

# Local Ice Therapy During Bouts of Acute Gouty Arthritis

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**ABSTRACT. Objective.** To evaluate the effect of local application of ice on duration and severity of acute gouty arthritis.

**Methods.** Nineteen patients with acute gout were enrolled and randomized into 2 groups. Group A (n = 10) received topical ice therapy, oral prednisone 30 mg PO tapered to 0 over 6 days and colchicine 0.6 mg/day. Group B was the control group (n = 9), given the same regimen but without the ice therapy. The patients were followed for one week.

**Results.** The mean reduction in pain for those patients treated with ice therapy was 7.75 cm (on 10 cm visual analog scale) with standard deviation  $\pm 2.58$  compared with 4.42 cm ( $\pm$  SD 2.96) for the control group. Using a Wilcoxon rank-sum test there was a significant difference ( $p = 0.021$ ) in pain reduction between the ice therapy and control groups. Joint circumference and synovial fluid volume also tended to be more effectively reduced after one week of therapy in the ice group compared with controls, but these did not achieve statistical significance.

**Conclusion.** The group treated with ice had a significantly greater reduction in pain compared with the control group. Although the clinical improvement was impressive, due to the small sample size we could not show statistically significant improvement in all the variables that tended to suggest that effect was more than simply analgesic. Cold applications may be a useful adjunct to treatment of acute gouty arthritis. (J Rheumatol 2002;29:331–4)

## Key Indexing Terms:

GOUT

COLD

INFLAMMATION

TREATMENT

Cooling can have a marked effect on joints. Cooling of the knee for more than 10 minutes reduces the intraarticular temperature by 2–3°C for as long as several hours<sup>1</sup>. Oosterveld, *et al*<sup>2</sup> found that intraarticular temperatures of the knee dropped from a mean of 89.4° to 72.5°F (31.9 to 22.5°C) within 30 minutes of ice chip application in healthy subjects. Other joints have not been studied in detail,

although it can be assumed that joints that are more superficial than the knee can be cooled with equal or higher efficiency. Rewarming takes a long time because of vasoconstriction; return to baseline temperature can take more than 1 hour<sup>3</sup>.

Cold modalities vary in effectiveness, ease of use, and expense. McMaster, *et al*<sup>4</sup> used adult canine thighs to evaluate the effectiveness of various cooling devices to lower deep muscle temperatures. They found that ice chips in a plastic bag was the most effective method, followed by use of frozen gel packs, endothermic chemical reaction packs, and finally, inflatable plastic envelopes injected with a gas refrigerant. Ice packs and frozen gel packs were significantly more effective than the latter 2 modalities.

Cooling the joint is known to have an anesthetic effect<sup>4</sup>. Cooling decreases hyperemia and has also been reported to decrease experimental crystal induced inflammation<sup>5</sup>. However, Loeb<sup>6</sup> speculated that the predilection of gouty arthritis and gouty tophi for the peripheral parts of the body may at least in part be because these parts are subjected to mean temperatures below 37°C. Cooling of the joints may cause crystal precipitation<sup>6</sup>; therefore one might be concerned that this could precipitate and worsen acute gouty attacks. We even occasionally see clinicians applying warm packs to gouty joints.

Information regarding the effect of ice therapy on gout is sparse. We evaluated the effect of local application of ice on evolution of acute gouty arthritis.

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Submitted March 21, 2001; revision accepted August 7, 2001.

