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REVIEW

Contemporary Management Strategies for Peyronie's Disease: A Comprehensive Review

Yousif M Basheer[1], Fatma Ahmed[1], Basheer Basheer[2], Giuseppe Mucci[3], Ranjith Ramasamy[4]

[1]College of Medicine, Ajman University, Ajman, United Arab Emirates; [2]College of Medicine, Jordan University of Science and Technology, Irbid, Jordan; [3]Regenerative Medicine, Bioscience Institute, Falciano, San Marino; [4]Urology Department, Jumeirah American Clinic, Dubai, United Arab Emirates

Correspondence: Ranjith Ramasamy, Email ranjithrama@gmail.com

Abstract: Peyronie's disease (PD) is an acquired fibrotic condition of the tunica albuginea that is characterized by penile curvature, deformity, pain, and impaired sexual function. PD is prevalent among all ages and is responsible for significant psychological distress. This review summarizes the current knowledge on PD, covering its epidemiology, risk factors, and natural history, along with a comprehensive overview of the medical, intralesional, and surgical options. While the traditional treatments are well-described in previous publications, the focus of the current review is on new biologic and regenerative treatments. Increasing attention to therapies such as platelet-rich plasma and stem cells is representative of the larger trend toward attempts at modifying the underlying fibrotic process, as opposed to merely correcting deformity. By integrating evolving regenerative strategies with proven therapeutic options and prioritizing patient-centered counselling, future PD care may become increasingly individualized, effective, and less invasive.

Keywords: Peyronie's disease, penile curvature, penile plaque, erectile dysfunction, review

Introduction

Peyronie's disease (PD) is an acquired fibrotic disorder of the tunica albuginea involving abnormal wound healing, leading to penile curvature, pain, deformity, shortening, palpable plaques and sometimes erectile dysfunction (ED).[1,2] In 1743, Francois Gigot de la Peyronie offered the first treatment for "induratio penis plastica", the condition that was subsequently named after him.[3] Peyronie's "plaque" (scar) forms due to abnormal production of the extracellular matrix, which is driven by increased myofibroblast activity and the upregulation of tissue inhibitors of matrix metalloproteinases, among other factors.[4]

PD is more prevalent than previously thought, with contemporary estimates ranging from 3% to 9% of men affected.[5,6] This is a significant increase from earlier reports suggesting a prevalence of less than 1%.[7] However, this difference is not due to a rise in prevalence but likely reflects underdiagnosis and methodological limitations in earlier studies. Improved awareness among both clinicians and patients may also partly contribute to greater recognition and diagnosis of the condition.

Prevalence figures also vary based on population and context. A community-based survey of 4432 men (mean age 57.4) reported a prevalence of 3.2%,[6] while a study of 448 men undergoing evaluation for ED (mean age 52.8) found a higher rate of 16%.[8] Among 534 men undergoing routine prostate screening, the prevalence was found to be 8.9%, with the mean age of affected individuals being 68.2 years.[5] Additionally, the prevalence of PD increases with age: 1.5% in men aged 30–39, 3% in men aged 40–49, 3% in men aged 50–59, 4% in men aged 60–69, and 6.5% in men over 70.[6] However, the true prevalence may be underestimated, as some men may be reluctant to report PD due to embarrassment, or older men may attribute it to aging. Men with ED who are not engaging in sexual intercourse may also be unaware of their condition, particularly if deformities are not apparent in the flaccid state.

PD typically manifests in two phases, an active (acute) inflammatory phase and a stable (chronic) phase. The active phase is characterized by penile pain and progressive deformity, while the stable phase is marked by plaque stability with the

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absence of pain and curvature progression for approximately 3–6 months, often occurring 6–12 months after disease onset.[9,10] Treatment varies depending on the phase of the disease, with severe pain usually resolving by the end of the active phase.[9]

In a review of 307 men with PD, the majority had dorsal curvature (46%), followed by lateral (29%) and ventral (9%).[11] In the largest study examining the natural history of untreated PD with at least one year of follow-up, 72% had dorsal, 17% had ventral and 11% had lateral curvature.[12] Pain in the active disease, typically occurring during erection, often resolves on its own as the disease transitions into the stable disease. In the same study of untreated men, 89% reported complete pain resolution.[12] The course of the disease differed among patients, of the aforementioned untreated men with curvature, about 12% had improvement, 40% remained stable, and 48% experienced worsening curvature. More than 80% also reported some loss of penile length.[12]

