Cardiac Involvement in Psittacosis

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Involvement of the heart during the course of psittacosis is extremely rare if one is to judge from the few reports in the literature. This prompted us to record the following case.

Case Report

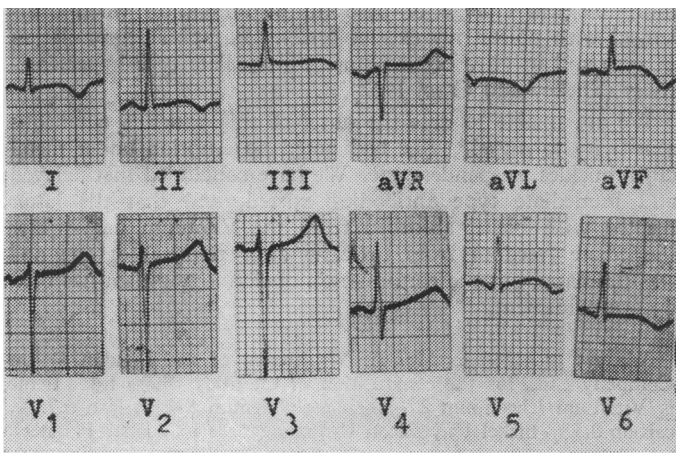
An 18-year-old youth was admitted to hospital on 16 July 1965. Five years previously he had been in hospital because of typhoid. He said that he often caught and handled wild birds only to release them soon after. The last occasion on which he had done this was two weeks before taking ill.

His illness began two weeks before admission with the appearance of severe headache and fever, the latter persisting till admission and varying between 37 and 38° C. Soon after taking ill he experienced pain in his throat and a burning sensation in the retrosternal area, which caused great difficulty in swallowing. In addition, he developed such severe pain in the muscles of both legs that he was unable to stand unassisted.

On examination he appeared to be mildly ill and pale. Oral temperature was 38.8° C., blood pressure 110/80, and radial pulse 90 a minute and regular. The heart was normal in size. Neither cardiac murmurs nor a pericardial friction rub were heard. All peripheral pulses were palpable and equal.

Numerous white shallow ulcers were visible on the soft palate and on the fauces. A few ulcers were seen on the oropharynx. There was no lymphadenopathy. Lungs were clear to percussion and auscultation. The liver and spleen were not palpable. His calf muscles were exquisitely tender on palpation and the slightest movement evoked severe pain. There were no signs of thrombophlebitis. Neurological examination was unrevealing.

The erythrocyte sedimentation rate was 11 mm. an hour (Wintrobe), haemoglobin 12.0 g./100 ml.; leucocyte and platelet counts were normal. Blood urea, electrolytes, bilirubin, alkaline phosphatase, and aspartate aminotransferase were normal. Blood lactic dehydrogenase was 220 units (normal range 83–185), hydroxybutyric



Electrocardiogram taken soon after admission. Negative T waves are seen in leads I, II, aVL, V_5, and V_6.

dehydrogenase 198 units (normal range 55–150), and aldolase 4.2 units (normal range 2.3–6.4). Thymol turbidity, thymol flocculation, and Lugol's test were normal. Serum iron was 30 μg./100 ml. Plasma protein was 8.5 g./100 ml. (albumin 3.5 g., globulin 5 g); electrophoresis on starch gel showed slight hypergammaglobulinaemia. The Wassermann reaction was positive, but reverted to normal five weeks later. Psittacosis complement-fixation test was positive in a titre of 1:320 on 7 July, 1:480 on 25 July, 1:240 on 23 August, and 1:160 on 9 September. Urinalysis was normal. The 24-hour urinary creatine excretion was 154 mg.

Chest *x*-ray examination on admission showed a normal heart and lungs. Fluoroscopy of the heart five days later was normal.

Electrocardiograms, repeated several times during his stay in hospital, showed sinus rhythm at a regular rate of 80 a minute. Negative T waves were seen in leads I, II, aVL, aVF, V_5, and V_6; the Q–T interval was prolonged to 0.42 second (see Fig.). An E.C.G. performed during his previous admission was normal.

