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A REVIEW ON AUTOIMMUNE DISORDERS

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Abstract:
Our aim is to review different autoimmune disorders covering information about more than 10 autoimmune disorders along with their introduction, cause, diagnostic tests and treatment options. Here the treatment options include the name of the medications and their efficacy, availability. It also includes the necessary surgical options. This review clearly gives the necessary inputs regarding most common autoimmune disorders like psoriasis, rheumatoid arthritis, pernicious anaemia, myasthenia gravis, rheumatic fever etc along with rare autoimmune disorders like multiple sclerosis, Systemic lupus erythematosus. In this review, we particularly focussed on the symptoms of autoimmune disorders in order to identify the disease in starting stage itself, we focussed on the diagnostic tests to guide that which test gives the accurate results and we also focussed on the treatment options to give the information which medications are efficient and where they are available. This review also gives clear information about surgical options available. As the autoimmune disorders affecting mostly children and various autoimmune disorders taking the lives of children, it is important to save the lives of our children; hence we thought it is important to focus on this issue.

Keywords: Auto-immune disorders, Psoriasis, Rheumatoid arthritis, multiple sclerosis, systemic lupus erythematosus.

Introduction:

In this review, we discussed about the following autoimmune disorders along with their symptoms, diagnostic tests, treatment etc

Rheumatoid arthritis

Rheumatoid arthritis (RA) is the disease affecting the lining of synovial joints which may lead to progressive disability. It is seen more frequently in female when compared to male. It is a chronic autoimmune disease observed more in elderly people.

Symptoms include swelling, redness, arthralgia. This disease limits the range of movement. Rheumatoid arthritis may lead to joint destruction, functional disability, socioeconomic burdens, pre-mature death. If the disease is so severe, it leads to keratitis, vasculitis, and rheumatoid nodules.

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Diagnosing the disease at the early stage itself i.e., within the 12 weeks after the appearance of first symptoms can serve as the improvement index to get most desirable outcomes from the medication used; hence there will be no functional obstructions for the RA patient. Early diagnosis gives the best results not only by providing relief from the disease but also reduces the cost expenses of the treatment.[1-3] Anyways, early diagnosis mostly relies on the patient's medical history, physical examination and blood tests, imaging tests, hence it still remains challenging.

There is no complete cure for RA at present. The treatment of RA is based on the aim to achieve a low disease activity state. Low disease activity state is measured by using the scales like Disease activity score, clinical disease assessment index etc[4]. Based on the above scale scores, the physicians adjust the treatment regimen. Nonsteroidal anti-inflammatory drugs gives relief from pain and stiffness of joints; these drugs can provide symptomatic relief but unable to minimize the progression of the disease. From the past 25 years, usage of Disease-modifying anti-rheumatic drugs (DMARDs) started gaining importance due to their ability to decrease the progression of the disease.[5] DMARDs include drugs like minocycline, auranofin, azathioprine, cyclosporine etc. DMARDs also include TNF-inhibitors like Amjevita, Renflexis, Erelzi, Cyltezo, Imradl, anti-CD20 antibody like Truximma, Rixathon RANKL antibody like Pralia, IL-6 receptor antibody like Kevzara and JAK inhibitor like Olumiant. However, despite of availability of many drugs, complete relief from the disease is not achieved and there is a need of new pharmacological innovations for the treatment of rheumatoid arthritis.

Inflammatory bowel disease

Inflammatory bowel disease is a combination of Crohn's disease and ulcerative colitis. Crohn's disease involves the inflammation of the lining of digestive tract and ulcerative colitis is the inflammation of any part of digestive tract.

It is observed that the mutations on NOD2 gene are responsible for inflammatory bowel disease. However, the risk of IBD increases with the consumption of protein from milk and meat and smoking and consumption of tobacco raises the risk of crohn's disease. This disease is mostly seen in adults; however cases are increasing in children[6].

