LEADING ARTICLE

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Sarcoidosis in India : Not so Rare!

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Abstract

Sarcoidosis is a systemic inflammatory disease of unknown aetiology. In India, sarcoidosis is an under-diagnosed disease. All ethnic groups in the Indian sub-continent are affected by sarcoidosis. Most of the Indian patients with sarcoidosis are males, and a majority of them present in their fourth or fifth decade of life. Many patients with sarcoidosis are asymptomatic with incidental findings on the chest radiograph. Depending on the organ systems involved, patients with sarcoidosis present themselves to various specialities, with pulmonary involvement being the commonest. The diagnosis is based on a compatible clinical and/or radiological picture, histopathological evidence of non-caseating granulomas in tissue biopsy specimens, and exclusion of other diseases that can produce similar clinical or histopathological appearances. The disease runs a benign course with spontaneous remission of the activity. Only a minority of the patients develop complicated disease though some degree of residual pulmonary function abnormality persists. A high index of clinical suspicion and focussed attempts to secure histopathological confirmation of the diagnosis early in the illness are required to ascertain the diagnosis. For asymptomatic patients with pulmonary sarcoidosis, no therapy is required. In symptomatic patients, or those with complications, corticosteroids remain the mainstay of treatment and sometimes steroid-sparing alternative treatments may be beneficial. Treatment should be tailored to suit the needs of the individual patient under close clinical monitoring.

Key words : Sarcoidosis, India.

Introduction

Sarcoidosis is an intriguing multisystem disorder of unknown aetiology that occurs worldwide and affects people of all ages and races[1,2]. Patients with this intriguing disease may often be asymptomatic with incidental findings of mediastinal lymphadenopathy on the chest radiographs. Depending on the organ systems involved, patients with sarcoidosis present themselves to various specialities with the lungs being the most commonly affected sites[2-4]. Sarcoidosis has been traditionally thought to be uncommon in tropical countries like ours and remained unreported from India till the late 1950s. Most sarcoidologists in India feel that remarkable resemblance to tuberculosis and lack of facilities to perform invasive diagnostic procedures and lack of awareness among physicians and pathologists regarding the disease have all been the reasons for under-reporting of the disease. However, several case reports and a few large series[3-8] have indeed been reported from India, indicating that the disease is not so rare as is presumed. In this review, we have attempted to provide a critical overview regarding various aspects of sarcoidosis as has been reported in the Indian context.

Burden of sarcoidosis

Prevalence of sarcoidosis is higher in the Scandinavian countries. In other areas of the world, epidemiological observations have been variable. The true burden of sarcoidosis in India is not clearly known as reliable epidemiological data are not available. It has been estimated that sarcoidosis constituted 10 to 12 cases/ 1,000 new registrations annually at a Respiratory Unit at Kolkata and 61.2/100,000 new cases seen at the Vallabhbhai Patel Chest Institute (VPCI), Delhi[9]. We feel that these observations are likely to be underestimates and the actual burden of sarcoidosis is likely to be more than what is reflected in these figures because many cases remain undiagnosed or unreported.

Over the last two decades, the patients with sarcoidosis seen by us at the All India Institute of Medical Sciences (AIIMS), New Delhi, one of the largest tertiary care referral centre were from almost every state in the country[3,4,10]. This observation and review of published literature from India suggests that sarcoidosis occurs in all ethnic groups and communities in India[5-8,11].

