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UPDATE ON ADULT DERMATOMYOSITIS

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ABSTRACT
Dermatomyositis is a systemic autoimmune disorder that is an inflammatory disease of the skin, muscles, and blood vessels with characteristic skin eruptions accompanied by proximal limb muscle weakness or atrophy. This condition can affect children and adults. The pathognomonic lesions are the heliotrope sign, Gottron's papule, and Gottron's sign Malignancy can occur in 10%- 20%, with nasopharyngeal cancer being the most common. Etiology and pathogenesis are autoimmune; other triggers include drugs, infection, sun exposure, and malignancy. The latest diagnosis criteria by EULAR/ACR 2017 and Sontheimer et al. can be used as a reference for diagnosis. Comprehensive management of dermatomyositis aims to decrease symptoms, increase quality of life, and early detection of malignancy. Topical therapy options for dermatomyositis are topical calcineurin inhibitors and corticosteroids. In addition, systemic therapy consists of steroid- sparing agents, antimalarials, and corticosteroids. The current treatment of dermatomyositis is the administration of intravenous Immunoglobulin (IVIG) and rituximab.

Keywords: dermatomyositis; diagnosis; risk factor; treatment

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INTRODUCTION

Dermatomyositis is an idiopathic inflammatory myopathy with characteristic cutaneous findings that occur in children and adults. (Bogdanov I, 2018) Idiopathic Inflammatory Myopathies (IIM) are more prevalent in females with peak ages of 5-14 years and 45-64 years. (Lewis M, 2019) The etiology of the cause of dermatomyositis is still not fully known, thought to be due to the production of a number of autoantibodies with varying specificities. (Menaldi S, 2019) The criteria for diagnosis of IIM and cutaneous dermatomyositis have good sensitivity and specificityas. (Leclair V, 2018) Treatment of deermatomyositis on systemic corticosteroid administration is currently is still the gold standard for the initial treatment of classic dermatomyositis. (Gouda W, 2022)

METHOD

This article was prepared using a narrative literature review approach to summarize current knowledge on adult dermatomyositis, including epidemiology, etiology and pathogenesis, clinical manifestations, diagnostic criteria, and management strategies. Relevant literature was identified through searches of PubMed, Google Scholar, and standard dermatology and rheumatology textbooks, such as Fitzpatrick's Dermatology and Andrews' Diseases of the Skin. The EULAR/ACR 2017 classification criteria were also used as key references. Search terms included dermatomyositis, adult dermatomyositis diagnosis, treatment, myositis-specific autoantibodies, and interstitial lung disease. Articles published in English and Indonesian within the last 10–15 years were reviewed. Selected publications were analyzed descriptively, and relevant findings were synthesized to provide a comprehensive overview of adult dermatomyositis.

RESULT

The literature review demonstrates that dermatomyositis (DM) is a rare systemic autoimmune inflammatory disease characterized by distinctive cutaneous manifestations and symmetrical proximal muscle weakness. The reported incidence ranges from 5 to 10 cases per million population per year, with a female predominance and a bimodal age distribution. Clinically, DM is classified into classic dermatomyositis (CDM) and clinically amyopathic dermatomyositis (CADM). Pathognomonic cutaneous findings include heliotrope rash, Gottron's papules, and Gottron's sign. Extracutaneous involvement may affect the lungs, joints, gastrointestinal tract, cardiovascular system, and is frequently associated with malignancy, particularly in adult patients.

Diagnostic evaluation includes assessment of muscle enzymes, electromyography, magnetic resonance imaging (MRI), skin or muscle biopsy, and detection of myositis-specific autoantibodies (MSAs). Autoantibodies such as anti–TIF1-γ, anti–MDA5, anti–NXP2, and anti–Mi-2 are associated with distinct clinical phenotypes, including increased risks of interstitial lung disease and malignancy. The EULAR/ACR 2017 classification criteria provide high sensitivity and specificity for the diagnosis of dermatomyositis. Management strategies include non-pharmacological measures such as photoprotection and malignancy screening, as well as pharmacological therapy. Systemic corticosteroids remain the first-line treatment for muscle involvement, while immunosuppressive agents, intravenous immunoglobulin (IVIG), and biologic therapies such as rituximab are effective in refractory cases.

