

## Acute gouty bursitis: report of 15 cases

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**SUMMARY** Fifteen cases of acute gouty bursitis were seen among 136 crystal-proved cases of gout. Bursal aspirate yielded yellow or pink fluid in 10, chalky white fluid in 1, and a small amount of bloody fluid in 4. Monosodium urate crystals were present in all. Bursal fluid leucocyte counts averaged  $2.9 \times 10^9/l$  compared with synovial fluid leucocyte counts that averaged  $25.5 \times 10^9/l$  in cases of articular gout ( $P < 0.05$ ). Gouty, septic, and idiopathic (traumatic) bursitis share clinical features, and detailed bursal fluid analysis is crucial for diagnosis.

Bursitis is a known manifestation of gout (Hench, 1941), but little information is available on its clinical and bursal findings. The present report describes 15 such cases seen by the Rheumatology Section at the Boston Veterans Administration Hospital over a recent 4-year period.

### Materials and methods

Only cases of proved gout of subcutaneous bursae were included in the study. All patients had monosodium urate (MSU) crystals in bursal aspirate detected by compensated polarising microscopy, and all fulfilled the preliminary American Rheumatism Association criteria for acute gout (Wallace *et al.*, 1977). All patients were male, reflecting the hospital population. When possible, bursal fluid was analysed according to standard techniques for synovial fluid (Cohen *et al.*, 1975). Chemical determinations were performed by automated methods. The clinical features, characteristics of bursal fluid, treatment, and outcome were reviewed and compared with 121 cases of acute gouty arthritis seen during the same time period. Statistical analysis was made by Student's *t* test.

### Results

All patients had physical evidence of acute inflammation at the site of the affected bursa (Table 1). The underlying joint was normal in 14 and had

evidence of synovitis in 1. Five other patients had concurrent synovitis in a distant joint. Four patients had tophi. Three patients were febrile (all less than  $38.5^\circ\text{C}$ ). Two patients had definite trauma, while in several others the possibility of trauma could not be excluded because of alcoholism. While 13 patients gave the history of 1 or more previous episodes of acute arthritis and 8 were thought to have gout, a definite diagnosis had previously been established in only 3. Serum urate levels were elevated in 6 and normal in 9. Renal function was normal in 8, borderline in 5, and abnormal in 2. Therapy consisted of standard doses of indomethacin or colchicine. Recovery was uneventful in all except 1 patient, who had a recurrence that later responded to an intrabursal corticosteroid injection.

In 4 patients only a trace of bloody bursal fluid could be obtained. Intracellular MSU crystals were found in 1 and extracellular MSU crystals in the other 3. In 1 other case the aspirated fluid was chalky white. In this case, as well as in 9 cases of olecranon bursitis and 1 case of prepatellar bursitis, detailed analysis of the fluid could be performed (Table 2). Bacterial cultures were all negative. The mucin clot was good to poor, leucocyte count averaged  $2.9 \times 10^9/l$  (range  $0.65-6.6 \times 10^9/l$ ), polymorphonuclear leucocytes averaged 34% (range 4-87%), and intracellular MSU crystals were seen in all.

In 121 cases of acute gouty arthritis seen concurrently full synovial fluid analysis could be performed in 77. In these the mean WBC count was  $25.5 \times 10^9/l$  (range  $0.1-152.4 \times 10^9/l$ ) ( $P < 0.05$  when compared with acute gouty bursitis). The synovial fluid polymorphonuclear leucocytes averaged 87% (range 1-100%). When only those patients

Accepted for publication 19 September 1978  
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Table 1 *Acute gouty bursitis: clinical and laboratory characteristics*

Patient	Age	Bursa	Duration of bursitis (days)	Associated synovitis	Previous attacks	Tophi	Serum urate (mg/dl)
1	51	Prepatellar	2	Ankle	—	—	8.1
2	57	Prepatellar	10	—	Episodic arthritis for 4 years	—	9.6
3	53	Prepatellar	2	—	Recurrent podagra	—	7.3
4	62	Prepatellar	2	Ankle	Episodic arthritis for 10 years	—	8.4
5	76	Bunion	8	Ankle*	Podagra 1 year earlier	—	11.4
6	52	Olecranon	28	—	Recurrent podagra for 4 years	—	8.1
7	66	Olecranon	3	Ankle	Episodic arthritis for many years	—	4.4
8	60	Olecranon	3	Elbow	Podagra 7 months earlier*	—	6.4
9	62	Olecranon	8	—	Recurrent knee arthritis for 2 years	—	8.2
10	61	Olecranon	30	—	Inflamed instep 5 years earlier	—	9.3
11	60	Prepatellar	1	—	Episodic arthritis for 1½ years	—	8.0
12	56	Olecranon	70	—	—	+	8.9
13	70	Olecranon	2	—	Episodic arthritis for 12 years treated as gout	+	5.8
14	66	Olecranon	2	Wrist	Arthritis for 1½ years*	+	8.7
15	64	Olecranon	2	—	Polyarthritis for 20 years	+	5.1

\*MSU crystals documented in aspirate. 1 mg/dl urate  $\approx$  0.06 mmol/l. Normal in our laboratory for males  $\leq$  8.2 mg/dl (0.49 mmol/l).