Aetiology

Inspite of the scientific and technological advances, the

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aetiological cause of sarcoidosis has not been unravelled. The cause of sarcoidosis is still obscure, continues to elude us and remains an enigma. It is also not clear whether sarcoidosis is caused by a *single agent*, *several related agents,* or *multiple factors*. Genetic factors have been thought to play a role in the pathogenesis of sarcoidosis for a long time. It is unlikely that any single gene is involved in the pathogenesis of sarcoidosis. Probably, genetically predisposed hosts are exposed to antigens that trigger an exaggerated immune response and this results in the formation of granulomas. Although no significant association has been reported between sarcoidosis and occurrence of HLA phenotypes, the most consistent allele found associated with sarcoidosis has been HLA-B8 in some of the western studies[12,13]. Recent immunogenetic study results based on the molecular typing of HLA alleles of sarcoidosis patients by the authors' group[14] revealed positive association of DRB1*11, DRB1*14, DQA1*0101/4, and DQB1*0503 class II alleles with the disease. Follow-up data suggested predominant occurrence of DRB1*14 and its linked DQ alleles in patients with insidious onset, advanced disease on chest radiographs, and chronic course with frequent relapses on tapering-off the prednisolone treatment. Furthermore, multivariate logistic regression analysis revealed that the presence of DRB1*11[odds ratio (OR) 9] and DRB1*14 (OR 7), and absence of DRB1*07 (OR 63 and DQB1*0201(OR 3) alleles, were independent predictors of sarcoidosis[14]. Observations on community out-breaks, work-related risk of sarcoidosis for nurses, and a study tracing the case contacts on the Isle of Man[15-17] suggest a person-to-person transmission, or shared exposure to an environmental agent. Transplantation of sarcoidosis by cardiac and bone marrow transplantation[18,19] suggests an infectious aetiology. These issues need further clarification in the studies addressing the microbial aetiology of sarcoidosis. Since many non-infectious agents present in the environment can induce granulomatous reaction, there appears to be some role for exposure to an environmental agent in the causation of granulomatous inflammation in patients with sarcoidosis[20]. Most of the current evidence suggests some role for beryllium[20]. While other agents such as clay soil, pine dust, and talc have been postulated, there is no significant published evidence to support this view. However, the pathogenetic mechanisms involved require further elucidation.

Clinical presentation

Clinical presentation of sarcoidosis as seen in Indian patients is contrasted with the clinical observations from the West in Table I. Sarcoidosis is predominantly a disease of adults, and seldom seems to affect children. While more than 70 per cent of the patients in the West were less than 40 years of age, sarcoidosis in India has a late onset by nearly a decade (Table I). While the disease has a slight female preponderance globally, sarcoidosis is commoner in males in India (Table I). This appears to be true even after giving allowance to the fact that males seek medical care more often than females in our hospitals.

Acute presentation in the form of "Löfgren's syndrome" and "Heerfordt's syndrome" (fever, parotid enlargement, facial palsy, and anterior uveitis) is uncommon in patients from the Indian sub-continent. Familial sarcoidosis has been described with a rate of at least 19% in affected black families and 5% in white families[28]. Familial sarcoidosis has occasionally been reported from India also[3,5,29,30]. In the series reported by us[3], familial involvement was observed in 6 of the 106 biopsy-proved patients with sarcoidosis. Subsequently, we have observed familial sarcoidosis in four more families. Thus, 10 of the 210 patients (5%) seen by us between January 1980 and August 2003 had familial involvement and brother-brother involvement was most frequently encountered.

The constitutional symptoms such as fever, fatigue, malaise, and weight loss have been observed to occur more often in Indian patients than in patients from the West (Table I). Fever-malaise dissociation has been reported to be common in Indian patients with sarcoidosis[3-5,10,11,29].

Pulmonary involvement is, by far the commonest, and has been reported in about 90 per cent of the patients[3-5,10,11]. Patients may complain of cough which may be non-productive, dyspnoea, and chest pain. Haemoptysis and clubbing are rare and chest signs on auscultation may be present in fewer than 20 per cent of patients. Airway hyperactivity has been reported in up to 20 per cent of patients with sarcoidosis[3,4]. Sometimes, reversible airways obstruction and clinical presentation mimicking bronchial asthma with seasonal exacerbations may also be encountered[3,4].

Table I : Clinical profile, laboratory characteristics, radiological findings, and treatment in Indian patients with sarcoidosis with those from the West.

