrogen figure of 98 and a creatinine of 1.82. Her leucocyte count was 13,400, and the urine never showed more than a trace of albumen. She died on January 2nd.

The striking features, clinically, were the remarkably good quality and evenness of the pulse up till within thirty-six hours of her death, it was usually about 80, the intense dyspnoea; and the constant precordial pain. At no time was a pericardial friction obtained, but, in spite of this and the normal electrocardiograph tracing, we felt that the condition was one of coronary disease with a terminal uræmia.

Unfortunately we did not have a second electrocardiogram taken. This would likely have shown changes and we recognize now that this should have been done.

### CORONARY THROMBOSIS

BY C. C. BIRCHARD, M.B., M.R.C.P. (LOND.)

The case to be reported is one of coronary thrombosis observed in the service of and through the kindness of Professor Campbell P. Howard at the Montreal General Hospital last autumn. It seems to be worthy of report, not so much for its rarity, for actually the condition is quite common, but mainly because it is only during the last few years that internists have acquired facility in its clinical diagnosis and an appreciation of the frequency with which it occurs. Formerly it was labelled in a large