Table 2 *Acute gouty bursitis: bursal fluid findings*

Patient	Volume ml	Colour	Viscosity	Mucin clot	WBC ( $\times 10^9/l$ )	% PMN	RBC ( $\times 10^9/l$ )	MSU crystals
1	Trace	Bloody	ND*	ND	ND	ND	Many	+ †
2	Trace	Bloody	ND	ND	ND	ND	Many	+
3	Trace	Bloody	ND	ND	ND	ND	Many	+
4	Trace	Bloody	ND	ND	ND	ND	Many	+
5	1	White	Viscous	Good	3.15	40	8.1	+ †
6	10	Yellow	Low	Poor	1.0	56	0.48	+ †
7	2	Pink	Low	Poor	4.65	73	ND	+ †
8	3	Yellow	Low	Fair	4.9	87	1.4	+ †
9	1	Yellow	Low	Poor	2.6	8	0.2	+ †
10	5	Pink	Low	Fair	1.25	4	56.8	+ †
11	0.5	Pink	Low	Poor	1.2	15	46.05	+ †
12	2.5	Pink	Low	Poor	0.65	15	30.4	+ †
13	0.3	Pink	ND	Fair	6.6	ND	104.5	+ †
14	10	Pink	Low	Poor	3.8	10	8.1	+ †
15	0.7	Pink with white particles	Low	Fair	2.15	50	35.15	+ †

\*ND=not done. †Intracellular urate crystals.

seen in the first 3 days of the attack were considered, the mean synovial fluid WBC count was  $39.2 \times 10^9/l$ , and the mean bursal fluid WBC count was  $3.9 \times 10^9/l$  ( $P < 0.05$ ).

## Discussion

Subcutaneous bursae develop in areas subject to pressure and frictional forces such as the tip of the elbow and in front of the knee. Tophaceous deposits have been described in bursal walls (Jaffee, 1972). As with rheumatoid nodules, they tend to be palpable in the free wall of the bursae rather than at the bottom, where the nodules of traumatic bursitis usually occur (Canoso, 1977). In addition, owing to the exposed nature of those bursae, effusions with spontaneous resolution in all probability occur in response to minor trauma. Thus, acute gouty subcutaneous bursitis might result from (a) shedding of

MSU crystals into the bursal sac (McCarty, 1977), and (b) transient increase in urate concentration during resorption of a traumatic effusion (Simkin, 1973).

An interesting feature of gouty bursal aspirates was the relatively low WBC and percentage of polymorphonuclear cells as compared with cases of articular gout. Other peculiarities of subcutaneous bursae have been described, such as the poor mucin clot in cases of traumatic bursitis (Canoso, 1977) and the fact that most cases of septic bursitis occur from direct penetration of skin bacteria rather than as a result of bacteraemia (Ho *et al.*, 1978; Canoso and Sheckman, in press). Thus, the structural similarities of bursal and synovial membrane (Key, 1932; Bywaters, 1965; Becker and Rauterberg, 1970; Psaila and Mansel, 1978) may conceal important differences in their biological behaviour so far poorly understood.

While gout was suspected on initial evaluation in most of our patients, exclusion of other common causes of acute bursitis such as trauma or sepsis was not possible on clinical grounds alone. Idiopathic (traumatic) bursitis may present with severe local inflammation resembling gout or infection (Canoso, 1977). Septic bursitis may present as an acute or subacute process usually without fever (Ho *et al.*, 1978; Canoso and Sheckman, in press). In addition both septic and gouty bursitis may be precipitated by local trauma, and concurrent gouty and septic bursitis has been reported (Ho *et al.*, 1978). Thus the importance of bursal aspiration with bursal fluid analysis cannot be overemphasised. As neither septic nor gouty bursal fluid need appear purulent, a low bursal fluid WBC or 'non-inflammatory' appearing fluid should not deter the clinician from performing compensated polarising microscopy, Gram stain, and culture.

The authors are indebted to Dr A. S. Cohen for helpful review of the manuscript.

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*Ann Rheum Dis* 1979 38: 326-328  
doi: 10.1136/ard.38.4.326

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