The patient's symptoms and signs subsided gradually during a two-weeks stay in hospital. Results of the serological tests were received after complete clinical recovery. Three weeks after discharge he was completely symptom-free, and an E.C.G. showed similar findings to those during his stay in hospital.

Comment

The diagnosis of psittacosis in this case is based on the serological findings, for a titre of 1:16 is regarded as positive (Rivers, 1952). The threefold rise in titre level to a peak of 1:480 is adequate evidence of active infection. The known limitations in interpreting the positive serology can be excluded rather readily. Both cat-scratch fever and lymphogranuloma venereum can produce false-positive reactions (Rivers, 1952; Krysler and Altman, 1961), but the clinical picture in our case confidently excludes these diseases. False-positive serological reactions for syphilis have been described during the course of psittacosis (Allison and Dick, 1954; Grist and McLean, 1964).

The finding of cardiac involvement is the most interesting aspect of this case. This was clearly shown by the electrocardiographic changes in the T waves and prolongation of the Q–T interval. Such changes are indicative of myocarditis. The complaint of retrosternal burning made by our patient suggests that pericarditis may have been present as well, but an unequivocal diagnosis of pericarditis cannot be made in the absence of a friction rub or evidence of effusion.

Clinical accounts of cardiac involvement in psittacosis are few in the literature. The case has been reported of a patient with serologically proved psittacosis who manifested signs of pneumonitis, encephalitis, and myocarditis (Vosti and Roffwarg, 1961). The outcome was fatal. Electrocardiogram showed T-wave inversion in leads I, II, aVL, and V_{1-6} and prolongation of the Q–T interval. Fatal myocarditis in a child was reported in which serological proof of psittacosis was sought (Jannach, 1958). The diagnosis was established after examining tissue preparations obtained from two pet parrots that died three weeks after the child's illness. Characteristic Levinthal–Cole–Lillie inclusion bodies were seen in tissues of mice infected with suspensions of the parrots' tissues, and in the myocardium of the dead child. A case of psittacosis in which myocarditis could be inferred from the clinical picture has been reported, but electrocardiographic evidence was not presented (Valero, 1953).

In addition to the few clinical descriptions of myocarditis some cases have been described in which only pathological evidence of psittacosis myocarditis was presented (Lyon, 1956). In three other cases the mild T-wave changes are not convincing evidence of myocarditis, and the associated manifestations may well have been due to pneumonic involvement. Psittacosis pericarditis is similarly rare and only one case was encountered in the literature of recent years (Schoenemann and Gläsel, 1965).

When admitted to hospital our patient was bothered by two additional symptoms—namely, difficulty in swallowing and inability to stand because of exquisite muscle pain. The dysphagia can be explained by the mucosal lesions in the mouth, and if pericarditis was indeed present it may have contributed as well. The occurrence of oral ulcers has been described in psittacosis (Rivers, 1952). The pronounced calf tenderness was compatible, clinically, with a diagnosis of myositis. However, neither muscle biopsy nor electromyography was performed. The possibility of thrombophlebitis (Rivers, 1952)

seems unlikely in view of the patient's age, the symmetrical involvement, and the severity of the subjective manifestations in contrast with the paucity of the objective findings.

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Urticaria after Insertion of Smith-Petersen Vitallium Nail

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The cause of chronic urticaria is usually elusive. The following case report suggests that the nickel contained in a cast cobalt-chromium alloy (Vitallium) was responsible for symptoms of immediate type allergy.

CASE REPORT

On 11 January 1965 a woman of 65 sustained a fracture of the right femoral neck. The next day a Smith-Petersen Vitallium nail was inserted under halothane anaesthesia. On the day after operation pruritus and generalized urticaria developed, and these symptoms persisted for the next 10 months. During this time there was radiological healing of her fracture, but there was little weight-bearing on the limb on account of anginal congestive cardiac failure and three episodes of cerebral thrombosis. A right-sided hemiplegia followed one of these strokes.