DISCUSSION

Dermatomyositis is a complex autoimmune disorder with a wide spectrum of clinical presentations. Cutaneous manifestations often precede muscle symptoms and may be the primary feature, particularly in patients with clinically amyopathic dermatomyositis, highlighting the importance of early recognition by clinicians. Advances in the identification of myositis-specific autoantibodies have improved understanding of disease heterogeneity and prognosis. These autoantibodies aid in predicting clinical outcomes, identifying patients at higher risk for malignancy or interstitial lung disease, and guiding individualized management strategies. The introduction of the EULAR/ACR 2017 classification criteria represents a significant improvement over the traditional Bohan and Peter criteria by providing greater diagnostic accuracy and applicability in both adult and juvenile populations. However, clinical judgment remains essential, especially in resource-limited settings.

Treatment of dermatomyositis requires a multidisciplinary approach involving dermatology, rheumatology, pulmonology, and oncology specialists. While systemic corticosteroids remain the cornerstone of initial therapy, increasing evidence supports the use of steroid-sparing agents, IVIG, rituximab, and Janus kinase inhibitors in refractory or severe disease. Early diagnosis, appropriate screening for associated malignancies, and comprehensive long-term management are essential to improve patient outcomes and quality of life in adult dermatomyositis.

Definitions

Dermatomyositis (DM) is a systemic autoimmune disease with a bimodal distribution affecting children and adults, characterized by inflammation and damage to the skin and muscle. (DeWane ME, 2020). Pathognomonic cutaneous findings and gradual, symmetrical proximal muscle weakening are the hallmarks of classic DM (CDM). (Cobos GA, 2020) Patients with clinically amyopathic dermatomyositis (CADM) have the typical cutaneous manifestations of dermatomyositis but do not experience muscular weakness. Clinically, amyopathic or hypomyopathic dermatomyositis is the next classification for amyopathic dermatomyositis. (Qudsiya Z, 2022).

Epidemiology

Age of illness onset for DM shows a bimodal distribution, peaking at 5 to 14 years and 45 to 64 years of age, respectively. Women are 2-3 times as likely than men to develop the condition. estimates of incidence and prevalence are remarkably similar, with incidence rates ranging from 5 to 10 per 1 million per year and prevalence estimates of 10 to 20 per 100,000. In their small cohort, 20% (6 of 29 cases) were clinically amyopathic. Analysis of data from large insurance claims databases in Japan and Taiwan estimated an annual incidence of 10 to 13 and 6 to 10 per million annually, respectively. (Lewis M, 2019).

Etiology and Pathogenesis

Dermatomyositis complex pathogenesis is multifactorial, complex and still not fully understood. It is underlined the significant significance of genetic and environmental variables, such as infections, vitamin D insufficiency, ultraviolet (UV) ray exposure, and medicines. (Marzecka M, 2022). After receiving the BCG, H1N1, tetanus toxoid, influenxa, and hepatitis B vaccines, some people have developed the unusual multi-organ chronic inflammation myopathy known as dermatomyositis (DM). (Rayavarapu S, 2013) Dermatomyositis has been reported following the BNT162b2 (Pfizer-BioNtech®) mRNA Covid–19 vaccination, in the absence of any additional trigger factors. (Gouda W, 2022). The damage phase, the expansion phase, the induction phase, and the susceptibility phase, are assumed to be the ordered stages through which DM develops. (Goldsmith LA, 2011).

