Learning points

- many drugs may induce a severe pressor reaction in patients with a phaeochromocytoma
- these drugs should not be administered to patients with hypertension when phaeochromocytoma is a possible diagnosis

and sulpiride. Some common drugs such as corticosteroids, insulin, morphine, pethidine, monoamine oxidase inhibitors and tricyclic antidepressants, have been known to elevate the blood pressure in phaeochromocytoma. Most physicians are aware of the paradoxical rise of blood pressure with β-adrenergic blockers given alone in phaeochromocytoma without concomitant alpha-blockade.

With out patient it was fortunate that two of us (SF and MRL) were aware of the interaction and, in the presence of neurofibromatosis, the diagnosis was then straightforward. Nevertheless, we feel that this dangerous effect of a very commonly used anti-emetic should be emphasised more strongly.

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Fibrate monotherapy and profound hypoalphalipoproteinemia

Sir,

A proposed benefit of fibrate therapy in dyslipidaemia is to increase serum high density lipoprotein cholesterol (HDLC) concentrations. A recent report by Murphy et al described two cases of profound reductions in serum HDLC and apolipoprotein A1 concentrations following combination therapy with probucol and a fibrate, ie, bezafibrate and fenofibrate, respectively. The authors postulated an interaction between probucol and the fibrates as the underlying cause of this phenomenon. However, substantial paradoxical reductions in HDLC have also been reported in association with fibrate monotherapy (ie, ciprofibrate, bezafibrate), although the pathogenesis is unknown. It is also unclear how soon after commencing monotherapy with the fibrate the fall in HDLC occurs or whether the fall is transient or persistent. We report observations which address these questions in a patient who exhibited this unusual response to ciprofibrate.

Case report

A 41-year-old hypertensive woman presented with mixed hyperlipidaemia exacerbated by excessive alcohol intake. Her antihypertensive medication was nifedipine. She had been treated with diet and bezafibrate over a three-year period during which HDLC ranged between 1.12 and 1.34 mmol/l. Apo E phenotype was confirmed as E3/3. Due to inadequate response to this therapy, her medication was changed to ciprofibrate 100 mg daily. Two weeks later, total cholesterol had fallen from 9.7 to 8.5 mmol/l but this was accompanied by a precipitate fall in HDLC from 1.05 to 0.12 mmol/l. This very low HDLC concentration was reflected in an equally low apolipoprotein A1 concentration of 0.12 g/l (reference range: 0.91–1.70). It was decided that ciprofibrate should be continued at the same dose but with fortnightly clinical and biochemical monitoring of the patient to assess whether this effect on HDLC was sustained. This therapeutic decision was discussed fully with the patient. Subsequent HDLC levels fluctuated between 0.12 and 0.80 mmol/l with four out of seven values being less than 0.20 mmol/l (figure). During this period, the median and range for total cholesterol and triglycerides were 8.5 (7.7–9.7) mmol/l and 4.8 (2.5–9.5) mol/l, respectively.

Ciprofibrate was stopped at three months because of lack of improvement in her lipid profile and two weeks later HDLC had increased from 0.13 to 0.90 mmol/l. Six weeks after discontinuing the medication, HDLC was 0.88 mmol/l (figure). The patient did not report any adverse clinical effects during her treatment with ciprofibrate.

This case demonstrates that substantial anomalous reductions in HDLC can occur in susceptible individuals in association with ciprofibrate monotherapy. This phenomenon may become evident as early as two weeks after initiating treatment which is much less than the four to six months reported in previous cases. Our patient also demonstrated wide variability in HDLC concentrations during treatment. Although most of the HDLC values were very low, there was a near-normal value of 0.80 mmol/l obtained 10 weeks into treatment. If the HDLC level had been assessed only at this time point, the very low values could have been missed. It is important that HDLC should be included in the routine monitoring of patients on lipid-lowering medication.

Fibrate monotherapy and profound hypoalphalipoproteinaemia.

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