On 8 December she was admitted to the Norfolk and Norwich Hospital for investigation of urticaria. Her major physical findings were right-sided flaccid hemiplegia, generalized urticaria, and dermographism. Laboratory findings were as follows: haemoglobin 13.4 g./100 ml.; W.B.C. 5,600/cu. mm. (neutrophils 70%, lymphocytes 22%, monocytes 5%, eosinophils 3%); E.S.R. 11 mm. in one hour (Westergren); direct Coombs test negative; blood urea 25 mg./100 ml.; total serum proteins 6.62 g./100 ml. (albumin 4.6 g., globulin 2.02 g.). *X*-ray picture of chest and urinalysis were normal.

Discontinuation of current treatment (barbiturates, digitalis, and diuretics) produced no lessening of urticaria, and challenge with the drugs she had received before operation—Omnopon and scopolamine—led to no exacerbation. Routine patch testing showed an eczematous response to a solution of 2% nickel sulphate at 48 hours. A similar response was produced by a Vitallium nail strapped to the thigh for 48 hours. During patch testing there was no increase in severity of urticaria, but severe non-eczematous periorbital oedema developed at 48 hours and resolved within the following 12 hours. A scratch test with 2% nickel sulphate solution resulted in an itching weal (3 cm.) at 10 minutes, and was associated with patchy erythema and swelling of the same forearm for one hour. A passive transfer (Prausnitz–Küstner) test performed on a volunteer was positive. Patch and scratch tests with the other constituents of a Vitallium nail were negative.

Since the patient was bedfast and her fracture had healed clinically, the nail was removed on 10 January 1966 under local anaesthesia. Within 24 hours spontaneous urticaria resolved but dermographism persisted. Exquisite and troublesome dermographism was still present one year after removal of the nail.

COMMENT

At first this patient's urticaria was thought to represent an allergic reaction to drugs given postoperatively. It was she who, for the wrong reason, suggested the correct line of investigation. She reported that during early married life she was said to be "metal sensitive," and that reports about her had appeared in the *British Medical Journal* and the daily press. A search through the *British Medical Journal* of the 1930s showed that she was referred to in a "Queries and Answer" series under the heading of "The Sympathetic Ring" (Jones, 1936). Her practitioner questioned the mechanism involved when the patient's gold ring became the colour of platinum when on her finger but reverted to a golden colour when placed on the mantelshelf overnight. Subsequent editorial comment (*Brit. med. J.*, 1936) summarized correspondence which suggested contact with mercury contained in soaps, ointments, and lotions. The patient had noted no skin change under her ring or other metal object.

Classically, the separate nature of immediate and delayed type allergy has been stressed. However, clinical experience and various reports in the literature suggest that these two types of allergy may exist in the same individual. Calnan (1956) noted the presence of urticaria in his series of nickel-sensitive patients, and Stoddart (1960) reported the case of a patient with delayed contact sensitivity to nickel who developed anaphylaxis after transfusion through a nickel-plated cannula. Shelley and Resnik (1965) studied seven patients with poison ivy dermatitis, and, in addition to showing skin sensitivity to poison ivy oleoresin ether extract, the patients showed the morphological change of basophil degranulation when challenged with poison ivy extract orally. Basophil degranulation in vivo and in vitro occurs during immediate type hypersensitivity (Shelley and Caro, 1962).

We are unable to date the onset of this patient's cutaneous sensitivity to nickel. She had noted no eruption under metal clips, buckles, suspenders, etc. We have concluded that delayed and immediate type antibodies existed, and that the latter reacted with nickel absorbed from the Vitallium nail. Her immediate freedom from urticaria after removal of the nail was dramatic, but the continuation of dermographism has been a disappointment.

We are indebted to Mr. A. W. Laxen, of the London Splint Company Ltd., for the composition of the alloy whose trade name is Vitallium (chromium 27–30%, molybdenum 5–7%, iron 0.75%, carbon 0.5%, nickel 1%, silicon 1%, manganese 1%, cobalt balance).

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