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Therapeutic effects of colchicine in the management of Peyronie's disease: a randomized double-blind, placebo-controlled study

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To determine effectiveness and safety of colchicine in Peyronie's disease. In all, 84 patients with Peyronie's disease who did not have calcified plaque were entered into study. The mean disease duration was 15 months. A medical history was obtained, and physical examination, penile X-ray, and dynamic penile duplex ultrasound were performed. Patients were randomly divided into group 1, those who received 0.5–2.5 mg colchicine daily for 4 months and group 2, who received placebo for the same period. Response to therapy was assessed objectively, during dynamic penile duplex ultrasound, as well as subjectively using International Index of Erectile Function (IIEF) questionnaire and measurements of pain, duration of disease, penile curvature, and plaque size. Differences before and after treatment and among the three Kelami classification groups were assessed. In total, 78 (92.8%) completed the whole treatment schedule. Pain resolved in 60 and 63.6% of the patients treated with colchicine and placebo, respectively ($P>0.05$). After therapy, in subjects and controls a reduction in the penile deformity was observed by 17.1 and 18.4% of the patients ($P>0.05$), and a decrease in plaque size was noticed by 10.5 and 10%, respectively ($P>0.05$). Objective measurements did not demonstrate any difference in plaque size or penile curvature. There were no substantial differences in response to treatment based on duration of disease or within the three Kelami classification groups. Significant drug-related adverse effects occurred in colchicine group and in two cases was treatment discontinued. Colchicine is no better than placebo in improvement of pain, curvature angle, or plaque size in patients with Peyronie's disease.
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Introduction

Peyronie's disease leads to a significant decrease in the quality of life of affected individuals. It was first reported by François Gigot de la Peyronie, Surgeon to king Louis XV of France, and is characterized by the formation of fibrous inelastic scar involving the tunica albuginea of the corpora cavernosa. It has been proposed that fibrosis and plaque formation in the tunica albuginea are the end results of inflammatory changes, which occur most commonly following trauma to the erect penis.[1] Plaque causes restricted expansion of the tunica albuginea during erection, which in turn causes curvature and may result in the inability to perform sexual intercourse.

Despite numerous treatment options, a standard nonsurgical therapy for the treatment of Peyronie's disease has yet to be identified. Oral agents include potassium aminobenzoate,[2] vitamin E,[3] tamoxifen,[4] procarbazine,[5] and colchicine,[6] among others. Intralesional therapy for Peyronie's disease includes steroids,[7] collagenase,[8] verapamil,[1] parathyroid hormone,[9] orgotein,[10] and interferon.[11] Also various modes of energy transfer, including orthovoltage radiation, ultrasound, short-wave diathermy, laser therapy, and shock wave lithotripsy have been used to treat Peyronie's disease.[12–14] However, the lack of placebo-controlled groups, limited patient numbers, inadequate follow-up time and inconclusive results, have compromised the interest of physicians in using these treatment modalities.

Previous studies have shown that Peyronie's disease is associated with upregulation of transforming growth factor (TGF)-β_1 expression,[15] a protein

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known to be involved in the inflammatory response, wound healing and in chronic fibrotic conditions. Colchicine is an antimicrotubule agent that inhibits collagen secretion from fibroblasts.[16] The effect of colchicine on the production of fibrosis and the lack of large scale clinical, randomized, and placebo-controlled study were the reasons that let us to perform this clinical trial.

Materials and methods

A total of 84 men, 35–60 y old (mean age 46 y) with Peyronie's disease 6–42 months (mean 15 months) in duration participated in this study. Of these patients, 62 (73.8%) had received one or more previous treatments for Peyronie's disease, including oral therapy with potassium aminobenzoate (82.2%), vitamin E (9.7%), and tamoxifen (8.1%). All patients were required to have ceased all medical therapy at least 8 weeks before the initiation of the study. The previous treatments had no effect when we started the study. At presentation, 48 patients (57%) had vascular risk factors, including smoking, history of elevated serum cholesterol or lipids, and diabetes, and 39 patients (46.4%) recalled penile trauma. Pain during erection was a presenting complaint of 48 (57%) patients. Totally, 34 patients (40.5%) with disease duration of larger than 1 y were considered to be in the late disease stage. The patients were previously informed about the research details and possible severe side effects, and agreed to cooperate. At baseline, the study and placebo groups were similar in demographic characteristics and risk factors.