PD is frequently associated with ED, with ED being reported in up to 32% of men diagnosed with PD.[12] However, the relationship between the two conditions remains uncertain, whether one precedes the other or if they occur concurrently. Notably, fibrosis of the corporal cavernosal blood vessels has been implicated in both the development of vasculogenic ED and PD, suggesting a common underlying pathogenic mechanism.[13–16] In another study involving 309 patients with active stage PD, ED was observed in 37.5% of men.[17] Out of 1120 men with PD who underwent penile color duplex ultrasonography, 30.8% complained of decreased penile rigidity, while 18.5% were unable to sustain an erection.[18]

Multiple studies have identified a range of potential risk factors associated with PD. History of invasive procedures on the penis and penile or perineal trauma are among the most consistently reported contributors with invasive procedures on the penis being reported as the strongest risk factor.[19,20] Despite the association with trauma, only 20–30% of men with PD recall a specific traumatic event preceding the development of the condition.[20,21] A positive history of a sexual partner with fibromatous lesions of the genital tract or surgical intervention on the genital tract is also frequently reported in patients with PD compared to controls.[19,20] A genetic predisposition also appears to play a role, as up to 22% of men with PD have Dupuytren's contracture, and approximately 6.7% report a family history of PD or Dupuytren's disease.[22] The two conditions share similar patterns of gene expression, particularly involving collagen degradation, ossification, and myofibroblast differentiation. Other conditions that have been weakly associated with PD include urethritis, radical prostatectomy, plantar fascial contractures, tympanosclerosis, urethral instrumentation, Paget disease, gout, and lipomas.[19,20]

Diabetes has long been linked to both the presence and increased severity of PD.[23] A recent study further emphasizes this connection, showing that early-onset diabetes and poor glycemic control, reflected by elevated HbA1c levels, are linked to both the early development and progression of PD, with worsening control after PD diagnosis correlating with the formation of penile plaque.[24] In contrast, other vascular or metabolic factors such as hypertension, smoking, and hyperlipidemia have shown inconsistent or indirect associations,[20] likely due to their relationship with ED rather than PD itself. A recent pilot study showed that both insulin resistance and non-alcoholic fatty liver disease (NAFLD) are associated with the presence of PD, with NAFLD identified as a predictor of PD presence.[25] Studies evaluating additional factors such as alcohol use, sexually transmitted infections, and prior urologic surgery have not demonstrated a consistent connection.[20,26]

PD has significant psychological and emotional effects on affected men. Studies have shown high rates of emotional distress, with nearly half of patients meeting criteria for clinical depression and over half reporting relationship difficulties related to the condition.[27] Many also experience reduced self-esteem, social withdrawal, and strain on intimacy, highlighting the need for clinicians to recognize and address the psychological impact of PD alongside its physical symptoms.

Numerous previous studies have provided comprehensive overviews of current medical and surgical treatment options for PD. While this review will summarize established therapies and highlight recent updates, our primary aim was to explore novel and emerging treatment strategies, particularly regenerative approaches such as stem cell-based therapy and platelet-rich plasma in the management of PD.

Oral Treatment

Phosphodiesterase Type 5 Inhibitors

By blocking the conversion of cyclic guanosine monophosphate (cGMP) to GMP, phosphodiesterase type 5 inhibitors (PDE-5 inhibitors) raise cGMP and nitric oxide (NO) levels. This increase may play a part in scar remodeling by preventing the production of collagen and encouraging fibroblast and myofibroblast apoptosis, decreasing oxidative stress-associated inflammatory change.[28] When 35 men with an isolated septal scar were given tadalafil at a dose of 2.5 mg per day for six months, 69% of them had septal scar resolution, compared to 10% of untreated controls.[29] However, the majority of participants had no palpable plaque or curvature.[29] Another study compared 50 mg of sildenafil daily with 400 IU vitamin E over 12 weeks and found both treatments led to significant reductions in plaque volume and curvature, with no significant difference between groups.[30] However, the sildenafil group experienced greater improvements in erectile function (IIEF scores) and pain reduction.[30] Similar, another study showed that PDE-5 inhibitors shorten pain duration during the active phase of PD, but with no statistically significant improvements in curvature progression.[31]

Importantly, emerging evidence also supports daily low-dose tadalafil in active PD. In a retrospective study of 191 men, those taking tadalafil 5 mg daily showed lower curvature progression at 12 weeks (25.9% vs 39.7%, $p = 0.042$), significant improvement in IIEF-5 scores (19.3 vs 11.2, $p < 0.001$), and reductions in PD Questionnaire (PDQ)-Overall and PDQ-Pain scores seen only in the treatment group.[32] Early daily tadalafil therefore appears to slow curvature progression and improve erectile function and PD-related symptoms.[32] The use of PDE-5 inhibitors, particularly daily tadalafil 5 mg, shows potential as a promising first line treatment option (Figure 1)., though overall clinical evidence remains limited.[10,29,32]