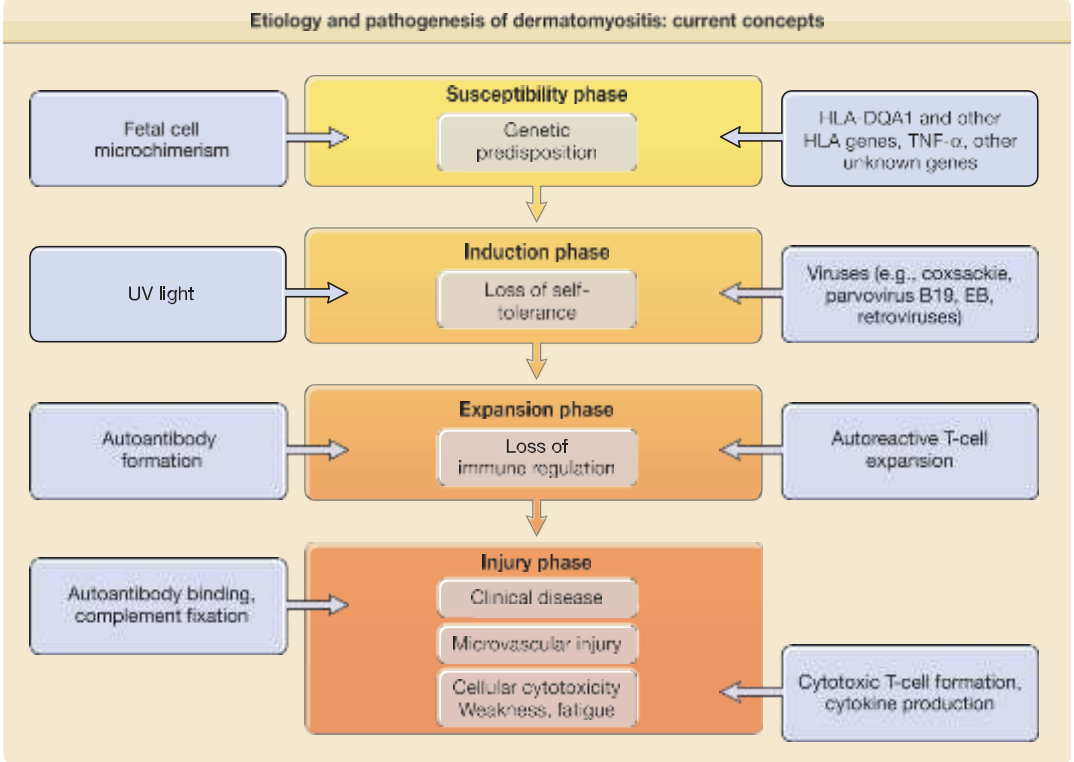


Figure 1. Etiology and pathogenesis of dermatomyositis: current concepts. See text for discussion. EB = Epstein–Barr; TNF-α = tumor necrosis factor-α; UV = ultraviolet. (Goldsmith LA, 2011)

Clinical Findings

Clinical manifestations of dermatomyositis are divided into classic dermatomyositis and clinically amyopathic dermatomyositis. In classic dermatomyositis, patients experience complaints of skin and muscle complaints, while in dermatomyositis clinical amiopathy there are no muscle complaints. (Coboos GA, 2020).

Cutaneous Findings

The pathognomonic lesions cutaneous feature of affected skin in DM is violaceous patches and plaques. Gottron's papules are the violet to pink papules that cover the metacarpal and

interphalangeal joints (Fig. 4-1). Heliotrope sign (Fig. 4-2); Gottron sign is symmetric macular violaceous erythema (Fig. 4-3). (Menaldi S, 2019) The scalp is one of the most commonly involved sites, only with pink to red erythema, with fine white scale (nonscaning alopecia). Trunk involvement is often found on the shoulder, known as the shawl sign, which may extend to the posterior upper arms. (Menaldi S, 2019)



Figure 2. Gottron papules. Light pink, ill-defined papules over the proximal. Deep red erythema, edema of the proximal nailfolds, and dilated nailfold capillaries are evident.



Figure 3. Heliotrope sign. Violaceous to pink erythema and edema on the upper eyelid.



Figure 4. Gottron sign. ill-defined violaceous erythema on the knees is seen, which has follicular accentuation in this patient.

Extracutaneous Findings

Dermatomyositis can involve organs such as lung, joint, digestive, cardiovascular, as well as internal organ malignancies. ILD is the most frequent pulmonary manifestation and cause morbidity and mortality in dermatomyositis patients. (Zhang L, 2016)

Examinations

Supporting examinations for the diagnosis of dermatomyositis are autoantibodies, muscle and skin biopsy, magnetic resonance imaging (MRI), electromyography (EMG), and muscle enzymes. (Hozumi H, 2016)

