Psoriasis and gout

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Summary

Ten cases of gouty arthritis are described in association with psoriasis. Eight were receiving intensive inpatient treatment for their skin condition. Diagnosis was based on clinical grounds or, in 3 cases, by compensated polarizing microscopy (CPM) of synovial fluid. All patients were male and 5 of them had conditions other than psoriasis known to predispose to hyperuricaemia. The patients appeared to fit into three groups: five had typical lower limb gout occurring in conjunction with long-standing extensive psoriasis; 3 patients had preceding features of inflammatory synovitis, one of whom subsequently developed typical distal interphalangeal involvement of peripheral psoriatic arthritis; two patients appeared to have coincident gout and psoriasis.

We believe that an association of gout with extensive long-standing psoriasis may exist particularly in male patients with an additional cause for hyperuricaemia. Long-term studies of a large population of psoriatics are required. Previous reports may have underestimated the incidence of gout because of failure to examine synovial fluid for crystals particularly from those patients with a subacute large joint arthropathy.

Introduction

The clinical features of gout are usually those of attacks of acute severely painful monoarthritis commonly affecting the big toe. Untreated such attacks resolve over 3 to 10 days; prompt treatment with colchicine or non-steroidal anti-inflammatory drugs relieves symptoms over 24 to 48 hr. Urate crystals are present in the synovial fluid and prolonged hyperuricaemia is a prerequisite for gout to occur. Hyperuricaemia can be due to an inherited metabolic abnormality as in primary gout, or secondary to an acquired condition.

Psoriatic arthritis takes at least 5 different forms, (Moll and Wright, 1973a), all of which are characterized by absence of rheumatoid factor and features of an inflammatory arthropathy associated with the psoriatic diathesis. Attacks of gout in patients with psoriasis have been described (Bonim and Kimberg, 1962; Kaplan and Klatskin, 1960; Kuzell et al., 1955; Leonard, O'Duffy and Rogers, 1978; Moll and Wright, 1973b; Venkatasubramanian, Blumm and Riddle, 1980; Zimmer and Demis, 1966). Some of these studies have suffered from being of a retrospective nature and diagnostic criteria for gout have not always been clear. To the best of our knowledge in none of these series were urate crystals identified in the synovial fluid. However, the study of Baker (1966) failed to find one case of gout in 650 cases of psoriasis even though he was looking specifically for arthritis. We report here our experience of the study of a large in-patient dermatological population over a period of 11 years.

The patients

The patients studied were seen at St John's Hospital for Diseases of the Skin between 1969 and 1980. All were in-patients except patients 4 and 10 who had been referred to the Department of Rheumatology, Hackney Hospital. Patients satisfied clinical and epidemiological criteria for diagnosis of gout (Bennet and Wood, 1966). Where possible synovial fluid was examined for crystals using compensated polarizing microscopy (CPM).

Details of the cases are shown in the Table. Our patients appear to fall into three groups: The largest group (cases 1, 2, 3, 4, 5) appeared to develop gouty arthritis complicating longstanding extensive psoriasis. Four of the 5 had typical lower limb acute gout, and all patients had a good response to non-steroidal, anti-inflammatory drugs (NSAID). Four patients had causes other than psoriasis for a raised plasma uric acid, cases 3 and 5 were receiving thiazides as treatment for hypertension and cases 1 and 2 had evidence of alcoholic hepatitis.