Symptoms include diarrhoea, fatigue, and abdominal pain, blood in faeces, reduced appetite, and unintended weight loss[7]

Treatment involves the usage of anti-inflammatory drugs like 5-amino salicylic acid. Other drugs like Infliximab can be used which are easily available. Severe cases of IBD can be treated by using immunomodulatory drugs like thalidomide[8].Treating IBD often involves use of medications that can diminish the symptoms and decrease the inflammation in the colon lining. A group of anti-inflammatory drugs including 5-aminosalicylic acid is commonly used to treat IBD[9]. Incase if medicines failed to treat IBD, then colectomy is considered which involves the removal of colon. Due to colectomy, ulcerative colitis can be resolved but crohn's disease can recur after surgery. Anyways, there always will be side effects for chemical compounds, hence the limited usage of drugs should be considered. Current investigation is aiming at the development of delivery of anti-inflammatory drugs only to the target areas of intestine.

Grave's disease

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Grave's disease is an autoimmune disorder which leads to the raise in the production of thyroid hormones; the overproduction of thyroid hormones is termed as hyperthyroidism. Grave's disease is the leading cause of hyperthyroidism. This disease mainly targets thyroid gland. Along with thyroid gland, it also affects other organs like eyes and skin[10].

This disease is known to be caused by thyroid stimulating antibody (TSAb) or Thyroid stimulating Immunoglobulin (TSI). It is identified that factors like excess iodine, post delivery changes, emotional stress and smoke can trigger the immune responses on susceptible gene leading to Grave's disease[11].

Grave's disease can result in Grave's orbitopathy which is the inflammation of extraocular muscles and proliferation of cells of adipose and connective tissue in eyes. It occurs due to the effect of thyroid stimulating antibodies. Other manifestations of Grave's disease include sweating, tremors, lid lag, Pretibal myxedema and thyroid acropachy etc[12].

Diagnosis of Grave's disease always starts with careful study of family medical history[13,14]. Physical examination should be done. Thyroid stimulating hormone (TSH) test is the initial test which helps to diagnose Grave's disease**.**

Family history of Grave's disease, presence of orbitopathy easily differentiates the Grave's disease from other types of hyperthyroidism. Measurement of TSH receptor antibody test also serves as validated tests for Grave's disease[15]. Another diagnostic test is Radioactive uptake scan with I-23 or I-131. Thyroid ultrasonogram with Doppler, T3/T4 ratio also serves as diagnostic tests for Graves's disease.

Treatment mainly concentrates on the control and reduction of production of thyroid hormones include usage of anti-thyroid drugs like carbimazole available in India. Carbimazole rapidly converts to Methimazole immediately in the body. Methimazole and Propylthiouracil are available in USA. Methimazole is the better choice as it is having less side effects but the problem is it is a teratogenic drug, hence it must be used from second trimester in pregnant women. Propylthiouracil can be used in pregnant women[16,17,18].

One should go for the thyroid function tests periodically (for every 4 to 6 weeks) while using anti-thyroid drugs for dose adjustment of drugs. If the pharmacological treatment fails, then the patient can go for thyroidectomy or RAI therapy.

Thyroidectomy can be done in patients having a very large goiter Thyroidectomy is preferred for patients with very large goiter, thyroid nodules and neck compressive symptoms. Post operative follow up should be done using levothyroxine in a dose of 1.6mg per kg body weight.[19,20].

RAI (Radioactive iodine) therapy can be done in non-pregnant adult cases. Patients who are unable to undergo surgery due to comorbid conditions and are contraindicated for anti-thyroid drugs can go for RAI therapy. Patient must take pregnancy test before undergoing RAI therapy.

Agents like sodium ipodate and iopanoic acid can also be used as these drugs can inhibit the conversion of T4 to T3. Iodide drops can also be used for the treatment of mild hyperthyroidism. Rituximab can induce remission in patients having Grave's disease but it is costly medicine[21].

Myasthenia gravis

Myasthenia gravis is an autoimmune disorder characterized by muscle weakness where the condition worsens with exertion, and improves with rest. At starting stage of the disease, only extrinsic ocular muscles gets affected, up on progression of the disease, other muscles like bulbar and muscles of limb also gets affected which is termed as generalized myasthenia gravis[22].