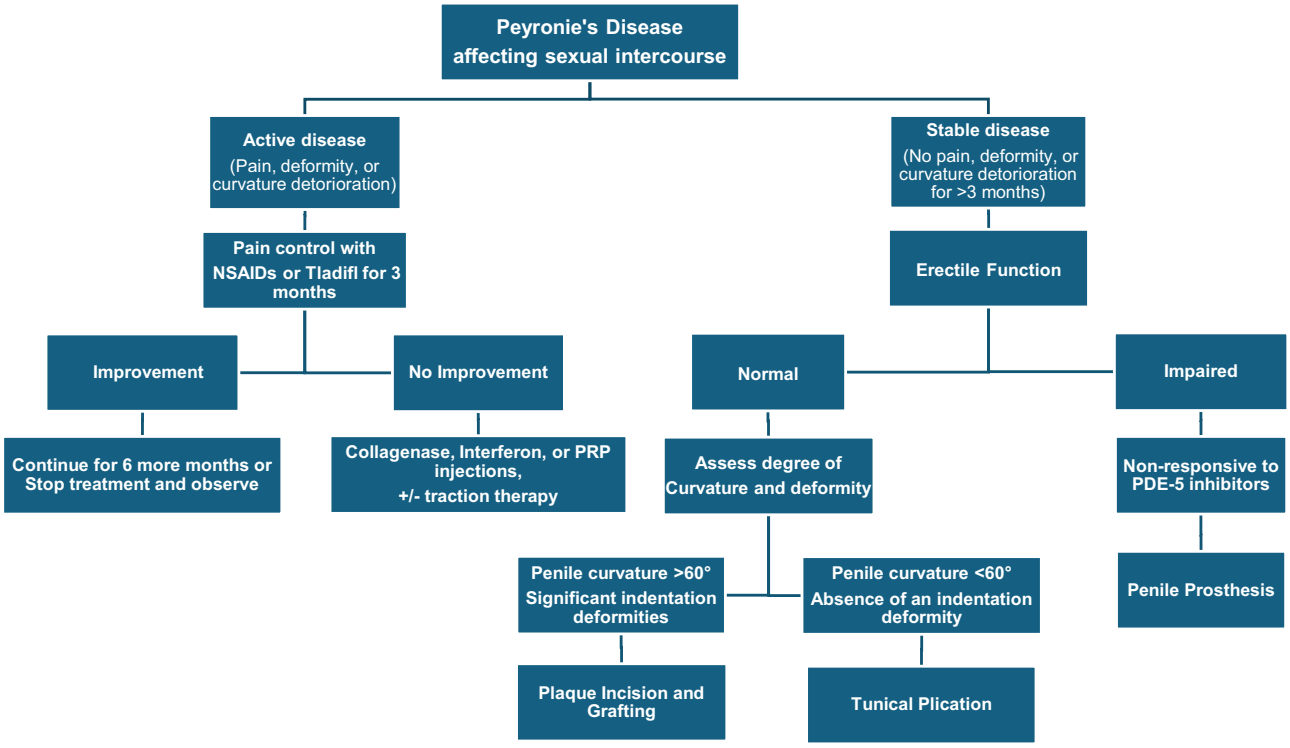

Figure 1 Treatment algorithm. Algorithm modified from the American Urological Association and Canadian Urological Association guidelines.[9,10]
Abbreviations: ED, erectile dysfunction; NSAID, non-steroidal anti-inflammatory drug; PDE5I, phosphodiesterase type 5 inhibitor, PRP, platelet-rich plasma.