Pretreatment assessment

A detailed medical history was obtained from each patient and all patients completed the International Index of Erectile Function (IIEF) questionnaire.[17] As part of the objective evaluation, all patients underwent dynamic penile duplex ultrasound before and after intracavernosal injection of 20-μg prostaglandin E_1. The degree of penile curvature was measured during a full erection using a protractor. For subjective judgment of penile deviation, patients were asked to sketch the curved erect penis. Plaque volume was estimated by multiplying the ultrasound determined length, width, and depth. Presence of calcification was evaluated with penile X-ray and during ultrasound. In all, 68 (81%) described significant pretreatment psychogenic concerns because of deformity and erectile compromise.

Treatment

The patients were randomly assigned to either of two groups of 42 subjects each. Group 1 was given 1 mg colchicine orally daily during week 1 of treatment followed by an increase to 2.5 mg daily for 4 months. Group 2 received a similar regimen of placebo. Treatment was administered in a randomized sequence that remained unknown to the patient and to the physician.

Post-treatment assessment

The effect of treatment was assessed at the end of the study and 1 month after cessation of treatment. The same interviewer evaluated subjective changes in pain, curvature, deformities, and erectile status. Objective changes were recorded with duplex ultrasound by the same ultrasound radiologist in patients completing the treatment protocol. The mean follow-up time was 16 months (range 8–22 months). In an effort to evaluate outcome based on varying degree of initial curvature and plaque length, a modified form of the Kelami classification system[18] was used. Patients were divided into three groups: class 1—plaque length 2 or less cm and curvature of 30° or less, class 2—plaque between 2 and 4 cm and 30–60° curvature and class 3— plaque greater than 4 cm and curvature greater than 60°. Patients who did not fit into any of the proposed classes (eg patients with plaque length of less than 4 cm and curvature greater than 60°) were considered in higher class.

The χ^2 test for qualitative and Student's *t*-test for quantitative data were used with the Yates correction factor for statistical analysis of the qualitative results of the differences in this study with $P<0.05$ considered significant. Statistical analysis was performed using the computer statistical package SPSS/4.0 (SPSS, Chicago, IL, USA) and SAS/6.4 (SAS Institute Cary, NC, USA).

Results

In total, 84 patients were recruited for the study, but 78 (92.8%) completed the whole treatment schedule. The prevalence and the nature of risk factors in colchicines- and placebo-treated groups were not statistically different. There were no statistical differences in plaque size, penile curvature, and disease onset in the two groups (Table 1). The mean disease duration was 15 months and was not significantly different between two groups. Before initiating therapy, subjective measurements of curvature in 79 (94%) patients were determined, which

was ventral (mean 26°, range 14–32°) by 2 (2.5%), dorsal (mean 45°, range 10–60°) by 42 (53.2%), and lateral (mean 26°, range 10–60°) by 35 (44.3%). More than one direction of curvature was described by 11 (14%) of men. Objective measurements of curvature following PGE_1 injection demonstrated ventral curve (mean 21°, range 10–30°) in two (2.5%) men, dorsal curve (mean 35°, range 10–60°) in 58 (73.4%), and lateral curve (mean 15°, range 10–60°) in 53 (67%). In addition, 34 (43%) patients had curvature in more than one direction. Post-treatment subjective curvature data were available for 78 of the 84 men who completed the study (Table 2). In 38 patients who received colchicines, six (17.1%) noticed a decrease in curvature (mean 32°, range 10–60°), 24 (63.2%) reported an increase (mean 25°, range 10–40°), and eight (21%) found no change. When treated with placebo ($n=40$), seven (18.4%) reported a decrease in curvature (mean 29°, range 10–60°), 22 (55%) noticed an increase (mean 27°, range 10–40°) and 11 (27.5%) had no change. The statistical analysis with the χ^2 test ($P>0.05$) and the Yates correction factor showed independence between colchicine and subjective changes in curvature, providing that colchicine was not statistically superior to placebo. Of the patients who completed therapy, 72 were objectively evaluated with duplex ultrasound (Table 3). Of the 35 patients who received colchicines, five (14.3%) had a decrease in curvature (mean 30°, range 10–60°), 22 (62.8%) had an increase (mean 30°, range 10–45°) and eight (22.9%) remained unchanged. Of patients treated with placebo ($n=37$), five (13.5%) had a decrease in curvature (mean 28°, range 10–55°), 23 (62.2%) had an increase (mean 33°, range 10–45°) and nine (24.3%) had no change. These parameters, showed no statistical differences between two groups.

