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SHORT REPORTS

Cerebral ischaemia after nifedipine treatment

Nifedipine, a calcium antagonist with strong vasodilating effects, is particularly effective in the treatment of angina pectoris,[1] chronic cardiac failure,[2] and systemic hypertension.[3] A low incidence of side effects has been reported.[3] Nevertheless, two recent reports draw attention to the risk of pulmonary oedema[4] and severe hypotension.[5] We recently observed two patients who developed cerebral ischaemia a few hours after taking nifedipine.

Case reports

Case 1—A 72-year-old man was admitted to hospital because of abrupt onset of right hemiparesis and aphasia. Ten years before ischaemic heart disease and slight diabetes mellitus had been diagnosed. He was treated with medigoxin 100 μg, glibenclamide 2·5 mg, and phenformin 25 mg daily. An electrocardiogram performed three days before admission showed atrial fibrillation, ectopic sporadic ventricular contractions, incomplete left bundle branch block, left ventricular hypertrophy, and signs of subendocardial ischaemia. For this reason, nifedipine 10 mg three times a day and hydrochlorothiazide 50 mg and amiloride 5 mg daily were added to treatment. On the second day of treatment, two hours after taking a nifedipine tablet, the patient suffered aphasia and right-sided weakness which lasted for a few minutes. The next morning the patient took another nifedipine tablet, and one hour later he developed the same disturbances. On admission to hospital the systemic arterial pressure and the pulse rate were normal and there were no clinical signs of cardiac failure. The neurological examination showed aphasia and right hemiparesis. Findings on electrocardiogram were similar to those previously noted. The chest radiograph showed slight pulmonary congestion and an enlarged heart. An echocardiogram showed left atrial and ventricular enlargement. Computed tomography showed an ischaemic area in the left hemisphere. Ultrasound examination showed no sign of stenosis or occlusion of extracerebral vessels. Two days after admission, the patient improved dramatically, and neurological examination showed no abnormality. Nifedipine treatment was discontinued, and there have been no further episodes in the following months.

Case 2—A 67-year-old woman was admitted to hospital for sudden loss of consciousness. She had long-standing systemic hypertension treated with hydrochlorothiazide 50 mg daily. Some days before admission nifedipine 10 mg three times a day was added to treatment to control hypertension. On the second day of treatment, two hours after taking nifedipine, the patient suddenly lost consciousness. Once awakened she complained of headache, drowsiness, and vertigo. On admission to hospital, systemic arterial pressure was 170/100 mm Hg and pulse rate 84/min. Neurological examination showed signs of cerebellar dysfunction. Signs of left ventricular hypertrophy and subendocardial ischaemia were present on electrocardiogram. The chest radiograph showed aortosclerosis. Findings on computed tomography were normal. The patient was discharged taking clonidine 150 mg and hydrochlorothiazide 50 mg daily; nifedipine was discontinued. There were no further occurrences during the follow-up.

Comment

Both our patients presented risk factors for development of cerebrovascular diseases. Nevertheless, the temporal relation between taking the drug and developing the symptoms raises the suspicion that the drug might have contributed to these episodes. In our view two possible mechanisms could be implicated: (*a*) transient severe systemic hypotension (not observed in our patients at admission to hospital); (*b*) a more effective vasodilating action of the drug on the peripheral resistance vessels rather than on cerebral vessels. Both factors could be the consequences of an unpredictable action of the drug in aged patients. To our knowledge, this is the first report of cerebrovascular disorders during nifedipine treatment. Further reports could indicate whether our observations were coincidental or whether nifedipine could actually precipitate cerebral ischaemia in aged patients with risk factors for cerebrovascular diseases

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Hepatitis B infection in glomerulonephritis

Persistent viral infections are well recognised causes of glomerulonephritis in animals. The only persistent virus infection in man which has been regarded as a common cause of chronic glomerulonephritis is hepatitis B: the prevalence of hepatitis B carriage in reported series of patients with glomerulonephritis has ranged from 0 to 50%.[1] The higher figures have important implications for dialysis and transplantation, as well as throwing an interesting light on the cause of glomerulonephritis. We therefore examined the prevalence of markers of hepatitis B infection in patients with glomerulonephritis in Newcastle upon Tyne.

Patients, methods, and results

From 1976 to 1980 we investigated 158 patients with various types of glomerulonephritis attending the renal clinic at the Royal Victoria Infirmary. One hundred patients with a similar age range and sex distribution who were suffering from other types of renal disease (polycystic kidneys, chronic pyelonephritis, essential hypertension) were studied as controls. The mean age (±1 SD) of the patients with glomerulonephritis was 39±16 years (range 15-76; 90 men, 68 women) and that of the controls 42±15 years