Dental infection as the cause of pyrexia of unknown origin –
two case reports

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Summary: Two cases of pyrexia of unknown origin are described in which no cause was found despite exhaustive inpatient investigation until occult dental infection was detected: extraction of the teeth involved was followed by resolution of the pyrexia. Dental infection should be considered as an unusual but eminently treatable cause of pyrexia of unknown origin.

Introduction

A patient with pyrexia of unknown origin (PUO) always constitutes a diagnostic challenge. In their classical paper published in 1961 Petersdorf & Beeson stated their criteria for the diagnosis of PUO as 'pyrexia of greater than 38.3°C on multiple readings during more than 3 weeks, of which the cause is not discovered after one week of inpatient investigation'.

Since then many publications have appeared which have classified causes of pyrexia of unknown origin into various aetiological groups (infectious diseases, neoplastic diseases, connective tissue diseases and others) some of which suggest a standard investigative approach (Sheon & Van Ommen, 1963; Jacoby & Swartz, 1973; Gleckman et al., 1977; Esposito & Gleckman, 1978; Esposito & Gleckman, 1979; Larson et al., 1982). Dental infections as a cause for PUO were not mentioned until the last decade (Berry & Silver, 1976; Levinson & Barondess, 1979).

We describe two patients with PUO. The cause of the pyrexia was discovered to be dental infection only after one month of hospitalization, during which time other causes had been excluded.

Case reports

Case 1

A 57 year old man was admitted to the Internal Medicine ward with retrosternal pain. Six months previously he had had an acute myocardial infarction, and since then he had been treated with quinidine, nifedipine and coumadin. Physical examination on admission revealed a generally fit man, with no abnormality except for a pyrexia of 38°C. Electrocardiogram showed only signs of an old inferior wall myocardial infarction.

Repeated laboratory investigations including erythrocyte sedimentation rate, full blood count, blood glucose and electrolytes, kidney and liver function tests, calcium, phosphorus, uric acid, amylase, and protein immunoelectrophoresis were all normal. Serological tests including Weil-Felix, Paul-Bunnell, Widal, Brucella antibodies, rickettsial complement fixation tests, cytomegalovirus and Epstein-Barr virus antibodies, rheumatoid latex test, antinuclear antibodies, VDRL, were all negative. Cultures of blood, urine, throat, faeces and cerebrospinal fluid were all sterile. A Mantoux test was negative. Stool examination for parasites was negative. Chest X-rays, sinus X-rays, intravenous pyelography, barium meal and enema, were all normal. Computed tomographic scan and ultrasound examinations of the abdomen were normal.

Because of the possibility of drug fever all medications were discontinued but fever persisted. A therapeutic trial of chloramphenicol caused the patient's temperature to return to normal, but the pyrexia returned when the chloramphenicol was discontinued.

After a month of investigation, the patient began to complain of pain in his teeth. Dental examination including panoramic dental radiography revealed advanced periodontitis. All the patient's teeth were extracted in stages and the pyrexia resolved gradually. One

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year after discharge the patient remains symptom-free.

Case 2

A previously healthy 61 year old man was admitted to the Internal Medicine ward with a 6 week history of pyrexia of up to 38°C every evening, frontal headache, fatigue, anorexia and a gradual loss of weight (6 kg). On admission he appeared pale and tired. There were no abnormalities on physical examination except for temperature of 38.2°C, small bilateral submandibular lymph nodes and widespread dental caries.

Laboratory investigations showed anaemia (haemoglobin 10 g/100 ml) with an erythrocyte sedimentation rate 70–100 in the first hour. The white blood cell count and differential, blood urea, glucose, electrolytes, calcium, phosphorous, alkaline and acid phosphatase, total proteins and liver function tests were all normal. LE cell test was negative. Serological examinations for Vidal, Weil-Felix, Brucella and rickettsial antibodies, Paul-Bunnel and VDRL were negative. Cultures of blood, urine, sputum, throat and stool were all normal. Sputum and urine cultures for Mycobacterium tuberculosis were normal. X-ray of chest, sinuses, skull, gallbladder, kidneys and digestive tract and bone marrow examination including culture, were normal.

Pyrexia and headache persisted in spite of a week's course of chloramphenicol. One month after admission all decayed teeth were extracted with subsequent resolution of the pyrexia and headache. He was discharged afebrile and remained well on follow-up.

Discussion

Despite advances in diagnostic techniques, PUO still constitutes a diagnostic challenge, especially in otherwise asymptomatic patients. The well-recognized causes of PUO include infections, malignant tumours, connective tissue diseases, and various uncommon diseases. In all of the published series, the percentage of patients who remain undiagnosed after thorough investigation varies between 5–39% (Esposito & Gleckman, 1979; Larson et al., 1982). During the last decade dental infection has been identified as a cause of PUO (Berry & Silver, 1976; Levinson & Barondess, 1979).

Most patients described, including ours, lacked specific symptoms, but pyrexia remitted after dental extractions. There is no doubt that dental infection must be included in the long list of possible causes of PUO as an eminently treatable condition.

References

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