The cause of the disease is particularly unknown, but the role of circulating antibodies directed against the nicotinic acetylcholine receptor in its pathogenesis is well established. During the past decade, significant progress has been made by giving vast information about the disease, leading to evolution of new treatment modalities and a significant reduction in

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morbidity and mortality. It is evident that MG is caused by antibodies against the acetylcholine receptor (AChR), which produce a compromise in the end-plate potential. A muscle-specific kinase has been recently found to be an antigenic target in MG patients without antibodies against the AChR[23].
Most cases are caused by immunoglobulin (Ig)G1 and IgG3 antibodies to the acetylcholine receptor (AChR). They produce complement-mediated damage and increase the rate of AChR turnover, both mechanisms causing loss of AChR from the postsynaptic membrane. The thymus gland is known to be involved in many patients, and there are experimental and genetic approaches to understand the failure of immune tolerance to the AChR.
Diagnostic tests involve AChR antibodies, MuSK antibodies, and CT/MR of anterior mediastinum for thymoma or thymic hyperplasia. Neurophysiological examination with repetitive nerve stimulation and jitter measurements are important in establishing the initial diagnosis, especially in patients without detectable antibodies [24].

The administration of acetylcholinesterase inhibitors (AChE) improves muscular weakness for several hours, but does not affect the course of the disease. At the beginning of corticosteroid therapy there is the risk of deterioration. Long-term therapy with corticosteroid should be avoided. With the use of immunosuppressive drugs, such as azathioprine, an effect on the myasthenic symptoms starts after several months of therapy. Plasma exchange or the intravenous immunoglobulins (IVIg) are used for myasthenic crisis. Both improve myasthenic weakness for just a few weeks. Thymectomy might influence beneficially the long-term course of non-paraneoplastic myasthenia gravis
Many patients complain of pain because of poor posture caused by the myasthenic weakness. Analgesic medication and orthopaedic therapy, including physiotherapy, may alleviate these frequent complaints. The risk of sleep apnoea syndrome is increased and there is a high prevalence of sleep disturbance among MG patients **[24]**. Sleep-related complaints may help to identify subjects at risk for abnormal breathing during sleep, even when daytime functional activity is judged normal.

Rheumatic Fever

Rheumatic fever (RF) is a multi-system inflammatory disease which occurs as a complication of GroupA Streptococcal infection. It is a non-contagious disease marked by inflammation and pain in the joints. RF may lead to chronic rheumatic heart disease which becomes the major health issue in developing countries.

Clinical manifestations include fever, malaise, pallor, arthritis (involving Pain in the knees, ankles, wrists and elbows)[25,26] , carditis, congestive heart failure, arrhythmias, valvular lesions, swollen cusps, papillary muscle dysfunction etc, uncontrollable body movements called chorea etc.

There are no particular diagnostic tests available for the diagnosis of RF. The diagnosis of RF is based on the revised Jones criteria. According to this criteria, diagnosis is based on the clinical manifestations of the disease, if the person is having 2 or more clinical manifestations of RF and that patient has a medical record of streptococcal infection, then the person is conformed of RF.[27,28] As the particular diagnostic test is not available, differential diagnosis should be done; the diagnosis of Juvenile rheumatoid arthritis and other diseases of

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connective tissue should be done. Echocardiogram and positive culture tests can be done to diagnose infective endocarditis.[29,30]

Treatment at basic step should aim at inhibiting streptococcal infection. Treatment with antibiotics is considered as the first line treatment for the treatment of RF and also antibiotics prevents recurring of RF[34]. Salicylates in a starting dose of 80 – 100mg/kg/day for 3-4 weeks. Naproxen (10-15mg/kg/day, bid) is an alternative drug. 25 Corticosteroids like prednisone at a dose of 2mg/kg/day (maximum, 60mg/day) for two weeks and after that, the dose is gradually tapered, reducing 20 to 25% of the previous dose every week found useful for the treatment of carditis. For severe carditis, combination ofsalicylates and methylprednisolone can be used. Diuretics and vasodilators may be used in patients with more severe haemodynamic decompensation. Digoxin should be used with caution because of the risk of toxicity in the presence of active myocarditis. Haloperidol (initial dose of 0.5 to 1mg/kg/day, maximum, 5mg/day)[31] or valproic acid (15-20 mg/kg/day)[32,33] are useful in the treatment of chorea.