Target of antibodies	Prevalence in DM	Function of target	Skin	ILD	Cancer	Muscle
TIF1	7-30%	Transcription and RNA metabolism	Classical skin eruption Poikiloderma	↘	↗	→
MDA5	6-26%	RNA helicase for antiviral response	Skin ulcers, palmar papules CADM Mechanic hands	↗	→	↘
NXP2	1.6-60%	Transcriptional regulation	Severe skin eruption, calcinosis Less Gottron's sign	↘	↗	↗
Mi2	4-45%	Transcriptional regulation	Classical skin rash	↘	↘	→
SAE	1-8%	Posttranslational modifications	CADM initially	? ↗ ↘	→	↘
ARS		Incorporate aminoacyl	Mechanic hands	↗	→	↗

Figure 5. Targets, prevalence, and clinical association of myositis-specific antibodies. ILD: interstitial lung disease; TIF: transcription intermediary factor; RNA: ribonucleic acid; MDA: melanoma differentiation-associated gene; CADM: clinical amyopathic dermatomyositis; NXP: nuclear matrix protein; SAE: small ubiquitin-like modifier-activating enzyme; ARS: aminoacyl tRNA synthetase. (Sena P, 2018)

Muscle Examination

1. Muscle Enzymes
   Serum muscle enzymes, such as and alanine aminotransferase [ALT], aspartate aminotransferase [AST], lactate dehydrogenase [LDH], aldolase, and creatine kinase, are fairly sensitive indicators of inflammation in the muscles at the beginning of the development of myositis. (James WD, 2020)
2. Electromyographic (EMG)
   Similar to muscle enzyme abnormalities, early in the disease course, myositis may be detectable on electromyographic studies in 70% to 90% of DM patients have muscular disease, and over time, the ability to identify myositis declines. (Lewis M, 2019)
3. MRI
   The thorough image of the muscle structure provided by MRI enables the identification and differentiation of the different disease processes. (eg, atrophy, calcifications, fibrosis, inflammation,or edema). (Lewis M, 2019)

Pathology

Histopathologic examination showed a picture of squamous epithelial lining layer with the structure and morphology of nuclei within normal limits and basal membrane intact. (Concha JSS, 2019)

Diagnostic Criteria

Dermatomyositis is diagnosed based on the patient's medical history, physical examination, and supporting examination. (Wolstencroft PW, 2018) The latest dermatomyositis diagnosis criteria from The European League Against Rheumatism / American College of Rheumatology (EULAR / ACR) in 2017 reported the sensitivity of dermatomyositis without biopsy is 87% and with biopsy 93%, the specificity without biopsy is 82% and with biopsy 88%. (Bottai M, 2017).