All patients had a palpable plaque before treatment. Pretreatment mean volume was 5.2 ml (range 0.9–7.6 ml) and 5.4 ml (range 0.8–7.4 ml) in subjects and placebo groups, respectively. Plaque size decreased in 10.5 and 10% of patients in groups 1 and 2, respectively. After treatment, the mean plaque volume was 5.9 ml in men who received colchicine and 6.1 ml in those who received placebo ($P>0.05$). Although we selected patients without any calcified plaque, calcification appeared in eight (21%) of the

Table 1 Demographic characteristics of patients

Variables	*Colchicine* n = *42*	*Placebo* n = *42*
Mean age, y (range)	45 (36–61)	47 (34–59)
Mean disease duration Months (range)	14 (6–40)	16 (8–43)
Pain during erection, no. (%)	25 (59.5%)	23 (54.8%)
Mean plaque volume, ml (range)	5.2 (0.9–7.6)	5.4 (0.8–7.4)
Objective penile curvature, No. (mean degree)		
Ventral	1 (28)	1 (24)
Dorsal	27 (42)	31(47)
Lateral	27 (13)	26 (17)

Table 2 Subjective effects of colchicine and placebo

Variables	*Patients (no.)*	*No. patients (%)*	
		Pretreatment status	*Post-treatment improvement*
Pain during erection			
Colchicine	38	20 (52.6)	12 (60)*
Placebo	40	22 (55)	14 (63.6)
Penile curvature			
Colchicine	38	35 (92)	6 (17.1)*
Placebo	40	38 (95)	7 (18.4)
Capable of intercourse			
Colchicine	38	25 (65.8)	24 (96)*
Placebo	40	27 (67.5)	25 (92.6)
Patient's perception of plaque volume			
Colchicine	38	38 (100)	4 (10.5)*
Placebo	40	40 (100)	4 (10)

*$P>0.05$ *vs* placebo.

Table 3 Objective effects of colchicine and placebo

Variables	*Patients (no.)*	*No. (%)*	*Median ± s.d.*	
			Before treatment	*After treatment*
Plaque volume (ml)				
Colchicine	37		4.75 ± 3.22*	4.86 ± 4.65
Placebo	35		4.65 ± 3.56	4.76 ± 4.39
Curvature angle (deg)				
Colchicine	37		36.16 ± 20.02*	34.65 ± 19.37
Placebo	35		36.71 ± 19.40	40.07 ± 18.19
Appearance of calcification				
Colchicine	37	8 (21)*		
Placebo	35	7 (17.5)		

SD, standard deviation.
*$P>0.05$ *vs* placebo.

patients who received colchicine and in seven (17.5%) who received placebo in follow-up period. This may indicate disease progression. Penile pain subsided in 60% of the patients who received colchicine and in 63.6% who received placebo ($P>0.05$).

Before treatment patients were asked whether they were capable or incapable of intercourse. Of the men who received colchicine ($n=38$), 25 (65.8%) thought they were capable, while 13 (34.2%) were incapable of successful intercourse due to lack of rigidity. These were 67.5 and 32.5% in placebo group, respectively. After treatment, 24 (63.2%) of the 38 patients who received colchicine had successful intercourse, while 14 (36.8%) were impotent. These were 62.5 and 37.5%, respectively, in men who received placebo. Statistical analysis disclosed no significant difference when colchicine was compared to placebo ($P>0.05$).

Patients were also studied based on duration of disease before initiation of therapy. A disease duration of 1 y or greater did not influence the treatment results in either group ($P>0.05$).

The severity of the disease did not affect the outcome. There were no significant differences across the three Kelami classes (Table 4). As mentioned above six patients did not complete the study. Significant drug-related adverse effects occurred in colchicine group and in two cases (4.8%) was treatment discontinued due to gastrointestinal upset with diarrhea. Four patients requested no further treatment due to any beneficial effect.