Psoriasis:

Psoriasis is a skin disease that results in red, itchy scaly patches, most commonly on the knees, elbows, trunk and scalp. Psoriasis is a lifetime inflammatory disease leading to complications like psoriatic arthropathy, psychological, cardiovascular and hepatic diseases. In the year 2014, WHO recognized this disease as a serious non-communicable and also revealed the distress occurring due to misdiagnosis, inadequate treatment and stigmatisation of this disease.[35]

The cause of psoriasis is still unclear but it is known that epidermal keratinocytes and T cells play a vital role in the development of psoriasis. Certain genes like human leukocyte antigens (Cw6, B13, B17) are known to be associated with psoriasis.[35] PSORS1 locus on chromosome 6p21 plays the greatest role in determining a patient's susceptibility of developing psoriasis. Certain environmental factors like sunburn, HIV infection, streptococcal infections, emotional stress and alcohol consumption also triggers psoriasis.[36]

Psoriasis manifests as plaques, flexurals, guttate, pustular or erythrodermic psoriasis. The most common form is plaque psoriasis which is indicated as pink plaques with silvery-white scale, typically in a symmetrical distribution and affecting especially elbows and knees, trunk and scalp. Bleeding points may be noted where scales have been removed which is regarded as Auspitz sign.[37] Flexural psoriasis presents without much scaling and may affect the axillae, sub-mammary and genital areas. Guttate psoriasis is manifested as papules mainly on trunk and limbs. In rare cases of severe uncontrolled disease, psoriasis causes a widespread erythematous rash (erythroderma) having potential complications including hypothermia, risk of infection, acute kidney injury and high-output cardiac failure. Nail pitting, onycholysis, oil spots (discoloration of the nail bed), dystrophy and subungual hyperkeratosis are seen in 50% of patients.[39]

Psoriasis is assessed by the extent of skin involvement (body surface area (BSA)) and the severity of erythema and scaling. Physician global assessment scale is commonly used along with Dermatology Life Quality Index (DLQI). Recognition of multimorbidities may influence psoriasis treatment choice.[38]

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Therapeutic options for psoriasis are topical therapy, phototherapy or systemic treatment. Topical treatment with vitamin D analogues (calcipotriol) or corticosteroids serves as first line therapy. Dithranol and tar preparations are less used as they irritate the skin. [40]

Second-line therapy includes phototherapy using narrowband ultraviolet B radiation (NB-UVB) and psoralen with ultraviolet A radiation (PUVA) and other agents like methotrexate, ciclosporin and acitretin.[41] methotrexate is the useful driug but it causes bone marrow suppression.

Multiple biological therapies such as adalimumab, etanercept, infliximab and certolizumab and IL-12/23p40 (ustekinumab), IL-23p19 (rizankizumab, guselkumab and tildrakizumab), IL-17 (ixekizumab and secukinumab), and IL-17 receptor (brodalumab) inhibitors can be used[42,43] .Oral small molecule inhibitors including apremilast (phosphodiesterase 4 inhibitor) and dimethyl fumarate are licensed for use in moderate–severe psoriasis, and trials are ongoing for small molecules blocking tyrosine kinase 2 in the Janus kinase (JAK) – signal transducer and activator of transcription proteins (STAT) pathway.[43]

Systemic lupus erythematosus (SLE)

Systemic lupus erythematosus (SLE) is an autoimmune disease where the person's immune system affects his/her body's own tissues leading to immense inflammation and damage to the organs. It is a multi-system disease affecting various organs like brain, lungs, skin, kidneys, blood vessels, joints etc. In this disease, the patient develops certain antibodies which are regarded as self antigens that leads to chronic inflammatory autoimmune disease.[44]

SLE is known to occur due to mutations on more than 100 genes in humans. Out of them, major histocompatibility complex genes, several human leucocyte antigen genes are considered as the main factor for the development of SLE. It is observed that although genetic factors are responsible for the development of SLE, the environmental factors like sunlight, certain drugs, diet, and infections activate SLE. It is also evident that androgens inhibit and estrogens enhance the expression of autoimmunity.[44]

Clinical manifestations of SLE include multiple system disorders like Lupus nephritis, neuropsychiatric SLE (involving cognitive disorder, confusion states, psychosis etc), musculoskeletal disorders like Synovitis, Jaccoud arthropathy etc and other manifestations like thrombocytopenia, pulmonary arterial hypertension.[44]

Antinuclear antibody titer is the marker test for the diagnosis of SLE. Other tests are antibody to double stranded DNA antigen test and antibody to sm nuclear antigen test.