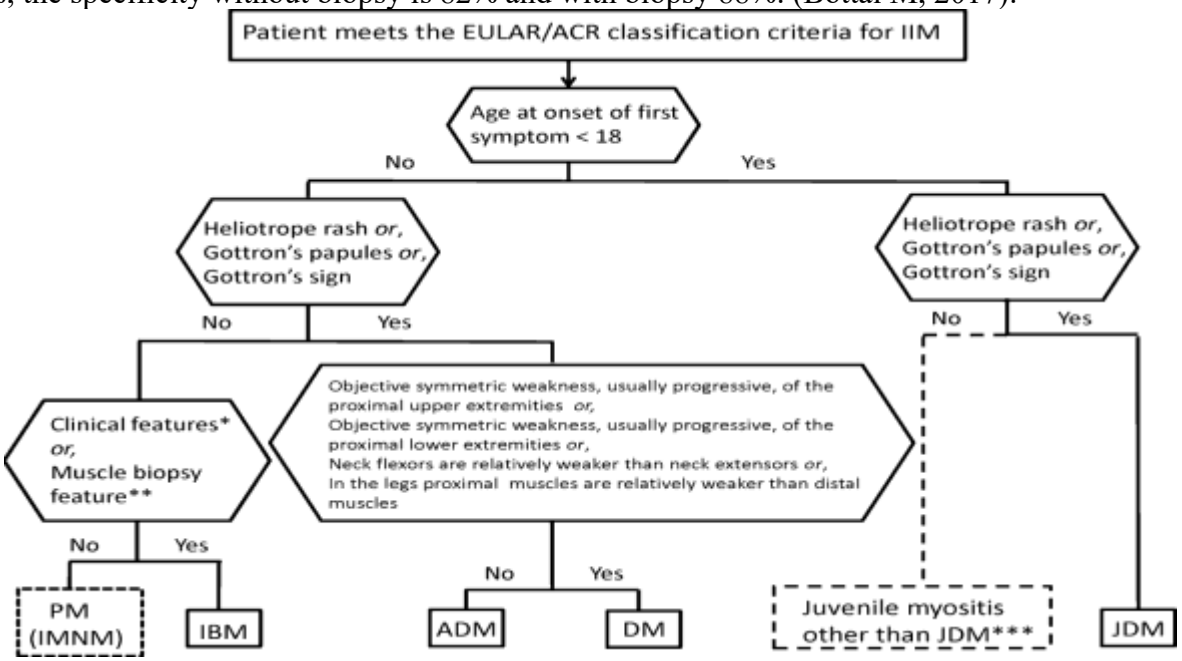


Figure 1. IIM Classification Algorithm based on EULAR/ACR 2017. (Bottai M, 2017)

Sontheimer indicated that in addition to biopsy of at least one site exhibiting histopathologic alterations compatible with DM, a diagnosis of CADM might be made if two major criteria or one major criterion and two minor criteria were present. (Lewis M, 2019).



TABLE-62-1

Sontheimer's Proposed Diagnostic Criteria for Cutaneous Dermatomyositis[1]

Diagnosis of cutaneous dermatomyositis requires:

1. Presence of two major criteria or one major criterion and two minor criteria *and*
2. Skin biopsy changes consistent with cutaneous dermatomyositis

Major Criteria

Heliotrope sign
Gottron papules
Gottron sign

Minor Criteria

Macular violaceous erythema involving (each area counts as one minor criterion)

- Scalp or anterior hairline
- Malar eminences of face, forehead, or chin
- V-area of neck or upper chest (V-neck sign)
- Posterior neck or posterior shoulders (shawl sign)
- Extensor surfaces of arms or forearms
- Linear streaking overlying extensor tendons of dorsal hands
- Periungal skin
- Lateral thighs or hips (holster sign)
- Medial malleoli

Nailfold capillary telangiectasia, hemorrhage-infarct
Poikiloderma
Mechanic's hands
Cutaneous calcinosis
Cutaneous ulcers
Pruritus

Table 2 . Sontheimer's Proposed Diagnostic Criteria for Cutaneous Dermatomyositis. (Lewis M, 2019)

Treatment

Every patient with DM needs a special therapeutic strategy that considers the seriousness of the cutaneous illness, the possibility of concomitant muscular disease, systemic involvement, other comorbidities, such hidden cancer, and the overall effect of the disease on a patient's quality of life. Treatment for skin conditions specifically aims to control cutaneous disease using the safest possible therapy combination. (Lewis M, 2019)

on – Pharmacotherapy

Photoprotection

It is significant to remember that dermatomyositis initially manifests as a skin condition. Therefore, it is crucial to start using skin preventive measures, such as wearing appropriate garments and using sunscreen with a minimum SPF of 15. (Lewis M, 2019)

Cancer Screening

A thorough evaluation is warranted following an initial diagnosis of DM because 10% to 20% of people with DM have a cancer that is related to it. (Lewis M, 2019)

Monitoring of Extracutaneous

If the illness worsens, extracutaneous symptoms in the heart, lungs, or muscles are observed. Each time a patient comes in, the neck flexors, deltoids, and quadriceps are tested for strength. (Lewis M, 2019).

Pharmacotherapy

Photoprotection, topical steroids, hydroxychloroquine (chloroquine), and quinacrine are the first-line treatments for skin complaints; systemic corticosteroids are utilized in the case of muscle symptoms. Methotrexate, mycophenolate mofetil, intravenous immune globuline (IVIG), and azathioprine are second-line treatments for skin and muscle complaints. Tofasitnib, leflunomide, dapsone, thalidomide, cyclosporine, and cyclophosphamide are third-line treatments, however rituksimab is utilized if muscle complaints are present. (Lewis M, 2019)

Topical Therapy: Corticosteroids and Calcineurin Inhibitors

By lowering erythema, scale, and pruritus and acting as an adjuvant to systemic medications, topical corticosteroids may have a temporary effect on DM skin disease. Unless in the mildest situations, they are doubtful to completely control skin complaints. Class I or II topical steroid creams or ointments are used on the thick skin of the elbows, knees, and hyperkeratosis on the hand. Topical calcineurin inhibitors such as pimecrolimus 1% cream or tacrolimus 0.1% ointment may have moderate efficiency in some cases of continuos DM. (Lewis M, 2019).