Discussion

Despite many advances in our understanding of pathophysiology of patients with Peyronie's disease, the true pathogenesis remains unknown. Although a number of medical therapies are available for Peyronie's disease, the lack of significant prospective and double-blind, placebo-controlled studies make it difficult to individualize efficacy rates.

It is generally agreed that trauma is the most likely inciting factor, but other contributors include autoimmune etiology,[19] upregulation of transforming growth factor (TGF)-β_1 expression[15] and cytogenic aberrations[20] also were proposed. TGF-β has gained considerable attention as a factor implicated in the cause of chronic fibrotic changes. The ability of TGF-β to induce its own production is considered the key to the development of excessive scarring and fibrosis.[21] Colchicine binds to tubulin, blocks mitosis and inhibits the function of polymorphonuclear leukocytes both *in vitro* and *in vivo*.[22] Colchicine can reversibly cause the disassembly of microtubules during interphase of the cell.[23] By disrupting microtubule organization, colchicine can interfere with any cell function dependent on these form-determining elements.[24] El-Sakka *et al*[25] assessed the effect of colchicine in animal model of Peyronie's disease. The rats treated with colchicine showed less collagen deposition and less elastic fiber fragmentation. The results of this study clearly show that administration of colchicine can significantly downregulate of TGF-β expression in the

Table 4 Kelami classification of post-treatment data

	Class I	*Class II*	*Class III*
Objective			
Colchicine treated (*patients no.*)	20 (57.1)	10 (28.6)	5 (14.3)
Increased curvature no. (%)	13 (65)*	6 (60)	3 (60)
Decreased curvature no. (%)	3 (15)*	1 (10)	1 (20)
No change in curvature no. (%)	4 (20)*	3 (30)	1 (20)
Placebo treated (*patients no.*)	20 (54)	11 (29.8)	6 (16.2)
Increased curvature no. (%)	12 (60)*	7 (63.7)	4 (66.7)
Decreased curvature no. (%)	3 (15)*	2 (18.2)	0 (0)
No change in curvature no. (%)	5 (25)*	2 (18.2)	2 (33.4)
Subjective			
Colchicine treated (*patients no.*)	22 (57.9)	10 (26.3)	6 (15.8)
Improved curvature no. (%)	3 (13.7)*	2 (20)	1 (16.7)
Increased curvature no. (%)	14 (63.7)*	6 (60)	4 (66.7)
No change in curvature no. (%)	5 (22.7)*	2 (20)	1 (16.7)
Placebo treated (*patients no.*)	23 (57.5)	11 (27.5)	6 (15)
Improved curvature no. (%)	4 (17.4)*	2 (18.2)	1 (16.7)
Increased curvature no. (%)	12 (65.2)*	6 (54.6)	4 (66.7)
No change in curvature no. (%)	7 (30.5)*	3 (27.3)	1 (16.7)
Improved sexual function			
Colchicine treated (patients no.)	0 (0)*	0 (0)	0 (0)
Placebo treated (patients no.)	0 (0)*	0 (0)	0 (0)

*$P>0.05$ *vs* Class I and Class II.

colchicine-treated group. Also in an uncontrolled pilot study colchicine was shown to decrease plaque size and improved penile curvature in approximately 50% of the 24 patients treated.[6] Kadioglu *et al*[26] conducted a nonrandomized trial of 60 men receiving up to 2-mg/day oral colchicine, with a mean follow-up of 10.7 months. In this group, pain improved in 95% of cases, and deformity in 30% of men. However, the beneficial effects of colchicine were not produced in our study. None of our analyses showed a significant subjective or objective response. Questions regarding the true efficacy of the reported studies without a proper control arm are valid. Poorly characterized protocols, limited follow-up, few patients numbers, no placebo or control groups and little objective measurements of change compromise most of these studies. The ideal candidates for medical treatment may include those who present with pain, have curvature of less than 60°, and a plaque volume of less than 5 cm without calcification.[1] Most of our patients had these criteriae.