## MATERIALS AND METHODS

**Patients.** We prospectively evaluated the influence of local ice application on severity of acute gout attacks. Nineteen patients with acute gouty attacks seen in the Rheumatology Clinic and during hospitalization at the Philadelphia VA Medical Center between February 1, 1996, to May 1, 1997, were enrolled into an institutional review board approved protocol. All patients had arthrocentesis during the acute gouty attacks and had confirmation of intracellular monosodium urate crystals. Synovial fluid leukocyte counts  $> 2000/\text{mm}^3$  or  $> 10$  leukocytes per high power field (HPF) were seen in 16 patients. Three patients with synovial fluid leukocyte counts  $< 2000/\text{mm}^3$  but with a clinical picture of acute gout were also included in the study (of these patients 2 were in the group treated with ice and one was in the control group). Patients were randomly assigned to one of 2 groups (by blindly drawing a folded paper note). Group A ( $n = 10$ ) received topical ice therapy, oral corticosteroids (prednisone tapered from 30 mg to 0 over 6 days (30 mg  $\times$  2 days, 20 mg  $\times$  2 days, 10 mg  $\times$  2 days) and colchicine 0.6 mg/day. Group B was the control group ( $n = 9$ ), given the same regimen but without the ice therapy. Allopurinol treatment was continued in the same dose if patients were receiving it prior to the attack. The targeted joints were knees ( $n = 9$ ), ankles ( $n = 3$ ), first metatarsophalangeal (MTP) ( $n = 5$ ), and metacarpophalangeal (MCP) ( $n = 2$ ). Four out of 10 patients in the ice group and 4/10 in the control group had oligoarticular gouty attacks. Other involved areas were the foot ( $n = 3$ ), MCP ( $n = 1$ ), ankle ( $n = 3$ ), olecranon bursa ( $n = 1$ ), MTP ( $n = 2$ ), and wrist ( $n = 1$ ).

**Treatment regimen.** Ice therapy, by application of ice packs with self-ties (Stay-dry ice packs, Tecnol model 11427) on the inflamed target joint for 30 min 4 times/day, was given to all patients in Group A. The patients were followed for one week.

At entry, the patients recorded a pain score (marked by the patient on a 10 cm visual analog scale, VAS) and the circumference of the affected joint was measured with a tape measure expressed in centimeters. The measurements were performed as follows: knee 5 cm below the superior patellar border, ankle diameter surrounding the medial and lateral malleolus, and first MTP or any of the 2nd–5th MCP measured by circumference around all MTP and MCP. These measures were repeated after one week. Serum uric acid, erythrocyte sedimentation rate (ESR), and synovial fluid analysis when possible were performed at baseline and after treatment with ice.

**Statistical analysis.** A nonparametric statistical test was necessary since normality could not be assumed due to a limited sample size. The Wilcoxon rank sum test<sup>7</sup> was used to compare the pain reduction between the ice therapy group and controls. This nonparametric method allows analyses of non-normal data. This procedure was repeated for comparisons in the reduction of joint circumference, reduction of synovial cell count, and reduction of synovial fluid volume between both groups. All analyses were performed using SAS version 6.12 with a 5% level of significance ( $p < 0.05$ ).

## RESULTS

Comparison of the pain score on VAS (cm) between the group treated with ice (Group A) and controls (Group B) is shown in Table 1. The mean reduction in pain for patients treated with ice therapy was 7.75 cm ( $\pm$  SD 2.58) compared to 4.42 cm ( $\pm$  SD 2.96) for controls. Using a Wilcoxon rank-sum test there was a significant difference ( $p = 0.021$ ) in pain reduction between the ice therapy and control groups. While the mean pain score was similar in both groups of patients at entry, the mean pain score was significantly lower in patients receiving one week of ice treatment compared with controls.

For joint circumference, the mean reduction for the ice group was 5.90 cm ( $\pm$  SD 3.84) compared with 3.83 cm ( $\pm$

SD 4.19) for controls. While the joint circumference tended to be more reduced in the ice therapy group than in controls, the Wilcoxon rank-sum test did not show significant difference between the 2 groups when ice therapy was used ( $p = 0.140$ ) (Table 2).

No significant difference ( $p = 0.749$ ) was found comparing the mean reduction in synovial cell count, 9341.67 ( $\pm$  SD 11,552.90) for the ice group compared with 9412.50 ( $\pm$  SD 9870.27) for controls (Table 3). Table 3 shows synovial fluid cell count only when there was an adequate volume to do a synovial fluid leukocyte count. Crystals continued to be seen in all fluids obtained. There was no suggestion that numbers were increased.

