

Delayed pressure urticaria

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Delayed pressure urticaria (DPU) is a physical urticaria. In physical urticarias, a reproducible whealing occurs in response to a specific physical stimulus. DPU is a mechanical urticaria in which pressure is the physical stimulus that causes the whealing. In this condition, delayed cutaneous erythema and edema occur in association with marked subcutaneous swelling after the application of a sustained pressure stimulus to the skin. These signs occur as early as 30 minutes and typically 4 to 6 hours later. Lesions may persist for up to 48 hours [1]. The response is dependent on the degree of pressure, duration of the stimulus, body site affected, and activity of the disease, which is variable in intensity [2]. Swelling of the hands and feet that is provoked by pressure is indistinguishable from swelling caused by idiopathic angioedema. DPU rarely occurs alone. Concomitant chronic idiopathic urticaria (CIU) and angioedema are usually present. DPU lesions are distinguished from CIU lesions, because the wheals of DPU are provoked. DPU is distinguished from other physical urticarias because the lesions are delayed in appearance, last for more than 24 hours, and often are associated with systemic complaints that can be disabling. In occasional patients in whom whealing seems to be provoked solely by a pressure stimulus at a single point in time, CIU and angioedema almost certainly have been present in the past or will appear in the future [3,4]. DPU is a chronic urticaria in which responses to pressure are variable from week to week [5]. This fact makes any response to therapy difficult to evaluate.

As in other physical urticarias, DPU occurs frequently with other physical urticarias [6] in addition to CIU and angioedema. Patients with DPU frequently demonstrate delayed dermographism (ie, a late-onset linear whealing response to a frictional stimulus). Some investigators consider delayed dermographism to be a manifestation of DPU [7]. A wheal-and-flare reaction may occur within minutes

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of stroking the skin; these reactions disappear and recur several hours later [8]. Whealing need not occur immediately, however, and may develop between 30 minutes and 6 hours later [9]. Because not all patients with DPU have delayed dermatographism, it can be considered as a separate disorder in some cases. The incidence of DPU varies depending on the dermatology department from which it is reported.

Early reports of delayed pressure urticaria

The first article to report DPU was published in 1929. The patient had developed large urticarial wheals after wearing a belt and developed swellings on the soles of his feet after walking. The relationship between the pressure applied and the lesions provoked was confirmed by placing a 15-kg tool box over the patient's shoulder. An erythematous swelling appeared 4 hours later in this area and remained for 48 hours. The patient also demonstrated delayed dermatographism [9].

Another early article underlined the close relationship between DPU and delayed dermatographism. The patient showed an immediate and a delayed dermatographic response to friction but also reacted to the pressure stimulus of wearing a belt and carrying a golf bag. The authors considered the diagnosis to be delayed dermatographism [7].

Pathogenesis

The pathogenesis of DPU is not known, although a number of mechanisms have been proposed. The timing of the reaction after the application of pressure to the skin, the morphology of the lesions, and the infiltrating cells seen on histopathologic examination resemble those of a late-phase reaction [10], although no early cutaneous reaction is visible.

It has been suggested that DPU may represent a type III or other reaction to an unknown allergy because of its timing and histopathology [11]; however, there is no evidence of primary vascular damage, direct immunofluorescence is negative, and complement levels are normal.

There have been reports implicating food allergens in DPU [12,13]; however, an elimination diet given to patients with DPU who showed a late cutaneous reaction to food allergens failed to have any impact on the disease, suggesting that food allergens are unlikely to be implicated in the pathogenesis of the disease [14].

Ryan et al suggested that mast cells have a role in DPU, as intradermal injections of compound 48/80 caused histamine release, which prevented the induction of lesions in the affected area [11]. Histamine is released in the lesions [15], but the unresponsiveness of the condition to antihistamine treatment makes it unlikely that histamine is of major importance in the pathogenesis of the reaction.