In our study, the colchicine therapy resulted a reduction in pain in 60%, decreased curvature in 17.1% without improvement in sexual function. In placebo-treated patients, pain reduced in 63.6% and curvature decreased in 18.4%. Although the responses obtained with colchicine were lower than obtained from placebo, but differences were not statistically significant. Prevalence and severity of risk factors were similar between two groups. This means that colchicine has acted like placebo.

The results of the current study clearly show that, administration of colchicine cannot eliminate the structural abnormality of the tunica and cannot suppress the prolonged expression of TGF-β. We postulate that production of TGF-β continues over the course of the disease, and it results in disease progression. Therefore, inhibition of the production of TGF-β must delay disease progression.

Treatment of Peyronie's disease will move towards halting and reversing the fibrosis and scarring responsible for this disease. In this study colchicine could not do this.

It has been suggested previously that response to therapy may be more suitable with early *vs* late disease stages.[25,3] The mean duration of disease before the start of this study was 15 months. In total, 50 patients with disease duration of less than 1 y were considered to be in the early stage, and 34 patients with disease duration of 1 y or longer were considered to be in the late disease stage. There did not seem to be a significant greater likelihood of success with early *vs* late disease stages. In this study, of the 84 patients, 82% were classified as Kelami class 1 or 2 suggesting a less severe disease state in this patient population. The severity of the disease did not affect the outcome since response to therapy did not differ among the three Kelami groups.

The lack of significant differences between early and late stage patients, as well as among the three groups with varying severity of disease, indicates a presumed persistence of immature fibroblasts that cannot respond to this treatment.

The acute inflammatory phase usually lasts between 6 and 18 months, and is characterized by pain, penile curvature, and nodule formation, often with a self-limiting course.[27] Medical treatment in this acute phase is recommended. In our study, the length of disease duration or severity of disease had no significant impact on the response to colchicine. This is similar to the reports in some other studies.[1,28] Also Husain *et al*[29]reported that, with extracorporeal shock wave therapy, there was a greater reduction in those patients who had disease duration of larger than 12 months.

It has been reported that there is a spontaneous remission over the natural history of Peyronie's disease, which may then be responsible for the improvement seen following drug therapy. The pain associated with erection almost uniformly resolves with time but the deformity does not. In a study of a small population (12 patients) Williams and Thomas[30] reported the highest spontaneous remission rate of 29% with respect to curvature. In the recent report on spontaneous remission by Kadioglu[31] who followed 63 men for a mean of 14 months spontaneous improvement rate was 7%, while 60% had no changes and 33% had worsening deformity. Also psychological effects due to Peyronie's disease were reported by 77% of the patients with Peyronie's disease.[3]

There was significant drug-related adverse effect in this study. The reason is the moderately higher doses of colchicine that has been used.

Clearly, the issue of placebo-controlled trials will remain the ultimate litmus test of treatment efficacy. However, we present our study as the largest group of men treated and evaluated following colchicine therapy with subjective and objective measures of deforming change.

These were not significantly different from the results obtained with placebo.

We agree that further studies must be done on this drug to define the role of colchicine in Peyronie's disease.

Conclusions

We concluded that 4 months of therapy with up to 2.5 mg colchicine daily in patients with Peyronie's disease, has no beneficial effect. Ethically designed placebo-controlled trials will add to our knowledge to development of effective therapy for Peyronie's disease.

