# Psoriasis as a side effect of β blockers

Doctors have been slow to recognise some of the side effects of β blockers in clinical practice. The oculomucocutaneous syndrome induced by practolol was not detected until experience with the drug had totalled one million patient years.[1] The most common cutaneous reactions to β blockers other than practolol include psoriasiform, lichenoid, or eczematous skin reactions.[2] β Blockade may also trigger true psoriasis.[2]

We followed the course of psoriasis that had erupted or worsened during β blockade once the β blocking agent was withdrawn.

## Patients, methods, and results

Ten patients receiving β blockers whose psoriasis had appeared (n=6) or noticeably worsened (n=4) during treatment were studied. Four patients showed characteristic nail changes (pitting and onycholysis), and three had joint symptoms. Two had skin lesions typical of palmoplantar psoriasis. The table shows the clinical details of the patients.

The β blocker was withdrawn from all patients except one (case 1), who was given an alternative β blocker because of aggravated anginal pain. In hypertensive patients the β blocker was replaced by calcium antagonists or angiotensin converting enzyme inhibitors, or both, and patients with coronary heart disease were given calcium antagonists and nitrates.

The extent of psoriatic lesions was examined in each patient. Antibodies to serum deoxyribonucleic acid and extractable nuclear antigens were determined, serum protein electrophoresis performed, and blood variables measured. Epicutaneous tests were performed in all cases with a standard basic series and eight different β blockers with 10% β blocker in white soft paraffin. Biopsy samples were also taken for direct immunofluorescence. Control examinations were performed in all patients one, three, six, and 12 months after the β blocker was stopped.

No complications resulted from changing the treatment. Epicutaneous tests gave negative results for both the basic series and the β blockers. Direct immunofluorescence gave negative results. Antinuclear antibodies were not detected, and no changes were found in blood variables or serum proteins.

In four patients (cases 1, 6, 8, and 10) psoriatic skin lesions healed completely after withdrawal of the drug; in case 6, however, the patient still had nail lesions. In all four patients psoriatic lesions had appeared during β blockade. In five others roughly 80% of the psoriatic skin lesions healed. In one patient (case 5) psoriasis recurred fully after 11 months' remission. Healing of nail lesions was almost equally good. In cases 1 and 10 recovery was complete, and in cases 4 and 6 the changes were noticeably reversed. In cases 3 and 4 about 90% of the palmoplantar psoriasis disappeared.

One patient (case 4) was rechallenged after seven months when he received atenolol for four months; psoriasis erupted over the entire body surface within two months.

## Comment

β Blockers have been reported to be associated with the recurrence or aggravation of psoriasis.[2-4] Arntzen *et al* found that seven of 23 patients with psoriasis had developed the disease while taking β blockers. The drug was withdrawn in four of the patients, and three of them recovered totally.[5] In the present series psoriatic lesions either resolved totally or were noticeably reduced after withdrawal of the β blocker in nine patients; there was one recurrence.

Psoriasis may be an inconvenient side effect of β blockade. We therefore suggest that β blockers be replaced by alternative treatment whenever safely feasible in all patients where psoriasis has developed or worsened during β blocker treatment.

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*Clinical details of patients*

| Case No | Age and sex | Type of psoriasis | Result of biopsy | Treatment triggered or worsened psoriasis | β Blocker taken before study and daily dose | Duration of β blockade before onset or worsening of psoriasis (years) | Other adverse effects | Recovery (%) after β blocker stopped |
|---|---|---|---|---|---|---|---|---|
| 1 | 70 F | Psoriasis vulgaris, nail psoriasis | Psoriasis vulgaris | Triggered | Metoprolol 100 mg | 0·5 | None | 100, 100*† |
| 2 | 85 F | Psoriasis vulgaris | Psoriasis vulgaris | Triggered | Propranolol 20 mg | 1·0 | None | 80 |
| 3 | 73 F | Palmoplantar psoriasis | Not done | Triggered | Timolol 10 mg | 2·0 | None | 90 |
| 4 | 56 M | Psoriasis vulgaris, nail psoriasis, palmoplantar psoriasis | Psoriasis vulgaris | Worsened | Atenolol 50 mg | 1·5 | Ocular changes, dry mouth, arthropathy | 80, 50† |
| 5 | 55 M | Psoriasis vulgaris | Psoriasis vulgaris | Worsened | Metoprolol 200 mg | 1·0 | Dry mouth, impotence, cold legs | Relapse |
| 6 | 37 F | Psoriasis vulgaris, nail psoriasis | Psoriasis vulgaris | Triggered | Metoprolol 50 mg | 0·3 | Arthropathy | 100, 50† |
| 7 | 53 M | Psoriasis vulgaris | Psoriasis vulgaris | Worsened | Propranolol 80 mg | 1·0 | None | 90 |
| 8 | 64 M | Psoriasis vulgaris | Psoriasis vulgaris | Triggered | Propranolol 80 mg, metoprolol 100 mg, atenolol 50 mg | 0·5 | Arthropathy, dry mouth | 100 |
| 9 | 53 M | Psoriasis vulgaris | Psoriasis vulgaris | Worsened | Atenolol 50 mg, pindolol 10 mg | 0·5 | None | 70 |
| 10 | 49 M | Nail psoriasis | Not done | Triggered | Labetalol 100 mg | 0·4 | None | 100 |

*β Blocker was continued (acebutolol 100 mg/day).
†First figure relates to psoriasis vulgaris, the second to nail psoriasis.

# Successful treatment of multiple liver metastases by liver perfusion

The prognosis of patients with liver metastases associated with colorectal cancer is poor: the average survival is about six months, and although there are reports of long term survival, virtually all patients are dead within two years.[1] Wood suggested, however, that although most patients with widespread metastases are dead within two to three years, 16% of those with solitary metastases are alive at five years.[2]

A recent report suggested that perfusion of the liver with heparin and fluorouracil reduces the development of liver metastases postoperatively.[3] We report on a man with multiple tumour deposits in the liver who survived for four years after treatment with perfusion.

## Case report

A man of 62 presented with a carcinoma of the right colon. At laparotomy he was noted to have multiple (pin head) tumour deposits in the left lobe of the liver; the right lobe was clear and there were no peritoneal seedlings. A right hemicolectomy was performed (no glands were affected). Subsequent histological examination confirmed secondary adenocarcinoma in the liver. Each lesion was