One study reported increased chemotactic activity of a substance with the dermatographic properties of leukotriene B₄ (LTB₄) in lesional skin [16]. In

another study, measurement of eicosanoids prostaglandin E₂, LTB₄, LTC₄, LTD₄, LTE₄, and 12 HETE in skin exudates collected immediately after pressure was applied and 6 hours later showed no significant increase compared with control levels [17]. This finding suggests that increased metabolism of arachidonic acid does not have a major part in the early or late development of lesions.

IL-6 is involved in the reaction in DPU [18]. The concentration of IL-6 increased significantly in suction blister fluid in lesional skin. Increased concentrations of IL-6 are found in lesional skin in several skin diseases and it is unlikely that it has a primary role in DPU. Eosinophil granule major basic protein is found extracellularly in the dermis in lesional skin, demonstrating that eosinophils are involved in the reaction [19].

Diagnosis and methods of pressure testing

As in all physical urticarias, reproducible whealing occurs in response to a specific physical stimulus. Pressure, defined as the force applied to a unit area of surface, produces reproducible whealing in DPU. The disease is variable, and remissions and exacerbations occur. The response to pressure stimulus is related to the severity of the disease, the amount of pressure applied, the duration of pressure application, and the anatomic area tested. Sites that previously have reacted to pressure have been found to be refractory to an additional pressure stimulus for at least 24 to 48 hours [2].

The diagnosis is based on history and reproducing the lesions, if possible. Because most patients with DPU have CIU and angioedema [3,4], DPU should be considered in all patients with CIU whose disease is unresponsive to antihistamines. Many patients are unaware of the pressure component of their disorder, and direct questioning is necessary to obtain the relevant history. Lesions occur after certain activities. Sitting on hard chairs provokes lesions on the buttocks and thighs. Patients who garden and cross their legs when sitting develop lesions on their knees.

Carrying a bag over the shoulder or arm induces lesions in these areas. Leaning against furniture, wearing seat belts, and lying on hard mattresses result in delayed swelling on the areas in contact with the pressure stimulus. Swelling of the feet that is indistinguishable from swelling caused by idiopathic angioedema occurs after walking, jogging, running, and climbing ladders. Hand swelling is provoked by carrying shopping bags, using screw drivers, and handling the steering wheel. Lesions occur frequently under tight clothing, bra straps (Fig. 1), sleeves, cuffs, shoes, watches, and belts. In patients with a history suggestive of DPU, pressure testing should be considered.

There is no standard method of pressure testing. A positive response after any form of pressure challenge consists of the appearance of palpable lesions after at least 30 minutes. Because most positive responses occur at 6 hours, observers usually read pressure tests at 6 hours. Ryan et al tested for DPU by hanging a 15-lb weight at the end of a crepe bandage over the shoulder, thigh, or



Fig. 1. Pressure swelling provoked by a bra strap.

forearms of patients for at least 15 minutes [11]. Using this method, the area to which the force was applied is not defined, so its reproducibility is not accurate enough for clinical trials, but the method is useful in clinical settings.

Warin suggested a simple outpatient test that the patient could perform at home. His clinic gave patients a glass sphere approximately 1.4 cm in diameter that would localize the pressure and form a weighted cuff. Patients were instructed to place three wine bottles full of water, one house brick, or about 4 kg of groceries in a plastic bag. A tea towel was looped through the handles of the bag and tied over the forearm while sitting down and resting the hand and wrist on a table. The glass sphere was placed beneath the tea towel on the upper surface of the forearm. Patients were instructed to leave the weight in place for 5 minutes and then remove it. The test was performed 6 hours before the patients' outpatient appointments. A visible and palpable swelling is considered positive [4].