References

1 Levine LA. Treatment of Peyronie's disease with intralesional verapamil injection. *J Urol* 1997; **158**: 1395–1401.
2 Carson CC. Potassium para-aminobenzoate for the treatment of Peyronie's disease: is it effective? *Tech Urol* 1997; **3**: 135–140.
3 Gelbard MK, Dorey F, James K. The natural history of Peyronie's disease. *J Urol* 1990; **144**: 1376–1381.
4 Ralph DJ, Brooks MD, Bottazzo GF, Pyror JP. The treatment of Peyronie's disease with tamoxifen. *Br J Urol* 1992; **70**: 648–653.
5 Morgan RJ, Pryor JP. Procarbazine (Natulan) in the treatment of Peyronie's disease. *Br J Urol* 1978; **50**: 111–116.
6 Akkus E *et al.* Is colchicine effective in Peyronie's disease? A pilot study. *Urology* 1994; **44**: 291–296.
7 Winter CC, Khanna R. Peyronie's disease: results with dermojet injection of dexamethasone. *J Urol* 1975; **114**: 898–903.
8 Gelbard MK, James K, Riach P, Dorey F. Collagenase versus placebo in the treatment of Peyronie's disease: a double-blind study. *J Urol* 1993; **149**: 56–60.
9 Morales A, Bruce AW. The treatment of Peyronie's disease with parathyroid hormone. *J Urol* 1975; **114**: 901–906.
10 Bartsch G, Menander-Huber KB, Marberger H. Orgotein, a new drug for the treatment of Peyronie's disease. *Eur J Rheumatol Inflamm* 1981; **4**: 250–255.
11 Duncan MR, Berman B, Nseyo UO. Regulation of the proliferation and biosynthetic activities of cultured human Peyronie's disease fibroblast by interferons-alpha, -beta and -gamma. *Scand J Urol Nephrol* 1991; **25**: 89–94.
12 Incrocci L, Hop W, Slob A. Current sexual functioning in 106 patients with Peyronie's disease treated with radiotherapy 9 years earlier. *Urology* 2000; **56**: 1030–1033.
13 Hauck E *et al.* Extracorporeal shock wave therapy in the treatment of Peyronie's disease. *Eur Urol* 2000; **38**: 663–670.
14 Frank IN, Scott WW. The ultrasonic treatment of Peyronie's disease. *J Urol* 1971; **106**: 83–88.
15 El-Sakka AI *et al.* The effect of surgical trauma on rat tunica albuginea. *J Urol* 1998; **159**: 1700–1706.
16 Diegelmann RF, Peterkofsky B. Inhibition of collagen secretion from bone and cultured fibroblast by microtubular disruptive drugs. *Proc Natl Acad Sci USA* 1972; **69**: 892–898.
17 Rosen RC *et al.* The International Index of Erectile Function (IIEF): a multidimensional scale for assessment of Erectile dysfunction. *Urology* 1997; **49**: 620–625.
18 Kelami A. Classification of congenital and acquired penile deviation. *Urol Int* 1983; **38**: 822–826.
19 Steward S, Malto M, Sandberg L, Colburn KK. Increase serum level of anti-elastin antibodies in patients with Peyronie's disease. *J Urol* 1994; **152**: 105–111.
20 Somers KD *et al.* Isolation and characterization of collagen in Peyronie's disease. *J Urol* 1989; **141**: 629–634.
21 Border WA, Ruoslahti E. Transforming growth factor beta in disease: the dark side of tissue repair. *J Clin Invest* 1992; **90**: 1–7.
22 Taylor AC. The mechanism of colchicine inhibition of mitosis. I. Kinetics of inhibition and the binding of H3-colchicine. *J Cell Biol* 1965; **25**: 145–150.
23 Tilney LG, Hiramoto Y, Marsland D. Studies on the microtubules in heliozoa. *J Cell Biol* 1966; **29**: 77–82.
24 Ehrilich HP, Bornstein P. Microtubules in transcellular movement of procollagen. *Nature (New Biol)* 1972; **238**: 257–261.
25 El-Sakka AI *et al.* The effects of colchicine on a Peyronie's-like condition in an animal model. *J Urol* 1999; **161**: 1980–1983.
26 Kadioglu A *et al.* Treatment of Peyronie's disease with oral colchicine: long term results and predictive parameters of successful outcome. *Int J Impot Res* 2000; **12**: 169–175.
27 Claudio T *et al.* Tamoxifen versus placebo in the treatment of Peyronie's disease. *J Urol* 1999; **162**: 2003–2008.
28 Levine LA, Goldman KE, Greenfield JM. Experience with intraplaque injection of verapamil for Peyronie's disease. *J Urol* 2002; **168**: 621–626.
29 Husain J *et al.* Extracorporeal shockwave therapy in the management of Peyronie's disease. *Br J Urol* 2000; **68**: 466–468.
30 Williams J, Thomas GG. The natural history of Peyronie's disease. *J Urol* 1970; **73**: 75–79.
31 Kadioglu A. Lessons learned from 307 men with Peyronie's disease. *J Urol Suppl* 2001; **165**: 202, abstract 838.