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Others

Vitamin E has been used for PD since 1948,[33] because of its antioxidant properties, but controlled studies,[34] including a trial of 236 men, have shown no improvement in curvature or plaque size, even when combined with L-carnitine.[35] Tamoxifen, proposed for its effect on TGF-β,[36] likewise failed to improve pain, plaque size, or curvature in a placebo-controlled study.[37] Omega-3 fatty acids have not shown benefit, and procarbazine remains ineffective and carries substantial systemic risks.[38,39]

The use of oral treatments with vitamin E, tamoxifen, procarbazine, omega-3 fatty acids, or vitamin E combined with L-carnitine is not advised.[9]

Colchicine, which interferes with tubulin and may prevent the buildup of collagen, has yielded conflicting results. While observational studies reported improvements in pain and curvature,[40,41] a randomized trial involving 78 men revealed no significant change in curvature or plaque size when compared to a placebo,[42] subsequent research found both benefit,[43] and no extra benefit when combined with vitamin E.[44] Potassium para-aminobenzoate (POTABA), an antifibrotic agent, demonstrated reductions in plaque size and slowed curvature progression in a 12-month placebo-controlled study with a high risk of bias.[45] Its value as a treatment option is limited by high cost, large daily doses, and a high rate of gastrointestinal intolerance. Pentoxifylline, which may reduce TGF-β–related fibrosis,[46] has shown mixed but encouraging results. A randomized study without a placebo found similar curvature and plaque improvements across groups, with better pain and erectile function outcomes when oral pentoxifylline is combined with verapamil intralesional therapy.[47] An observational study reported plaque stabilization or improvement in 92% of men on pentoxifylline versus 44% of untreated controls.[48] More recently, in a study of 307 men with early-stage PD, combination therapy including perilesional pentoxifylline injections showed greater overall improvements in plaque volume and curvature compared with oral therapy alone.[49] Coenzyme Q10 has shown good promise in early stable PD, with a randomized trial of 186 men demonstrating improvements in pain, erectile function, and reductions in plaque size and curvature.[50]

While colchicine, POTABA, pentoxifylline, and Coenzyme Q10 may be considered on an individual basis or as part of a combined treatment approach,[10] the overall evidence remains limited, and current data are not strong enough for formal recommendations until larger, well-designed studies are completed.[9]

Mechanical Treatment

Penile Traction

Penile traction works by gently stretching the penis over time. This can change the way connective tissue and collagen behave in PD, which may help reduce plaque tightness, improve elasticity, and preserve length.[51] In small studies, men who used traction for a few hours each day over several months saw modest improvements in curvature, small increases in flaccid length, and slight gains in girth.[52,53] Some studies have combined traction with oral or injection treatments, but the results are inconsistent.[54–56] It must be noted that all studies vary in the time period the devices are applied and the manner in which this is performed. How the therapy is used in terms of: device type, daily duration, and amount of stretch, varies a lot, and patients are often left to figure it out themselves. While there is still a need for larger, high-quality trials, traction is low-risk and relatively inexpensive, making it a reasonable option for men with PD.[10]

Radiation Therapy

Eight observational studies have examined the use of radiation therapy (RT) for PD, using a wide range of doses, from about 2 Gy up to 45 Gy.[57–64] Follow-up was often long, with several studies reporting outcomes beyond five years. Most were single-arm studies; only one compared two different RT regimens with a no-treatment group.[58] Across these studies, outcomes varied widely. Curvature improved in a minority of men (roughly 6–52%), but some also reported worsening. Similar patterns were seen with plaque size. In the one study that included an untreated comparison group, rates of improvement were essentially the same regardless of whether men received RT or no therapy at all.[58] Pain often improved, but again, the no-treatment group showed similar changes.[58] Because PD symptoms naturally fluctuate over time, and pain in particular often improves on its own, these uncontrolled designs offer little evidence that RT has any meaningful effect. Most outcomes relied on patient impressions rather than objective measurements, further limiting the

strength of the findings. Considering the absence of demonstrable benefit and the risks associated with radiation exposure, RT is not recommended for the treatment of PD.[9]

Low-Intensity Shockwave Therapy

Low-intensity Shockwave therapy (LiST) uses focused shock waves on the penile plaque with the idea that it might soften the tissue and improve blood flow. Early observational reports showed mixed results, some showed no change at all in curvature, while others claimed big improvements but the methods were inconsistent, and the results did not line up well with each other.[65–69] However, most observational studies that addressed plaque outcomes were consistent in claiming there was no measurable benefit.[65,66,68] When LiST was tested in randomized studies, the findings were much more consistent: it helped with pain, but it did not change curvature or reduce plaque size.[70–72] A randomized trial of 100 men showed exactly that: less pain, but no structural improvement.[70] Reviews of the broader literature show the same thing.[73] Because of this, guidelines only support LiST for pain control in PD, but it is not recommended for treatment of curvature or plaque.[9,10] There are also some concerns about possible downsides, like fibrosis or new scarring, so its use is generally limited.[74]