No significant difference ( $p = 0.454$ ) was found comparing mean synovial fluid volumes between the 2 groups, although synovial fluid volume tended to decrease after ice treatment. In cold treated joints volume was 37.33 ml ( $\pm$  SD 26.17) for the ice treatment compared with 21.25 ml ( $\pm$  SD 23.94) for controls (data not shown). However, 3 patients in the ice group had complete resolution of their effusion in contrast to none in the control group.

There were no significant differences between these 2 groups in their ESR (mean ESR on entry in the ice treated group 47 mm/h and controls 65 mm/h) and uric acid (UA) levels (mean UA level on entry in the ice treated group 9.7 mg/dl, controls 9.1 mg/dl). There were no complaints concerning the ice treatment and the subjective responses of the patients were favorable.

## DISCUSSION

Information regarding the effect of ice therapy on gout is sparse. One of the investigators (HRS) with Dorwart, *et al*<sup>5</sup> applied local ice to dog knees injected with monosodium urate (MSU) crystals. This study showed that joint fluid volume and synovial fluid leukocyte counts were lower in the cooled knees compared with the non-cooled contralateral knees in all dogs. Dorwart, *et al* also showed that synovial fluid leukocytes that were exposed to cold had less phagocytosis of MSU crystals. Crystal phagocytosis was diminished on the cooled side in all but one case, with mean number of cells containing crystals 61% in control knees and 31% in cooled knees.

Hunter, *et al*<sup>8</sup> showed that cold increased the viscosity of synovial fluid in normal cat joints. Increased viscosity might, in turn, impede the movement of white cells toward crystals, resulting in less phagocytosis and therefore possibly less inflammation. Light microscopic examination of cooled MSU injected dog knees (after 4 h of cooling) revealed that the injected dog knees had less vasodilatation, less lining cell hyperplasia, less interstitial escape of intravenously injected carbon, and less white cell infiltration, compared with control knees<sup>5</sup>. This provided further evidence that the joints were less inflamed when they were cooled. Weinberger, *et al*<sup>9</sup> studied the effect of ice therapy

Table 1. Comparison of pain score between patients treated with ice and controls.

Patient	Ice		Joint	Patient	Control		Joint
	On Entry	After 1 Week			On Entry	After 1 Week	
1	2.5	0	knee	1	10	8	MTP
2	4.5	0	ankle	2	9	5	knee
3	10	1	MTP	3	10	1	knee
4	10	3.5	ankle	4	7	0	MTP
5	10	0	knee	5	10	9	MTP
6	10	0	knee	6	9.5	1.5	ankle
7	10	1	MTP	7	7.5	6.2	knee
8	10	0	MCP	8	9.5	6.5	knee
9	9	2	knee	9	10	5.5	MCP
10	49.5	0.5	knee				
Mean	8.55	0.8			9.6	4.74	

Table 2. Comparison of joint circumferences (cm) between patients treated with ice and controls.

Patient	Ice		Joint	Patient	Control		Joint
	On Entry	After 1 Week			On Entry	After 1 Week	
1	42	35	knee	1	27	25	MTP
2	30	26	ankle	2	44	36	knee
3	34	26	MTP	3	45	43	knee
4	34	27	ankle	4	25	24.5	MTP
5	42	36	knee	5	24	23.5	MTP
6	59	44	knee	6	30	28	ankle
7	24	23	MTP	7	50	45	knee
8	24	21	MCP	8	65	52	knee
9	49	45	knee	9	25.5	24	MCP
10	46	42	knee				
Mean	38.4	32.5			37.3	33.4	

Table 3. Comparison of synovial cell count (WBC/mm<sup>3</sup>) between patients treated with ice and controls.