	Sharma SK *et al* (n = 210)*	Other Indian studies (n = 409)†	Western studies (n = 8137)‡
Females	39	43-71	57-61
Under 40 years	34	25-66	70-86
Thoracic involvement	98	61-97	88-99
Ocular involvement	17	08-40	4-27
Erythema nodosum	07	02-20	11-34
Other Skin lesions	35	10-24	–
Parotid enlargement	11	03-15	0.5-6
Neurological involvement	10	01-11	0.3-9
Fever	56	35-54	–
Constitutional symptoms§	42	14-57	–
Arthralgias/arthritis‖	39	18-35	40
Cardiac involvement	07	0-12	03
Peripheral lymphadenopathy	43	19-42	8-27
Hepatomegaly	42	14-42	12
Splenomegaly	17	02-27	0.3-10
Radiologic stage at presentation			
0	01	01-03	4-13
I	34	45-62	58-65
II	57	30-34	22-31
III	08	07-18	7-13
Kveim positive	Not done	45-96	73-84
Mantoux negative	88**	59-97	55-70
Hypercalcaemia	13††	18-40	0.7-18
Hypercalciuria	32††	10-49	2-50

All values expressed as percentages are corrected to the nearest round figure.

** Data appended from "Sharma SK, Mohan A. Sarcoidosis: global scenario and Indian perspective. Indian J Med Res 2002; 116: 221-47" (reference 4);† Cumulative figures from five series reference 5 (n = 125), reference 6 (n = 40), reference 7 (n = 155), reference 8 (n = 60), and reference 21 (n = 29);‡ Cumulative figures from six series reference 23 (n = 818), reference 24 (n = 797), reference 25 (n = 2006), reference 26 (n = 3676), reference 27 (n = 125), reference 28 (n =715);§ Constitutional symptoms included weight loss, fatigue, malaise;‖ Clear distinction between arthralgias and arthritis is not clearly mentioned;** Tested in 184 patients;†† Tested in 148 patients.*

Cutaneous involvement occurs in about 11 to 34 per cent of patients with sarcoidosis (Table I). These include erythema nodosum, lupus pernio, plaques, maculopapular lesions subcutaneous nodules, changes in old scars, alopecia, and hypo- and hyper-pigmented areas. Sarcoidosis skin lesions seldom produce itching or pain, and they do not ulcerate. Ocular involvement may occur in 4 to 27 per cent of patients with sarcoidosis (Table I). All the parts of the eye or orbit can be affected. Uveitis is the most common manifestation and the clinical presentation may be acute or chronic[31]. Other ocular manifestations include conjunctival follicles, lacrimal gland enlargement, dry eye (keratoconjunctivitis sicca), dacryocystitis, and retinal vasculitis[31]. Peripheral lymphadenopathy has been observed in 8 to 44 per cent of patients (Table I). Rarely, intra-abdominal lymphadenopathy may also be present[3,4]. Joint symptoms have been described in 30 to 50 per cent of patients with sarcoidosis (Table I). These may be acute

and transient, or chronic and persistent. Acute onset arthritis – often reported from the west – is uncommon, and arthralgias are far more common than arthritis in Indian patients (Table I). Unilateral or bilateral painless parotid gland enlargement occurs in 1 to 10 per cent of patients (Table I) and may result in "chipmunk cheeks" appearance.