The second group (cases 6, 7, 8) had features of a low grade synovitis before the diagnosis of gout. Case
<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Duration (years)</th>
<th>Severity*</th>
<th>Treatment</th>
<th>Psoriasis</th>
<th>Gout</th>
<th>Response to NSAIDS**</th>
<th>Plasma urate (mmol/litre)</th>
<th>Secondary association with hyperuricaemia</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44</td>
<td>5</td>
<td>+++</td>
<td>Dithranol PUVA*</td>
<td>Subtalar</td>
<td>Acute</td>
<td>+++</td>
<td>0.5</td>
<td>+</td>
<td>Biochemistry of alcoholic hepatitis</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>3</td>
<td>+++</td>
<td>Dithranol PUVA*</td>
<td>R 1st MTP L Knee</td>
<td>Sub-acute</td>
<td>+</td>
<td>0.33</td>
<td>+</td>
<td>Liver biopsy showed fatty infiltration compatible with alcoholic effect</td>
</tr>
<tr>
<td>3</td>
<td>54</td>
<td>40</td>
<td>+++</td>
<td>Topical steroids</td>
<td>R 1st MTP</td>
<td>Acute</td>
<td>+++</td>
<td>0.55</td>
<td>+</td>
<td>Hypertensive, taking thiazide</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>35</td>
<td>+</td>
<td>Dithranol Lassar's paste</td>
<td>Ankle</td>
<td>Acute</td>
<td>+++</td>
<td>0.53</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>68</td>
<td>12</td>
<td>+++</td>
<td>L 1st MTP L Knee</td>
<td>L 1st MTP</td>
<td>Acute</td>
<td>+++</td>
<td>0.51</td>
<td>+</td>
<td>Hypertensive uraemia controlled with methyldopa and thiazide</td>
</tr>
<tr>
<td>6</td>
<td>55</td>
<td>26</td>
<td>+++</td>
<td>Hydroxyurea</td>
<td>R 1st MTP Knees</td>
<td>Sub-acute</td>
<td>+</td>
<td>0.57</td>
<td>+</td>
<td>History suggested large joint psoriatic arthritis</td>
</tr>
<tr>
<td>7</td>
<td>35</td>
<td>6</td>
<td>+++</td>
<td>Dithranol</td>
<td>R ankle &amp; 1st MTP</td>
<td>Acute</td>
<td>+</td>
<td>0.52</td>
<td>-</td>
<td>Previous history of plantar fasciitis. Subsequently developed TIP involvement and tenosynovitis of index finger.</td>
</tr>
<tr>
<td>8</td>
<td>37</td>
<td>20</td>
<td>+</td>
<td>None</td>
<td>Knees</td>
<td>Sub-acute</td>
<td>+</td>
<td>0.55</td>
<td>-</td>
<td>Previous history of non-specific urethritis. Subsequently had two episodes of knee swelling with morning stiffness—no crystals seen in synovial fluid.</td>
</tr>
<tr>
<td>9</td>
<td>54</td>
<td>1½</td>
<td>+</td>
<td>None</td>
<td>R 1st MTP</td>
<td>Acute</td>
<td>+++</td>
<td>0.57</td>
<td>-</td>
<td>A publican</td>
</tr>
<tr>
<td>10</td>
<td>58</td>
<td>?</td>
<td>+</td>
<td>None</td>
<td>L wrist</td>
<td>Acute</td>
<td>+++ (colchicine)</td>
<td>0.57</td>
<td>+</td>
<td>Subsequently R 1st MTP involved. High alcohol intake</td>
</tr>
</tbody>
</table>

*Severity in terms of extent: + = few plaques, ++ = intermediate involvement, +++ = most of trunk and limbs.
**+++ = Good response, relief of pain and swelling within 2-3 days. ++ = Moderate response, relief of pain and swelling after 3 days.
PUVA, orally administered psoralen plus longwave ultraviolet light; MTP, mepas tarsophalangeal joint; TIP, terminal interphalangeal joint.
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7 went on to develop typical terminal interphalangeal involvement of psoriatic arthritis with tenosynovitis. Cases 6 and 8 suffered recurrent bouts of knee effusion associated with morning stiffness. The onset of gout was subacute in two of the three and it was in those with knee involvement in which crystals were identified in synovial fluid (cases 2, 6 and 8). A moderate response to NSAID (in terms of time taken for relief of pain and swelling) was a feature of this group.

Cases 9 and 10 appear to represent the coincidence of psoriasis with gout. The skin lesion is difficult to implicate in the pathogenesis of the hyperuricaemia because the skin involvement was trivial and of short duration. Case 10 was associated with a high alcohol intake and case 9 was a publican. High alcohol intake may have played a part in the production of hyperuricaemia in both of these cases. Typical case reports are given.

Case 1

A 54-year-old saleswoman with a 5-year history of extensive psoriasis involving all four limbs and trunk was admitted to hospital because of worsening of his skin condition previously controlled with topical steroids. He had been seen two years before following a severe attack of pain and swelling of the right first metatarsophalangeal joint and had been found to be hyperuricaemic. Following initial treatment with indomethacin 50 mg 3 times daily he was commenced on allopurinol 100 mg 3 times daily but discontinued this after a few months. Alcohol consumption was between 3 to 4 pints of beer per day over 24 years. Examination revealed widespread guttate psoriasis affecting trunk, legs and arms, with scalp plaques; nail pitting was a feature of some toes. He was treated with topical dithranol. One week after admission he developed an acutely tender left subtalar swelling with redness of the skin. This took him off his feet. Treatment with indomethacin 50 mg 3 times daily relieved pain and swelling over 2 days. Serum urate at this time was 0.5 mmol/litre (normal 0.2–0.42 mmol/litre). Slide latex test negative; erythrocyte sedimentation rate 75 mm/hr, neutrophilia. Following discharge the patient has remained asymptomatic but has a persistently elevated serum uric acid.

