

Case reports

Case 1—A 73 year old man, admitted because of palpitations, was started on quinidine sulphate 250 mg four times daily after a 24 hour Holter electrocardiogram recording had shown supraventricular tachyarrhythmia. He gave no history of physical or mental illness and was taking no medication. The results of physical examination and routine laboratory tests were normal, apart from a creatinine clearance of 54 ml/min. Some 90 minutes after the first dose of quinidine he developed visual hallucinations, delusions, and psychomotor agitation. A plasma quinidine concentration at that time was 0.8 mg/l (therapeutic values 3-6 mg/l). Twenty four hours later he had recovered completely.

Case 2—In a 67 year old man with palpitations an electrocardiogram showed recurrent supraventricular arrhythmia; treatment with quinidine sulphate 250 mg thrice daily was started. He had a history of mild hypertension but was receiving no treatment. Physical examination before the administration of quinidine was said to have shown no signs of cardiac failure and his blood pressure was 175/95 mm Hg. Routine laboratory investigations gave normal results, apart from a creatinine clearance of 64 ml/min. Two hours after receiving his first dose of quinidine he developed psychotic features consisting of psychomotor hyperactivity with paranoia and vocal hallucinations and was admitted to hospital. These features gradually subsided over 20 hours; his plasma quinidine concentration on admission was 1 mg/l.

In both patients the results of physical and neurological examination as well as of the laboratory investigations (liver function values, serum electrolyte concentration, and blood gases) were normal during the psychotic state. Additional neurological investigations (computed tomography of the brain and electroencephalography) showed no organic brain disease.

Comment

Though both our patients had impaired renal function, quinidine elimination has been said to be unimpaired in patients with poor renal function.^{3,4} Furthermore, plasma quinidine concentrations were subtherapeutic in both patients, which makes quinidine toxicity unlikely. Hence we assume that idiosyncrasy was the responsible mechanism.

So far we have found no report of psychosis presenting as the only manifestation of idiosyncrasy to quinidine. Given that the drug is widely used, we believe that this possibility should be borne in mind.

- 1 Goodman LS, Gilman A. *The pharmacological basis of therapeutics*. New York: MacMillan, 1980.
- 2 Reynolds JEF, ed. *Marindale. The extra pharmacopoeia*. London: Pharmaceutical Press, 1982.
- 3 Kessler KM, Lowenthal DT, Warner H, Gibson T, Briggs W, Reidenberg MM. Quinidine elimination in patients with congestive heart failure or poor renal function. *N Engl J Med* 1974;290:706-9.
- 4 Swisher WP, Wedell HG, Cheng JTO, et al. Studies of quinidine plasma levels and rate decline following cessation of quinidine administration. *Am Heart J* 1954;47:449-52.

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Is smoking a risk factor for pneumonia in adults with chickenpox?

Pneumonia is a serious complication of chickenpox in adults, with a mortality of up to 20%.¹ Risk factors have not previously been shown; identification of patients with such risk factors offers the possibility of antiviral chemoprophylaxis. We report on 29 patients admitted with varicella infection.

Patients, methods, and results

We studied 29 adults admitted from March 1985 to February 1986 with cutaneous varicella infection. Smoking histories were taken, and chest radiography and tests of lung function (forced expiratory volume in one second, forced vital capacity, the forced expiratory flow between 25% and 75% of the forced vital capacity (FEF₂₅₋₇₅), and carbon monoxide transfer factor) were performed in 20 of the patients on admission. Fisher's exact test was used for statistical analysis.