Anti-malarials like hydroxychloroquine and chloroquine are found useful in the treatment of SLE.[45] In patients of SLE without major complications, anti-malarials are enough Glucocorticoids like methylprednisolone, mometasone furoate can topically applied on the scalp, palms and soles for the treatment of lesions. To spare the glucocorticoids, immunosuppressants drugs like azathioprine, mycophenolate mofetil, methotrexate can be used. If serious orgen infections are there, cyclophosphamide or glucocorticoids are indicated. Azathioprine and cyclosporine can be used as an alternative to glucocorticoids.

Weight control and regular exercises are considered as the important aspects to reduce the effect of SLE. Low dose aspirin, vit-D, calcium, statins and blood pressure drugs are found useful.[46] Immune-compromised patients should be vaccinated especially against influenza and pneumococci.

Prophylactic measures comprise ultraviolet (UV) light protection, abstinence from smoking, and avoidance of medications known to trigger SLE. Consistent light protection includes

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wearing sun protective clothing, keeping the head covered, and using sun blockers with chemical and/or physical UV-A/UV-B filters (SPF 50+).

Pernicious Anemia

Pernicious anaemia is an anaemia occurs due to deficiency of vitamin B12 (cobalamin). It occurs due to lack of intrinsic factor as the intrinsic factor is required to bind to cobalamin and enhances the absorption of cobalamin at terminal ileum. Gastric autoantibodies against both IF and parietal cells are seen in pernicious anaemia patients, hence this disease is regarded as autoimmune disorder.

Autoimmune gastritis is characterized by the destruction of gastric parietal cells and the resulting lack of the glycoprotein intrinsic factor secreted by these cells. The antibodies identified with autoimmunity are intrinsic factor antibodies (IFA) and parietal cell antibodies (PCA). Parietal cell antibodies work against the parietal cell proton pump ATPase. The primary targets of parietal cell antibodies are the alpha and beta proton pump subunits. Research has determined that parietal cell antibodies are immunoglobulins from the M, G, and A isotypes, which operate against both subunits.[47]

Symptoms may include fatigue, pallor, paresthesia, incontinence, psychosis, and generalized weakness, decreased mental abilities, bloating, gastric discomfort, painful red tongue, diarrhoea, jaundice etc.[48]

Schilling urinary excretion test can be done for the diagnosis of pernicious anaemia. Homocysteine test, Methylmalonic Acid test, serum anti-IF antibodies test, presence of lesions of autoimmune fundic gastritis especially in the absence of *H. pylori* (in collected samples).

Treatment includes intramuscular injection of 1000 mcg B12 every day or every other day during the first week of treatment and injections every week, subsequently followed by monthly injections. Oral dose of 1000 to 2000 mcg/day as an alternative to intramuscular injection is also found effective. Oral dosage is recommended only for patients unable to take IM injections, sublingual and intranasal formulations of B12 are also available. [49]

Periodic monitorings are required as the erythrocyte count increases within first 3 days of therapy. Decrease of biochemical markers such as MMA and plasma homocysteine levels have been observed in the first five days of treatment. A clinical interview must be conducted every year to monitor for new symptoms like epigastric pain, dysphagia, iron deficiency, and/or others that can require gastroscopic investigation.

Type 1 diabetes

The disorder type-1 Diabetes is a disorder where the pancreas failed to produce adequate amount of insulin or the pancreas cannot produce insulin at all. It is also known as Insulin dependent diabetes or juvenile diabetes.