Case 6

This 55-year-old Ugandan Asian had suffered with psoriasis localized to his scalp for 26 years. Twelve years previously he had had an episode of acute pain and swelling of the left first metatarsophalangeal joint which confined him to bed. This resolved over 3 weeks with bed rest. Since that time he had recurrent swelling of both knees lasting up to 3 months at a time and associated with morning stiffness usually less than one hour. This was controlled with a variety of non-steroidal anti-inflammatory drugs. For the previous 3 years the psoriasis had become more extensive and had been treated with methotrexate and subsequently with hydroxyurea. Because of poor control he was admitted for further treatment with dithranol. Examination showed extensive psoriasis involving trunk, legs and scalp, toe and finger nails showed dystrophic changes with pitting. He had bilateral, warm, knee effusions. Serum urate was 0.52 mmol/litre. X-ray of knees showed no abnormality. Aspiration of the left knee joint synovial fluid revealed numerous urate crystals identified by CPM. The fluid contained 13.3 × 10⁹/litre white cells with 89% polymorphs. Treatment with indomethacin 50 mg three times daily resulted in regression of pain and effusion over one week. The patient had no further joint complaints when seen at 6 months follow-up.

Case 9

A 54-year-old publican was referred to hospital because of an acutely painful right first metatarsophalangeal joint. He had had eight such attacks over the preceding 20 years involving the same joint and usually relieved by bed rest over 2–3 weeks. Over the preceding 18 months he had developed a few psoriatic plaques over his knees and elbows. Alcohol intake was 3–4 pints/day. Examination revealed a swollen tender right first metatarsophalangeal joint with redness and a few extensor surface psoriatic plaques. Blood uric acid was 0.55 mmol/litre. He was treated with phenylbutazone 100 mg 3 times daily and symptoms abated over one day.

Discussion

This study cannot reflect the incidence of gout in the psoriatic population as a whole. The patients were drawn from a highly selected group with unusually extensive disease requiring intensive in-patient treatment. Nor can it prove a causative association of psoriasis with gout—this would require long-term study of a large population of patients with psoriasis. However, some of the cases described suggest that there is a significant association between the two diseases.

Mean uric acid levels appear higher in psoriasis especially when the disease is severe (Baker and Wilkinson, 1979). It is of interest that all the patients were male and that 5 had other conditions known to predispose to hyperuricaemia. Some had several such conditions raising the possibility of a cumulative
effect on raising plasma urate. Thus case 10 had extensive psoriasis being treated with phototherapy and was coincidentally uraemic and receiving a thiazide for hypertension. Most of our patients required energetic drug treatment—a factor which may have contributed to hyperuricaemia.

Our findings of gout in association with psoriasis are at variance with the study of Baker (1966) who failed to find any case of gout in an out-patient study of 650 cases of psoriasis. This is explicable on the basis of the highly selected nature of the patients, in the present study reflecting the severest form of a usually mild disease. Other studies (Bonim and Kimberg, 1962; Kaplan and Klatskin, 1960; Kuzell et al., 1955; Leonard et al., 1978) have reported a significant association of gout with psoriasis. The study from the Mayo Clinic (Leonard et al., 1978) found that the incidence of gout among hospitalized psoriatics was 5% compared with 0·18% in a control group—a highly significant difference.

It is well recognized that psoriatic arthritis may mimic gout (Barber, 1931; Sherman, 1952). In Wright's study (1959) this was particularly so in the subgroup of male patients with distal interphalangeal involvement. The 3 cases in which synovial fluid examination revealed uric acid crystals all had a subacute onset and finding of crystals was unexpected and emphasizes the importance of looking for crystals even in those patients in whom it is not clinically diagnosed. Venkatasubramanian and colleagues (1980) have recently described the association of psoriatic arthritis with true and pseudo-gout. This, together with our own findings, raises the possibility of crystal-induced synovitis being implicated in some cases of 'psoriatic arthritis'. Failure to examine synovial fluid for crystals may have led to underestimation of the association of gout with psoriasis.

Acknowledgments

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References


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