Illig and Kunick designed an apparatus in which metal rods are held vertically in place, resting on a patient's back, using a thick plastic sheet of material (Perspex) to keep the rods in place. The weight applied can be increased by adding additional known weights to the rods. The times of application of the weights vary depending on the severity of disease [20]. Modifications of this instrument have been used by other investigators studying DPU. Lawlor used a similar instrument with rods of 1.5-cm diameter and pressures of between 2.29 kg and 4.79 kg that were rested on the back for between 12 minutes and 15 minutes (Fig. 2) [17]. Barlow used rods that were 1.5 cm in diameter and weights of 2.5 and 3.5 kg resting on the anterior thighs for 20 minutes [21]. A positive response at 6 hours is shown (Fig. 3).

Estes and Yung used a calibrated dermatographometer to reproduce lesions in two patients. They applied pressures of 48, 73, 103, 136, 200, and 234 g/mm²



Fig. 2. Pressure instrument adapted from Illig and Kunick [20] with weighted rods resting on a patients' back.



Fig. 3. Experimentally produced pressure papules using the dermographometer shown in Fig. 4.



Fig. 4. A calibrated dermographometer. The setting of 10 on this dermographometer corresponds to a pressure of application of 9.75×10^5 Pascals. The tip is pressed perpendicularly into the skin and held in place for 5, 15, 30, 70, or 100 seconds.

for 10 seconds to the abdomen, arms, and back, and inspected the sites for up to 24 hours. They showed a threshold whealing response of 136 g/mm^2 on the abdomen. By applying these pressures to the back for 5, 30, 60, 120, and 180 seconds, respectively, they demonstrated that the time needed to produce a whealing response was inversely proportional to the degree of pressure applied [2].

Lawlor used a modification of the previous method as a pressure test. A calibrated dermographometer (Fig. 4) with a spring-loaded tip measuring 0.9 cm in diameter was held firmly and pressed against the skin at right angles to the back lateral to the spine. The pressure applied was 9.75×10^5 Pascals (99.4 g/mm^2), and the durations of application were 5, 15, 33, 70, and 100 seconds (see Fig. 4; Table 1).

Table 1
Settings used to pressure test with a dermographometer

Marking	Pressure (Pascals)	Time (s)
10	9.75×10^5 (99.4 g/mm^2)	5
		15
		33
		70
		100



Fig. 5. Experimentally produced pressure swelling using the pressure instrument shown in Fig. 2.

These times of pressure application produced a graded response in terms of increasing papule size and an increase in the number of positive responses from 5 to 100 seconds (Fig. 5) [5]. Barlow also used a dermatographometer to pressure test. A dermatographometer set at 100 g/mm² for 70 seconds proved optimal for diagnosis [21]. If purchasing these instruments from a small instrument firm, it is important that pressures of 9.75×10^5 Pascals can be applied. Alternatively, university and hospital medicine and physics departments can design and supply these instruments.

A skin biopsy performed in a patient with difficult urticaria may point toward the diagnosis of DPU; the condition should be differentiated from urticarial vasculitis, which also may demonstrate pressure-induced wheals. Urticarial vasculitis shows venulitis with leucocytoclasia or fibrinoid necrosis. In DPU, there are no vessel-related changes. The inflammatory infiltrate is more striking than that of CIU. The histopathologic changes usually are located in the dermis and subcutaneous tissue, but the inflammatory infiltrate may extend deeply into the subcutaneous fat. In lesions present for less than 5 hours, neutrophils and eosinophils predominate. In lesions present for longer than 24 hours, the infiltrate is composed mainly of mononuclear cells and eosinophils [1]. Immunohistochemistry of lesions has shown up-regulation of E selection at 6 hours and 24 hours after pressure application [22]. Using paraffin-embedded tissue and immunofluorescence technique, major basic protein, the core protein of the eosinophil, has been demonstrated extracellularly in the connective tissue, indicating that degranulation of eosinophils occurs [19]. Direct immunofluorescence examination for deposition of immunoreactants in the affected skin is negative [2,3,11].