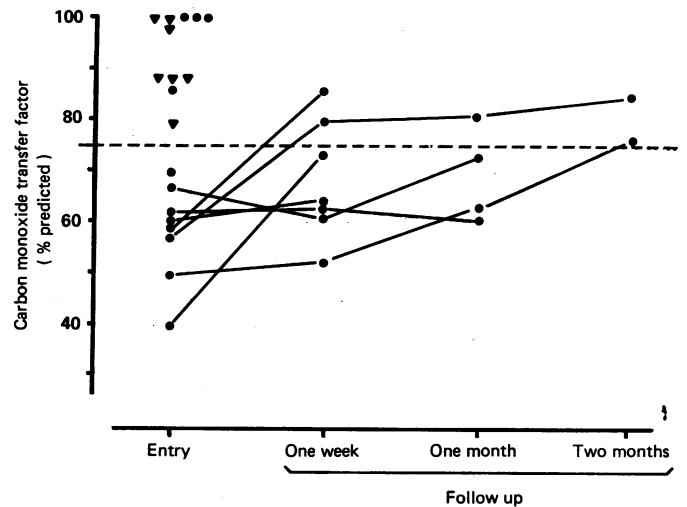
Of the 29 patients, 19 were regular or recent smokers and 10 had never smoked. Seven of the 19 smokers but none of the non-smokers had pneumonia ($p=0.032$).

The 20 patients who underwent lung function tests comprised 13 men (mean age 31 (SD 12)) and seven women (mean age 27 (6)); 12 were regular or recent smokers and eight had never smoked. Of the 12 smokers, five developed clinical pneumonia (confirmed radiologically), another three had an abnormal carbon

monoxide transfer factor (<75% predicted) but no evidence of pneumonia, and four had no evidence of pneumonia or a reduced carbon monoxide transfer factor. One smoker with pneumonia was pregnant. None of the eight non-smokers tested developed pneumonia; only one had an abnormally low carbon monoxide transfer factor, but she had a history of extensive pulmonary tuberculosis. The association between smoking and low carbon monoxide transfer factor was significant ($p=0.0066$). All patients with radiologically confirmed pneumonia received intravenous acyclovir 10 mg/kg eight hourly for five days; all had improved considerably at one week. None required ventilation.

Seven of the eight smokers with a low transfer factor had serial lung function tests at one week, one month, and two months after treatment or until results were normal. The figure shows the findings in these smokers, although several were lost to follow up. One of the patients with a low carbon monoxide transfer factor at one month was retested at six months, when the value was low normal.

Ventilatory function was normal (>75% predicted) in all patients except for the FEF₂₅₋₇₅ value, which was reduced (<75% predicted) in patients with pneumonia but improved as the carbon monoxide transfer factor improved, except in one patient. In the three smokers with a reduced carbon monoxide transfer factor without pneumonia the FEF₂₅₋₇₅ was normal. Chest radiography in all patients at follow up did not show any evidence of obstructive airways disease or emphysema; there was no history of respiratory disease in any patient.



Carbon monoxide transfer factor in the patients admitted with chickenpox. (The patient with tuberculosis was excluded.) Broken line indicates lower limit of normal. ●=Smoker. ▼=Non-smoker.

Comment

Chickenpox pneumonia occurred only in smokers, an association not previously reported. The carbon monoxide transfer factor was reduced in some smokers without pneumonia; their normal ventilatory function suggests that this was due to subclinical chickenpox pneumonitis rather than smoking itself. The reduction of carbon monoxide transfer factor in our patients with chickenpox pneumonia confirms previous findings,² but such a reduction has not been described in adults without pneumonia. We also showed a reduction in FEF₂₅₋₇₅, indicating small airways obstruction.

Pneumonia occurs more commonly in adults than children with chickenpox. In severe cases assisted ventilation is necessary, but since acyclovir became available in 1981 none of the patients admitted to this unit with chickenpox (some with pneumonia) has required it (B K Mandal, personal communication). It may, therefore, be worth while giving acyclovir to adults with chickenpox who smoke.

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- 1 Knyvett AF. The pulmonary lesions of chickenpox. *Q J Med* 1966;139:313-23.
- 2 Bocles JS, Ehrenkrantz NJ, Marks A. Abnormalities of respiratory function in varicella pneumonia. *Ann Intern Med* 1964;60:183-95.

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