Hypercalcaemia and hypercalciuria are often encountered in patients with sarcoidosis. Persistent hypercalcaemia and hypercalciuria can result in nephrocalcinosis, nephrolithiasis, and renal failure. Hypothalamic or pituitary involvement can result in diabetes insipidus. Uncommonly, hypothyroidism, hyperthyroidism, hypothermia, adrenal suppression, and anterior pituitary involvement can occur[3,4]. Clinical evidence of myocardial involvement has been reported in about five per cent of patients with sarcoidosis and ranges from benign arrhythmias or high-degree heart block to sudden death[32]. Endomyocardial biopsy is useful in demonstrating granulomas and confirming the diagnosis of cardiac sarcoidosis. Cardiac involvement has seldom been systematically studied in Indian patients with sarcoidosis, and abnormalities observed on the electrocardiogram (ECG) have been the most commonly described abnormalities[3-5,10,11]. The gastrointestinal tract is involved in less than one per cent patients with sarcoidosis[2-4]. Although granulomas may be found in 50 to 80 per cent of liver biopsy specimens, palpable hepatomegaly has been observed in 8 to 43 per cent patients (Table I). Hepatic involvement rarely causes portal hypertension, hepatic failure, or increased mortality related to liver dysfunction[3,4,33,34]. Clinical evidence of the involvement of the nervous system occur in 0.3 to 13 per cent of patients with sarcoidosis (Table I). Cranial nerve involvement, particularly facial palsy and hypothalamic and pituitary lesions, are common; space-occupying mass lesions, and peripheral neuropathy, have also been reported (Table I). Similar observations have been reported in the studies from India[3-5,10].

Sarcoidosis can virtually involve every body system and unusual presentation of the disease can be a diagnostic problem. There have been very few reports regarding uncommon manifestations in patients with sarcoidosis from India and these are depicted in Table II[3,4,33,35-40]. Awareness regarding uncommon manifestations of sarcoidosis will facilitate early confirmation of diagnosis.

Radiological manifestations

The chest radiographs reveal abnormalities in more than 90 per cent of the patients with sarcoidosis at presentation. The characteristic radiological finding in patients with pulmonary sarcoidosis is bilateral hilar lymphadenopathy. Pulmonary sarcoidosis is staged by the traditional radiographic criteria as follows: stage 0 – normal chest radiograph; stage I – bilateral hilar lymphadenopathy without parenchymal infiltrates; stage II – bilateral hilar lymphadenopathy with parenchymal infiltrates; and stage III – parenchymal infiltrates without hilar lymphadenopathy. In the western literature, most of the patients had stage I disease, while most of the Indian patients with sarcoidosis presented with stage II disease (Table I). In Indian patients with sarcoidosis, it has been observed that while the chest radiographs may look startling, patients may manifest minimal symptoms and this has been termed *clinico-radiographic dissociation*)[3-5,11].

Parenchymal infiltrates that are often bilateral and symmetrical involving the upper lobes more often are evident in 25 to 50 per cent of the patients with pulmonary sarcoidosis[3,4]. Other patterns observed include reticular, reticulonodular, or focal alveolar opacities, miliary mottling, or ground glass appearance. Pleural involvement is rare and includes small pleural effusion which may be bilateral at times, thickening, pleural plaques or nodules. Occurrence of bilateral recurrent pneumothoraces has also been reported[41]. While computerised tomographic scan (CT scan) of the chest is *not routinely required* for diagnostic evaluation or follow-up of patients with sarcoidosis, it is useful in detecting enlarged lymph nodes or parenchymal infiltrates that are not evident on the conventional chest radiograph and is therefore more useful in patients with atypical or uncommon manifestations. Characteristic features of sarcoidosis on CT scan include central bronchovascular thickening and nodularity, miliary nodules, thickening of interlobular septae, luminal irregularity, ground-glass attenuation, architectural distortion, conglomerate masses, honeycombing and cystic destruction, alveolar consolidation, parenchymal and pleural nodules. The lesions are most commonly located along the

peribronchovascular sheath lymphatics and sometimes in subpleural and interlobular septal lymphatics[42]. CT scan of the abdomen is also useful in identifying uncommon lesions such as lesions in liver and spleen, retroperitoneal and abdominal lymphadenopathy[3,4]. CT scan and gadolinium (gadopentetate demeglumine)- enhanced magnetic resonance imaging (MRI) have been found to be useful in localising neurosarcoidosis and sarcoidosis of the bones[3,4,10].

Table II : Uncommon manifestations observed in patients with sarcoidosis from India.