Site-Specific Treatment

Topical Therapy

Verapamil Gel

Interest in topical verapamil arose from its use in scar modulation, but early testing showed that the medication does not cross the tunica albuginea,[75] making a direct effect on Peyronie's plaques unlikely. Even though a later controlled trial reported less pain and patient-reported curvature improvement with verapamil gel compared with placebo, the study did not include objective curvature measurements.[76] Given these limitations, current guidance does not support topical therapy for PD outside of clinical studies.[9]

Iontophoresis

Iontophoresis has been studied as a method to push charged drugs through the skin into the tunica albuginea, to improve local delivery. Results of iontophoresis with verapamil have been inconsistent: one study noted significant improvements in plaque volume and curvature,[77] while another found no real benefit.[78] Because the evidence is mixed and the treatment is time-consuming, iontophoresis is not advised for managing PD.[9]

Intralesional Therapy

Verapamil

Intralesional verapamil (ILV) has been employed as a localized therapy for PD for more than two decades. Mechanistic investigations demonstrate that verapamil interferes with PDGF beta-mediated collagen production and suppresses early fibroblast proliferation, processes that are fundamental to plaque development.[79,80] The technique, along with the earliest clinical experience, was first documented in 1994,[81] where a biweekly injection schedule was shown to stabilize or reduce plaque dimensions and improve curvature. Subsequent years saw progressive refinements to the administration protocol,[82] although early approaches generally relied on injections delivered every two weeks for roughly a 12-week course.[81,82] While no available animal model fully mimics human PD, ILV has consistently demonstrated histological reductions in collagen and elastin fibers as well as improvements in erectile function in experimental systems.[82] Across published reviews, most clinical reports describe improvements in curvature, plaque size, and pain, despite the notable variability in dosing and technique between studies.[82,83] Symptomatic relief of pain tends to occur early in the treatment course, a pattern also noted when ILV is administered alongside oral antioxidant therapy.[84] Overall, ILV has been examined in 11 controlled study designs, comprising two randomized controlled trials and nine observational studies.[9,85–87]

Despite advances in the understanding of PD biology and refinements in local therapeutic approaches, key aspects of the optimal ILV regimen remain unsettled. Treatment success appears to hinge on factors such as injection volume, concentration, frequency, and duration, with more extended courses of higher concentration

ILV producing the most favorable outcomes in younger patients who present with small plaques yet marked curvature.[83] Adverse effects are predominantly local, typically bruising, swelling, or discomfort at the injection site, while systemic safety remains excellent. Direct comparisons between ILV and alternative treatments are challenging because available studies frequently involve patients in the active phase of the disease, employ heterogeneous protocols, use differing endpoints, lack imaging confirmation of precise plaque targeting, and sometimes yield conflicting results.[9] Factors consistently associated with a better treatment response include higher verapamil dilutions, younger age, and more pronounced baseline curvature.[85,87] In line with this evidence, the American Urological Association continues to recommend ILV as a therapeutic option for appropriately selected patients with PD.[9]

Interferon Alpha-2b

Intralesional interferon therapy for PD has been studied extensively, by one large multicenter randomized trial,[88] and eight observational studies.[89–96] Interferon inhibits fibroblast activity involved in plaque formation,[97] and is usually administered biweekly for six weeks.[88] Clinical outcomes show meaningful benefit, including a 13.5° reduction in curvature compared with saline in a randomized study of 117 men,[88] and a follow-up series of 127 patients in which 54% achieved at least a 20% curvature improvement with an average overall reduction of 9°.[89] The follow-up alpha-2b study demonstrated curvature improvement without affecting penile vascular parameters and this effect was independent of pretreatment curvature or disease duration.[89] Evidence from early-stage disease shows no significant treatment effect, indicating that intralesional interferon is most appropriate for patients with stable PD.[98] Common adverse effects include sinusitis, flu-like symptoms, and localized injection reactions, which are generally manageable with hydration and non-steroidal anti-inflammatory medications.[9,85] Severe systemic reactions have been reported only at higher doses.[97] Interferon alpha-2b is considered an appropriate option for men with curvature of 30 degrees or more and without significant plaque calcification (Figure 1).[9]

Collagenase

Collagenases are enzymes capable of breaking down interstitial collagen. The US Food and Drug Administration (FDA) and Health Canada (HC) have both approved Xiaflex™, collagenase clostridium histolyticum (CCh) for the treatment of PD (Figure 1).[9,10] Direct injection of clostridial collagenase into PD plaques has shown meaningful clinical benefit, including notable reductions in penile curvature and plaque dimensions, along with improvements in patient-reported PD symptom burden across several trials. Two major multicenter, randomized, placebo-controlled studies (IMPRESS I & II),[21,99] helped define which patients are most suitable for intralesional collagenase combined with penile modelling: individuals with stable disease, curvature between 30° and 90°, absence of isolated hourglass deformity or calcified plaques, and intact erectile function (with or without pharmacologic support). In these trials, men who received CCh achieved an average 34% reduction in curvature (−17.0±14.8°), compared with an 18% reduction (−9.3±13.6°) in those given placebo, along with significantly lower PD bother scores.