It occurs due to the destruction of pancreatic β cells which produces insulin.[50,51] Symptoms include polyphagia, polydipsia and polyuria.[52]

Fasting blood glucose tests and rapid blood glucose tests are the marker tests for the diagnosis of Diabetes mellitus. However, in the year 2009, the American Diabetes Association formulated the guidelines which includes glycated haemoglobin(HbA_{1C}); glycated haemoglobin test reports the average blood glucose concentrations over 3 months.[53]

Treatment involves the administration of insulin exogenously for lifetime which is known as insulin replacement therapy. Once the diagnosis of diabetes is made, an important goal of

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therapy is to maintain the average glucose as near the normal range as possible without causing unacceptable amounts of hypoglycemia. The goal for most patients with diabetes is to maintain an HbA1c < 7.0% (estimated average glucose of < 154 mg/dL) if that can be achieved without hypoglycemia.The HbA1c is typically checked at least twice a year to confirm that the goals of therapy are being met, or more frequently if the patient is not meeting goals of therapy and the management plan is being actively adjusted. Although the medical team of care providers can provide guidance on the insulin regimen, it is usually up to the patient (or in some cases a parent or caretaker) to administer the regimen, which can be complicated and time-consuming.[56].Patients must use an array of point-of-care tests to get the information they need to make day-to-day decisions about their self-care.

Multiple sclerosis

Multiple sclerosis (MS) is a chronic autoimmune neurological disease affecting the myelinated axons in the CNS, causing the destruction of myelin sheath and the axons to little extent.It is a potentially disabling disease of the brain and spinal cord (central nervous system). Initially, the patients of multiple sclerosis suffer from reversible episodes of neurological problems and leads to irreversible deterioration up on progression of disease.[54]

The cause of the multiple sclerosis is still unknown, the main genetic risk associated with MS resides in HLA-DRB1*15 and/or other loci in strong linkage disequilibrium with this allele. Genome-wide association studies have identified more than 150 single nucleotide polymorphisms associated with MS susceptibility. It is identified that combination of genetic susceptibility and other factors like infections, metabolism etc causes multiple sclerosis.
Symptoms include Vision problems, Tingling and numbness, Pain and spasms, Fatigue and weakness, Balance problems and dizziness, Bladder and bowel dysfunction, Sexual dysfunction, Cognitive problems, More than 30% of MS patients have moderate-to-severe legs spasticity. A common manifestation of MS is unilateral numbness affecting one leg that spreads to involve the other leg and rises to the pelvis, abdomen, or thorax etc. Trigeminal neuralgia may occur.

Evidence of damage in atleast two areas of brain and spinal cord serves as a diagnostic tool for multiple sclerosis. Imaging techniques like MRI, CT scan etc, spinal tapping for the examination of oligoclonal antibodies and autopsy. [55]A standard baseline profile should include anti-nuclear antibody, vitamin B12 and thyroid function. Syphilis and human immunodeficiency virus 1 serology are recommended.

Treatment of various symptoms, complications and acute relapses associated with Multiple Sclerosis is crucial. MS includes treatment of acute relapses, disease-modifying drugs, and treatment of various symptoms and complications associated with MS. The optic neuritis treatment trial (OPTT) is pivotal in establishing the standard for treatment. For mono ocular optic neuritis High dose intravenous (IV) methyl prednisolone in a dose of 1 g/day for 3 days followed by a short course of oral steroids in tapering dose was shown to be significant in hastening the rate of recovery. IV methyl prednisolone in doses of 1 g/day for 3–5 days is mostly used by neurologists.[77] Whenever the patients do not respond well to the initial 3–5 days of parenteral methyl prednisolone therapy, the course may be repeated once again. The relapsing forms of Multiple Sclerosis can be treated with the disease-modifying drugs (DMDs) and these drugs have multiple benefits such as decreasing the frequency and severity of relapses or preventing relapses, lowering the risk of development of disability or provide a sustained improvement; also improve quality of life; and minimize the MRI lesion burden

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and reduce the development of CNS atrophy. The drugs that are currently available in India are interferon-beta (IFN-β), glatiramir acetate, mitoxantrone, and the very recently launched Natalizumab (Tysabri). The strength of the individual doses and the injection routes differ for Avonex (prepackaged as a 30 mg dose, once a week IM route) and Rebif (22 mg and 44 mg, 3 times/week, SC route) which are forms of interferon β-1a. Other drugs that can be used are Glatiramer acetate (Copaxone), mitoxanthrone, natalizumab.

Conclusion:

In this review, we have discussed about various autoimmune disorders along with their etiology, symptoms, diagnostic tests and treatment which helps the medical aspirants to get a brief knowledge on particular autoimmune disorders. This review also provides basic knowledge to common people about certain autoimmune disorders.

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