Patient	Ice		Joint	Patient	Control		Joint
	On Entry	After 1 Week			On Entry	After 1 Week	
1	750	No fluid	knee	2	68500	45500	knee
2	850	No fluid	ankle	3	1500	150	knee
5	4850	650	knee	7	11750	1400	knee
6	21000	350	knee	8	5800	2850	knee
9	28450	1250	knee				
10	2400	No fluid	knee				
Mean	9716.7	750			21887.5	12475	

on synovitis induced by intraarticular injection of zymosan to hind joints in rabbits. They found less cellular infiltration and less synovial cell hyperplasia in the joints treated with ice (30 min/day for 10 days). Thus, synovitis and inflammation were suppressed by lowering intraarticular temperature in animal models.

We studied the effect of cold therapy on acutely inflamed gouty joints in humans. Compared with the control group,

the group treated with ice had a greater reduction in pain and the difference was statistically significant compared to controls. Some controls did surprisingly poorly, possibly because of the rapid tapering of the steroids, which was done in both groups. A short course of systemic corticosteroids is useful for acute gout, especially if there are contraindications to nonsteroidal antiinflammatory drugs (NSAID)<sup>10</sup>. The protocol specified corticosteroids for

uniformity, as some patients could not have taken NSAID. Some of our patients did have renal involvement as well as oligoarticular gout, necessitating use of corticosteroids.

Patients treated with ice also tended to have greater reduction in joint circumference, although this did not achieve statistical significance. This may simply be related to small sample size. Alternatively, it may reflect the difficulty in performing and averaging circumferences from different sites or might suggest less effect on the inflammatory changes in the periarticular tissues. Better resolution of the synovial fluid volume when measurable was not found to be statistically significant, although 3 patients in the ice group had complete resolution of their effusion in contrast to none in the control group, and again the trend was generally in favor of greater improvement with ice treatment. No significant difference was found comparing the mean reduction in the synovial cell count.

Our clinical impression was that the group treated with ice had much less severe courses of their attacks than did the control group. The patients, most of whom had previous gouty attacks, described lessened severity of attacks with ice treatment compared with previous attacks. Complete resolution at one week was seen only in those treated with ice, although virtually all were improved. Although the clinical improvement was impressive, due to the small sample size we could not show statistically significant improvement in all variables.

Our study suggests that cold applications may be a useful adjunct to treatment of acute gouty arthritis. All our patients

were aggressively treated with oral corticosteroids. Whether the same result would occur with other drug regimens has not been tested.

## REFERENCES

1. Hollander JL. Collagenase, cartilage and cortisol [editorial]. *N Engl J Med* 1974;290:50-1.
2. Oosterveld FG, Rasker JJ, Jacobs JW, Overmars HJ. The effect of local heat and cold therapy on the intraarticular and skin surface temperature of the knee. *Arthritis Rheum* 1992;35:146-51.
3. Kaul MP, Herring SA. Superficial heat and cold. How to maximize the benefits. *Phys Sports Med* 1994;22:65-74.
4. McMaster WC, Liddle S, Waugh TR. Laboratory evaluation of various cold therapy modalities. *Am J Sports Med* 1978;6:291-4.
5. Dorwart BB, Hansell JR, Schumacher HR Jr. Effects of cold and heat on urate crystal-induced synovitis in the dog. *Arthritis Rheum* 1974;17:563-71.
6. Loeb JN. The influence of temperature on the solubility of monosodium urate. *Arthritis Rheum* 1972;15:189-92.
7. Hollander M, Wolfe DA. Nonparametric statistical methods. New York: John Wiley and Sons; 1973:67-113.
8. Hunter J, Kerr EH, Williams MG. The relation between joint stiffness upon exposure to cold and the characteristics of synovial fluid. *Can J Med Sci* 1952;30:367-77.
9. Weinberger A, Giler S, Nyska A. Treatment of inflammatory synovitis with ice application [abstract]. *Arthritis Rheum* 1995;38 Suppl:S242.
10. Groff GD, Franck WA, Raddatz DA. Systemic steroid therapy for acute gout: A clinical trial and review of the literature. *Semin Arthritis Rheum* 1990;19:329-36.