Although generally well tolerated, adverse events such as penile hematoma, localized pain, and swelling were reported. Corporal rupture is a rare but recognized complication and may require surgical correction.[100–102] The effectiveness of this therapy has not been assessed in men with hinge deformities, ventral curvature, hourglass narrowing, or curvature outside the 30°–90° range.[21,99] Ongoing work continues to refine treatment regimens and to explore outcomes in patient populations beyond the original trial criteria, which is essential for understanding its broader applicability.[85,103,104]

Corticosteroids

Current guidelines advise against the use of corticosteroids for intralesional therapy in PD.[9] Although corticosteroids were among the earliest agents evaluated for injection therapy,[105] the clinical benefits observed were minimal and accompanied by an unfavorable profile of adverse effects. Notably, in several studies, placebo injections have produced improvements comparable to those seen with corticosteroids, suggesting that the mechanical disruption of the plaque, rather than the drug itself, may have accounted for the therapeutic response.[106] Given the combination of limited efficacy

and risks such as wound infection, local bruising, and bleeding, the routine use of intralesional corticosteroids in PD is not supported.[107]

Hyaluronic Acid and Botulinum Toxin A

Hyaluronic acid (HA) is a glycosaminoglycan with recognized immunomodulatory properties, particularly downregulation of inflammatory cytokines. Thus, it has been used therapeutically to limit inflammation and scar formation. Clinical evidence from two retrospective analyses and a more recent prospective study indicates that HA may contribute to reductions in plaque dimensions and penile curvature, along with improvements in erectile rigidity.[108–110] Botulinum toxin, which has demonstrated antifibrotic and anti-scarring activity, has also been explored as a potential intralesional therapy. In the single available study assessing botulinum toxin A for PD, the treatment appeared safe and yielded improvements in curvature, though broader confirmation is still lacking.[111] The evidence base remains insufficient to support routine clinical use of either HA or botulinum toxin. Their administration should be restricted to research settings with comprehensive patient counselling regarding the limited data available.

Platelet-Rich Plasma

Platelet-rich plasma (PRP) which is derived from peripheral blood, and prepared by centrifuging the blood sample to isolate the platelet-rich fraction, has been used in treating musculoskeletal injuries, wound healing, and dermatological conditions.[112] Recent studies have highlighted platelets' role beyond clotting as they are rich in growth factors (GFs),[113] which are believed to have potential in regenerative medicine. Key GFs released by platelets during inflammation include platelet-derived GF, fibroblast GF, epidermal GF, insulin-like GF (IGF), and interleukins.[114] PRP's properties are said to promote tissue proliferation and collagen deposition by activating fibroblasts.[115] Consequently, PRP has gained interest as a biologic option for PD. However, it remains an experimental, off-label therapy with no established evidence grade and is not currently recommended in clinical guidelines. Several small contemporary studies in PD have reported modest improvements: a 36-patient protocol combining PRP, needle tunneling, and modeling showed a reduction in curvature from 57.5° to 40.9°,[116] while a 65-patient series demonstrated mean improvements of 16–17° after roughly six injections, accompanied by reduced intercourse-related pain and better sexual function in many men.[117] A two-center pilot cohort of 38 patients who received two injections 4 weeks apart, reported a median change from 55° to 45° and smaller plaque dimensions without adverse events,[118] and a Phase 2 randomized placebo-controlled crossover trial noted improvement from 40° to 25° at six months in the group receiving PRP first.[119] Despite these positive findings, controlled evidence remains limited, study protocols vary widely, and sample sizes are small. Although PRP may be considered for managing PD in carefully selected patients (Figure 1), they should be informed about the lack of regulatory approval, and its use should be regarded as investigational therapy. Further standardized clinical trials are necessary to establish its efficacy.