Clinical features and natural history

The whealing in DPU consists of erythema and cutaneous and subcutaneous swelling. Infrequently, a bulla appears over an urticated lesion. Edema of the

hands and feet may be difficult to distinguish from idiopathic angioedema. The lesions may be itchy, burning, or painful or may cause all of these symptoms. Arthralgia sometimes presents in joints close to the pressure-induced lesions. During severe exacerbations, some patients experience a flu-like illness with malaise, tiredness, and rigors [1,11]. The time of onset of lesions after a pressure stimulus to the skin varies. In one series, swelling appeared as early as 30 minutes after pressure and as long as 9 hours after pressure (mean onset of swelling, 3.5 hours) [1]. In other series, most patients reported swelling after 4 to 6 hours [11,16]. In this condition, which has systemic features, laboratory abnormalities can be found. The erythrocyte sedimentation rate can be elevated. The reported incidence varies from 17% [5] to 71% [16].

Levels of the protease inhibitors α -1 antitrypsin and total antitrypsin have been found to be increased significantly, suggesting an acute-phase reaction [23]. A neutrophil leucocytosis without eosinophilia may occur. Between 20% [1,16] and 33% [16] of patients have been shown to have an increase in neutrophil levels.

In one study, the age of onset of DPU ranged from 5 to 63 years. The peak onset occurred during the third and fourth decades. The duration of the disease ranged from 1 to 40 years until remission or the time the study was performed. The mean duration of the disease was 9 years; however, the condition fluctuates, and remissions of up to 1 year have occurred during the course of the disease [1]. Each patient's disorder varies greatly in severity over weeks to months [4,5]. As previously mentioned, the reported incidence of DPU in patients presenting to a dermatology department varies. The incidence over a 32-year period was 2% among 2310 patients with urticaria [24]. Barlow reported an incidence of 37% in 135 patients who were pressure tested consecutively in an urticaria clinic [21]. This higher incidence may reflect a specific interest in the physical urticaria, the methods used to study the disease, and the fact that clinical idiopathic urticaria itself is frequently more severe in areas of the body where pressure is applied (ie, under belts or clothing). The higher incidence reported suggests that it is common for patients with chronic urticaria to have areas that worsen with pressure. It is debatable as to whether this condition is mild DPU or is a pressure-induced worsening of CIU and angioedema.

Impact of delayed pressure urticaria on quality of life

The impact of DPU on quality of life has been studied [25]. Two questionnaires were used: a disease-specific, purpose-designed questionnaire (urticaria questionnaire) and the Nottingham Health Profile (NHP). A total of 69 patients with CIU and 73 patients with DPU completed self-administered questionnaires.

Data from the urticaria questionnaire referred to the month before completing the questionnaire. This questionnaire covered facets of everyday life, including home management, personal care, recreation and social interaction, mobility, emotional factors, sleep and rest, and work. Whereas patients with CIU reported problems in all areas, patients with DPU were significantly more limited in the

distances that they could walk ($P = 0.001$). They also had a diminished ability to run ($P = 0.04$) and a decreased tolerance to prolonged standing ($P = 0.015$). They were more restricted in the type of clothing that they could wear ($P = 0.002$), and they had more difficulty gardening ($P = 0.03$).

The NHP [26] is a validated health-status measure used in general medicine and is divided into two parts. Part I demonstrated an overall restriction in mobility, pain, sleep, energy, social interaction, and emotional reactions in patients with CIU. The pain score was significantly higher in patients with DPU than in patients with CIU ($P = 0.002$). Part II of the NHP demonstrated that patients with DPU had significantly more problems related to their employment ($P = 0.001$) and were more restricted in the pursuit of their interests and hobbies ($P = 0.04$) than were patients with CIU.