Systemic Therapy

Systemic medicines are needed for the majority of cutaneous patients with diabetes. The selection of a systemic medication should be customized for every individual and is based on the existence of other DM symptoms, primarily myositis or lung disease.

1. Systemic Corticosteroids
   The fundamental component of early therapy for those with DM and active muscle illness has continued to be systemically administered corticosteroids. Prednisone is normally begin at doses of 0.5–1 mg/kg/day, however intravenous methylprednisolone may be required if the muscle engagement is severe or potentially fatal. (Cobos GA, 2020) Within four weeks of starting treatment, steroid therapy should relieve symptoms; then, over the course of ten weeks, the steroid dose should be gradually decreased to 1 mg/kg on a daily basis. (Okogbaa J, 2019)
2. Antimalarials
   Antimalarials are typically regarded as first-line agents for skin disease. The addition of quinacrine to chloroquine or hydroxychloroquine may be more effective than a single agent. (DeWane ME, 2020) Dosage for adults is 200 mg twice daily by mouth, and the dosage for children is 2 to 5 mg/kg/day by mouth. (Okogbaa J, 2019)
3. Steroid-sparing agent
   a. Methotrexate
      Methotrexate is frequently administered in doses of 25 mg once a week along with a 1 mg daily folic acid supplement. In the organizations where the authors work, MTX is administered at a dose of 10 mg/week, with monitoring laboratory tests being drawn after two weeks. (Cobos GA, 2020)
   b. Mycophenolate Mofetil (MMF)
      The dosage is then raised in several patients to 1.5 g twice daily after being titrated up to 1 g twice daily. The therapeutic effects of MMF are not apparent until 6–12 weeks into treatment. (Okogbaa J, 2019)
   c. Azathioprine
      Once the steroid is decreased to 15 mg/day, the dosage of azathioprine is 2 to 3 mg/kg/day tapered to 1 mg/kg/day. Lower dosage at 25 mg intervals every month; the daily maintenance dose is 50 g. (Okogbaa J, 2019).
   d. Calcineurin Inhibitor
      Rarely used to treat muscle or skin inflammation, but there is improvement in creatine kinase and increased muscle strength in patients with dermatomyositis and polymyositis. The dosage of cyclosporine used with corticosteroids is 3-3.5 mg/kg/day. (Griger Z, 2017)
4. Intravenous Immunoglobulin
   The plasma from several donors is pooled to create intravenous immunoglobulin (IVIg). Intravenous immunoglobulin is efective for both refractory cutaneous disease and myositis. (Cobos GA, 2020) The dosage of intravenous immunoglobulin is 2 g/kg in divided doses once per month for 3 months. (Cobos GA, 2020)
5. Rituximab
   Rituximab is a chimeric monoclonal antibody that specifically targets the B cell CD20 antigen protein. It is a successful cure for the extracutaneous signs and symptoms of diabetes mellitus, especially DM-related interstitial lung disease and refractory myositis. (Cobos GA, 2020).

6. Janus Kinase Inhibitor (JAK)
   An intracellular signaling pathway called Janus kinase (JAK)-signal transmitter and accelerator of transcription (STAT) is used by cytokines, such as interferons and interlukins, as well as other molecules to transmit messages from the outer layer of the cell to the nucleus. (Cobos GA, 2020)

CONCLUSION

A uncommon idiopathic inflammatory illness with a variety of manifestations that might have varying levels of systemically and cutaneous manifestations is called dermatomyositis. The cause of DM is still a mystery. Many environmental and genetic variables may have an impact on how DM develops. the most common cause of drug-induced dermatomyositis is hydroxyurea. Dermatomyositis has been reported following the BNT162b2 mRNA Covid – 19 vaccination. To enhance studies in IIM, verified categorization standards are required as the Bohan and Peter criteria are now out of date. Thus, the new EULAR/ACR IIM classification standards are unquestionably better and represent a significant advancement in the field. As demonstrated in this instance, treating dermatomyositis calls for a multidisciplinary approach. The chance of survival and result are improved when individuals are treated well with both pharmaceutical and non-pharmacological methods.

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