Stem Cell Therapy

Adipose-derived mesenchymal stem cells are attractive because they are abundant, easy to isolate, and possess immunomodulatory properties, including secretion of growth factors that promote angiogenesis and regulate immune activity.[120,121] They can also reduce fibrosis by downregulating profibrotic genes such as COL1A1 and ACTA2.[122,123] Animal studies show that adipose-derived mesenchymal stem cell therapy improves erectile function and reduces tunica albuginea fibrosis in both active and stable PD models,[124–126] and genetically modified adipose-derived stem cells expressing interferon alpha 2b further enhance functional recovery.[127] Early human work using placental matrix-derived mesenchymal stem cells demonstrated considerable regression of plaques and moderate improvements in penile curvature.[128] However, human studies to evaluate the feasibility of stem cell therapy for PD remain scarce.[129]

A recent pilot study investigating autologous adipose-derived regenerative cells in men with chronic PD found no statistically significant changes in penile curvature, though notable improvements were reported in secondary outcomes such as plaque size and patient-reported distress.[130] Mesenchymal stem cell-based treatments for male sexual dysfunction are advancing into clinical trials, with preliminary data suggesting that intrapenile injection of autologous bone marrow or adipose-derived mesenchymal stem cells is safe.[131] While preclinical studies show promise for their use in penile fibrosis, further research is needed to clarify their mechanism of action and determine the ideal timing and mode of administration for clinical use.[132]

These therapies should be regarded as investigational at this stage, with rigorous, well-designed clinical trials necessary to confirm their safety profile and therapeutic benefit before they can be considered for broader clinical application. Patients can access information on ongoing regulated trials through the clinical trials registry. Caution is necessary when interpreting studies combining stem cells with other modalities, such as stromal vascular fraction and shock wave therapy because of methodological limitations and lack of controlled design.[133]

Surgical Treatment

Surgical management is the definitive therapy for men with refractory or severe PD that compromises sexual intercourse or causes significant distress. The main indication for surgery is the resolution of the active phase of the disease, which is confirmed by 3 months of stable penile deformity and 6–12 months from the onset of the disease. Performing surgery during the stable phase appears to reduce the risk of postoperative recurrence or progression of deformity.[9,134–137] Preoperative evaluation should cover a detailed assessment of the penile length, degree of curvature, erectile function, and response to pharmacologic therapy in cases of ED.[136] For patients with PD and ED unresponsive to pharmacotherapy, penile prosthesis implantation is recommended, either alone or combined with adjunctive straightening procedures such as modeling, tunical plication, or incision/excision with or without grafting.[135] The use of synthetic grafts is discouraged due to higher complication and infection rates.[9,135]

Tunical Plication

Tunical plication, also referred to as penile plication, shortening procedure, or corporoplasty, is the preferred method for the correction of mild to moderate penile curvature (<60°), in the absence of an indentation deformity (Figure 1). The procedure achieves penile straightening by reducing the length of the convex (unaffected) side of the penis, which restores the axial alignment during erection and preserves erectile function.[134,135] The Nesbit corporoplasty laid the foundation of shortening procedures, which include elliptical excision of the contralateral tunica albuginea from the maximal curvature point, followed by closure with non-absorbable sutures.[138] Subsequent modification directed their focus on reducing invasiveness, preserving penile length, and minimizing postoperative complications. The Yachia technique replaced excision with a longitudinal tunical incision closed transversely.[139] The Essed-Schroeder (TAP) procedure modified this concept by omitting tunical incision or excision entirely, and relying on non-absorbable sutures to bunch the tunical tissue. Usage of Knispel's inverted sutures reduced suture palpability.[140,141] Modern adaptations, such as the 16- or 24-dot Lue technique, use a series of plication sutures in a Lembert pattern, with the number of sutures tailored to the degree and distribution of the deformity.[142] Baskin-Duckett and Baskin-Levine TAP modifications combine limited tunical incisions with plication.[143]

Despite all the procedural modifications, no single tunical plication technique revealed clear superiority in long-term outcomes or patient satisfaction levels. Generally, clinical outcomes of tunical plication procedures are favorable. Rates of complete penile straightening range from 85% to 100%, with overall patient satisfaction ranging from 52% to 100% across multiple retrospective series evaluating Nesbit, Yachia, Essed–Schroeder, and related plication techniques.[135] Penile shortening remains the most encountered postoperative concern, reported in up to 22–69% of patients particularly among those with severe (>60°) curvatures.[135] Palpable knots are reported in 13.2% to 27.4% of cases, though only a minority describe them as bothersome.[144] Residual curvature, altered penile sensation, or ED are uncommon complications.

