While she was in hospital the "itching spots" developed: these were pink, slightly raised papules of 1.5-2.5 mm in diameter scattered over the whole body. Skin biopsy specimens were immediately taken from a new lesion and from nearby normal skin. Immunofluorescence staining showed the presence of tiny deposits of IgM and complement around small vessels in the upper dermis and slight mononuclear perivascular infiltrate. In the normal skin the reaction was far less pronounced. Traces of IgM were present perivascularly, but there was little complement. These changes were considered to be compatible with the deposition of circulating IgM immune complexes and activation of complement causing vasculitis.

Comment

As IgM is normally absent in the deposits of membranous glomerulonephritis the presence of IgM in the glomeruli of our patient (and the patient of Rai *et al*³) is remarkable. Our patient also shows that, though IgM is present in the normal skin of patients with primary biliary cirrhosis,⁵ cutaneous lesions and vasculitis occur only in the presence of immune complexes containing IgM and complement. In patients with primary biliary cirrhosis a high proportion of immune complexes contain IgM; most are complement fixing.² We believe that this report strengthens the case for IgM glomerulonephritis and cutaneous vasculitis being extrahepatic manifestations of primary biliary cirrhosis, which may well be mediated by the deposition of complement binding IgM immune complexes.

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Somatostatin in paroxysmal supraventricular and junctional tachycardia

Somatostatin acts as a negative inotropic agent in isolated spontaneously beating auricles of guinea pigs. It produces this effect by direct action on atrial myocardial cells, possibly by reducing the influx of calcium ions into these cells.¹ It has been shown that the cholinergic postganglionic neurons of the cardiac vagus contain material similar to somatostatin that appears to be tetradecapeptide somatostatin.² We observed a decrease in heart rate in a group of patients with psoriasis treated with somatostatin.³ These experimental and clinical observations led us to give intravenous somatostatin to six patients with paroxysmal supraventricular and junctional tachycardia to see whether it would restore sinus rhythm.

Patients, methods, and results

We studied four women and two men with recurrent attacks of electrocardiographically documented paroxysmal supraventricular or junctional tachycardia who had been admitted to hospital. They comprised a 62 year old woman (case 1) with paroxysmal supraventricular tachycardia; two

women aged 38 and 40 (cases 2 and 3), with paroxysmal supraventricular tachycardia in the Wolff-Parkinson-White syndrome; a 71 year old man (case 4) with paroxysmal supraventricular tachycardia in the sick sinus syndrome and with a permanent cardiac pacemaker; and a man and a woman aged 34 and 27 respectively (cases 5 and 6) with paroxysmal junctional tachycardia.

We observed the patients 30 minutes to 24 hours after the onset of paroxysmal tachycardia. Administration of cyclic somatostatin (Serono, Italy) was started 10-15 minutes after ineffective vagal manoeuvres. Altogether 25 µg somatostatin in 0.154 mol saline (1/10 volume/volume dilution) was injected as a bolus every two minutes while continuous electrocardiography was performed. Voluntary informed consent was obtained from each patient.

Electrocardiogram in lead II showing transition from paroxysmal supraventricular tachycardia to sinus rhythm in case 3. Paper speed 25 mm/s.

Sinus rhythm was restored in five of the six patients, after administration of 175-200 µg somatostatin—that is, about 15 minutes after the start of treatment. The figure shows the transition from paroxysmal supraventricular tachycardia to sinus rhythm in case 3. Despite the administration of 500 µg somatostatin intravenously sinus rhythm was not achieved in the remaining patient, who had paroxysmal junctional tachycardia; a decreased ventricular rate with variable but transient atrioventricular dissociation was observed, however, soon after the treatment.

Comment

This report shows that intravenous administration of somatostatin may interrupt paroxysmal supraventricular tachycardia in man. Somatostatin is a potent inhibitor of several hormonal secretions. This effect has been related to an impaired uptake of calcium ions, which may be the common mechanism for the blocking action of somatostatin in different secretory cells.⁴ In addition, in isolated β cells somatostatin reduces the accumulation of intracellular calcium by its primary activation of cell membrane permeability to potassium ions.⁵ In guinea pigs somatostatin produces a negative inotropic action associated with a calcium antagonist effect, probably dependent on binding to specific atrial receptors.²

The effect of somatostatin on supraventricular tachycardia is probably caused by an action on the atrioventricular node, related most probably to a calcium antagonist effect. Somatostatin has no effect on sodium or potassium adenosine triphosphatase⁶ and does not have a beta blocking action.¹

In *Bufo marinus* toads stimulation of the vagus nerve evokes release of somatostatin with a consequent decrease in the heart rate and force of atrial contraction.² Possibly in man too somatostatin is present in vagal fibres and may play a physiological part in the autonomic control of cardiac action. Its minimal untoward effects and absence of toxicity make it a possible agent for treating arrhythmias.

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