Quality of life also was accessed using the Dermatology Life Quality Index (DLQI) [27]. The DLQI looks at six areas of daily life: symptoms and feelings, daily activities, leisure, work and school, personal relationships, and the effects of treatment. Questions refer to the week before filling in the questionnaire. A higher score indicates a greater impairment of quality of life [28]. Patients with DPU had higher scores in all areas than did patients with CIU. Symptoms and feelings, leisure, and work and school were the most affected areas. Half of the patient with DPU had taken time off work during the preceding week because of their condition. Although the patients examined may represent a more severely affected group than other patients with DPU, it is clear that DPU may cause considerable impairment in quality of life.

Treatment of delayed pressure urticaria

There is no single effective drug treatment for DPU, although several reports suggest that effective treatments have been found. Patients should be given explanations about the condition and the limitations of drug treatment. The relationship between pressure-induced swellings should be made clear. Activities that cause the problem may have to be modified or stopped. Patients may be advised to wear loose clothes with elasticized waists and to wear soft, slightly bigger shoes. Many patients find sneakers to be useful. The associated CIU and angioedema may respond to treatment with adequate doses of antihistamine, giving some overall improvement.

It is difficult to assess therapeutic response to drugs in DPU, because the disease is variable, and spontaneous remissions occur. Most investigators have found antihistamines to be completely unhelpful as therapy for DPU lesions [2,3,11]. One report suggests that cetirizine (10 mg every 8 h) is an effective treatment. Investigators have reported a reduction in pressure-induced wheals and a smaller cellular infiltrate in pressure-induced lesions after treatment [29].

Prednisolone may be helpful, usually in dosages of at least 30 mg/d; however, this improvement does not occur in all patients. The side effects produced would mitigate against using this higher dosage over a long period, but it may be used

over short periods for acute severe exacerbations, such as severe swelling of the feet that stops the patient from walking. Some patients have been maintained on smaller doses of prednisolone [2,3,11].

There are conflicting results about the efficacy of new steroidal anti-inflammatory drugs. Aspirin in large dosages (3900 mg/d) suppressed the response to pressure challenge and decreased the pain and tenderness of the deeper lesions, but did not suppress the disease completely [3]. It made it possible to decrease the prednisolone dosage. Ibuprofen and indomethacin had a similar effect [3]. A double-blind, placebo-controlled trial of indomethacin (75 mg/d) did not demonstrate any therapeutic response [1]. In a 7-week study, the treatment plan of nimesulide at 100 mg twice daily for 3 weeks, nimesulide at 100 mg every morning and ketotifen at 2 mg in the evening for 2 weeks, and ketotifen at 2 mg twice daily for 2 weeks was reported to have improved DPU to an equivalent degree compared with prednisolone given at 40 mg/d for 3 weeks, prednisolone given at 30 mg/d for 2 weeks, and prednisolone given at 20 mg/d for 2 weeks. Of the 29 patients completed this study, 70% had negative pressure tests at its completion [30]. Nonsteroidal anti-inflammatory medication can worsen the associated CIU.

Therapies that have their primary effect on neutrophil chemotaxis have been used. Colchicine at 1 mg/d was shown to be ineffective in a double-blind, placebo-controlled trial [5]. Dapsone (50 mg/d) cleared all five patients to whom it was given in an open study [31]. Administering the dosage used in inflammatory bowel disease (4 g/d), sulphasalazine controlled DPU in two patients. The drug was started at 500 mg/d and increased by 500 mg at weekly intervals. Folic acid (1 mg) was administered concurrently to counteract folate malabsorption [32]. The leukotriene antagonist Montelukast controlled a single patient when given in a dosage of 10 mg/d for 1 week [33].

Although the literature suggests that drug treatment for DPU helps, this finding has not generally been the authors' impressions. The search for newer treatments must continue. In patients whose quality of life is impaired severely because of chronic disease, it would be justifiable to perform a therapeutic trial using cyclosporin or intravenous immunoglobulin. The dosages used would be those used in CIU (ie, cyclosporin at 3 mg/kg/d or intravenous immunoglobulin at 0.4 g/kg/d for 5 days, repeated as necessary) [34].

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