Plaque Incision and Grafting

Plaque incision or excision is reserved for patients with complex deformities, including those with penile curvature >60° (Figure 1), significant indentation deformities such as notching, hourglass, or hinge effects, and for men without pre-existing ED or high risk of postoperative ED.[134,135] Unlike tunical plication, which corrects curvature by shortening the convex side, grafting techniques restore penile length and axial symmetry by expanding the concave (affected) side through plaque incision or partial excision, followed by placement of a graft to cover the tunical defect.[85,136,138]

Various graft materials have been described, including autologous tissues such as the saphenous vein, dermis, and buccal mucosa, as well as allografts and xenografts like pericardium and small intestinal submucosa.[85,139–141] Although

synthetic grafts were historically used, contemporary AUA guidelines strongly advise against their use due to higher risks of infection, contracture, and reduced elasticity.[9,135]

Studies report penile straightening rates of 83.6%, with overall patient satisfaction ranging from 75% to 80%.[145] However, grafting procedures carry a higher risk of postoperative ED compared with shortening techniques. Additional complications include recurrent curvature, up to 44% with pericardial grafts,[146] and 37–75% with submucosal grafts,[147] and graft contracture (up to 25%).[148] Thorough preoperative assessment of erectile function, deformity severity, and patient goals is essential before choosing grafting.[9]

Recent studies emphasize individualized graft selection, meticulous microsurgical technique, and minimal dissection to preserve neurovascular integrity and optimize outcomes. When appropriately selected, plaque incision and grafting provide durable curvature correction and satisfactory penile length preservation in men with severe or complex PD deformities.[143,148]

Penile Prosthesis

Penile prosthesis implantation is the treatment of choice for men with PD who have ED unresponsive to pharmacotherapy (Figure 1). In these patients, fibrotic plaque, penile curvature, and compromised rigidity make intercourse difficult, and pharmacologic agents often fail to restore functional erections. A prosthesis provides reliable rigidity and can simultaneously address penile deformity.[9,149]

Mild curvature may partially correct itself once the cylinders are expanded, while more pronounced deformities can be managed intraoperatively with manual modelling.[150] In complex cases, tunical reconstruction with plaque incision, with or without grafting, can be performed before or after prosthesis placement depending on intraoperative findings.[151] Small defects are sometimes covered with graft material if needed, with the primary goal of restoring length and achieving a straight, functional erection. Advanced length-restoration techniques can yield a median length increase of about three centimeters.[152,153]

Outcomes are generally excellent, with length-restoring techniques providing meaningful gains; early sliding-technique cases reported increases of up to 3.2 cm, and a subsequent 23-patient series showed an average 2.8 cm improvement with 90% of patients satisfied with both cosmetic and functional results.[153] The main source of dissatisfaction remains perceived shortening.[154,155] Complications include device-related issues, urethral injury during modelling, residual or recurrent curvature, temporary changes in glans sensation, and, rarely, glans ischemia. Revision surgery may be required for recurrent deformity, device failure, or persistent curvature, with prosthesis-based reconstruction remaining the most effective option in such cases.

Surgical correction remains the cornerstone of treatment for men with PD whose deformity persists despite the resolution of the active phase and significantly limits sexual function. The choice of technique depends primarily on curvature severity, deformity characteristics, baseline erectile function, and patient expectations (Figure 1). Shortening procedures such as tunical plication provide high straightening rates with a low risk of postoperative ED, making them suitable for men with mild to moderate curvature and preserved rigidity. For patients with severe or complex deformities, plaque incision or excision with grafting offers better preservation of penile length, although this comes with a higher incidence of de novo ED. Men with PD and medically refractory ED benefit most from penile prosthesis implantation, which reliably restores rigidity and allows simultaneous correction of curvature through modeling, plication, or limited grafting when required. Overall, careful patient selection, preoperative counseling, and meticulous intraoperative technique are essential to achieving optimal functional and satisfaction outcomes across all surgical modalities.

Conclusion

PD is a fibrotic disease that results in penile deformity, ED and serious emotional distress. Modern treatments include intralesional therapies and surgical treatment in patients with severe deformities. Novel regenerative therapies such as platelet-rich plasma and stem cell therapy appear promising at an early stage in terms of their potential to reduce fibrosis, reduce curvature, and improve sexual function, offering avenues for future care. Yet evidence is scarce, and large, well-designed studies are necessary for defining the safety, efficacy and role in a multimodal treatment field. Ongoing study

and clinical experience with these advances in care can further develop treatment options, offering more personalized and minimally invasive strategies for men affected by PD.

Disclosure

The authors report no conflicts of interest in this work.

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