Thoracic
- superior venacaval obstruction
- transient paralysis of phrenic and recurrent laryngeal nerves
- bullous lesions in the lungs
- chronic respiratory failure
- narrowing of main bronchi, fixed upper airways obstruction
- clinical presentation mimicking bronchial asthma with seasonal exacerbations
- pleural effusion, nodules, and pneumothorax

Gastrointestinal
- cirrhosis of liver with portal hypertension

Abdominal
- intra-abdominal lymphadenopathy
- hypodense lesions in liver and spleen

Renal
- sarcoid nephritis
- gross digital clubbing

Cardiac involvement
- complete heart block
- congestive heart failure
- supraventricular, ventricular ectopics, and recurrent ventricular tachycardia

Neurological
- bilateral sequential facial nerve palsy
- optic neuritis
- proximal myopathy
- multiple brainstem lesions
- pituitary stalk lesion
- optic atrophy

Others
- breast lumps
- glaucoma

Data is from references 3,4,33,35-40.

Laboratory investigations

Cutaneous anergy is considered to be a cardinal feature of sarcoidosis. Tuberculin skin test is often negative in nearly two-thirds of the patients with sarcoidosis (Table I). Haematological abnormalities include anaemia, leucopaenia, lymphopaenia, eosinophilia, and monocytosis among others[3,4,10]. Polycythaemia has occasionally been reported. Since several steroid-sparing alternative treatment modalities such as cytotoxic agents can affect these parameters, physicians treating sarcoidosis should be aware of these abnormalities. Hypercalcaemia has been reported to occur in 2 to 63 per cent of patients with sarcoidosis and hypercalciuria is three times more common than hypercalcaemia[3-5,10]. Elevated serum aminotransferase, alanine aminotransferase, and alkaline phosphatase have also been described[3,4,10]. In a large Indian study[3], hyperuricaemia was observed in 41 per cent of the patients tested. All the patients with hyperuricaemia had stage II disease[3]. Ureteric stones occurred in five of the 210 patients seen by the authors[4]. These observations merit further study.

Pulmonary functions

The lung function abnormalities of sarcoidosis include decreased lung volumes and diffusing capacity[3,4,10]. However, these abnormalities are not specific for sarcoidosis and are typical for interstitial lung disease of any aetiology. Pulmonary function abnormalities are present in about 20 per cent of the patients with stage I sarcoidosis, but occur in 40 to 70 per cent patients with stages II or III[3,10,11]. Airways involvement has been observed in about a third of the patients with sarcoidosis[3,4]. Very little has been systematically documented regarding the pulmonary function abnormalities in patients with sarcoidosis and the effect of prednisolone treatment on them from India. In the only study of its kind reported from India[3], pulmonary function abnormalities were suggestive of a mixed obstructive and restrictive ventilatory defect; however, an obstructive defect was predominantly observed. Review of published evidence suggests that the physiological abnormalities poorly correlate with the pathological findings[1].

Bronchoscopy

Flexible fibreoptic bronchoscopy (FOB) and bronchoscopic techniques such as trans-bronchial lung biopsy (TBLB), endobronchial biopsy, transbronchial needle aspiration (TBNA), and bronchoalveolar lavage (BAL)[43-46] have been found to be useful for studying the disease and procuring tissue for the confirmation of a diagnosis of sarcoidosis. Bronchoscopic appearances of sarcoidosis include nodules, plaques, erythema and cobble stone appearance. TBLB has a high diagnostic yield because the lesions of sarcoidosis are distributed along the bronchovascular bundle[7,47].

BAL has very little practical clinical or prognostic utility and has been utilised as a research tool[43-46]. Classically, BAL fluid reveals lymphocytic alveolitis with elevated CD4/CD8 ratios and increase in cell recovery[43-46]. A negative correlation between bronchoalveolar lavage lymphocytes and pulmonary diffusing capacity[45] have also been reported in patients with sarcoidosis. In patients with sarcoidosis, glucocorticoid receptor (GR) content of the BAL fluid cells is increased and a major contribution to this is made by the lymphocytes[44]. Log transformation of the ratio of lymphocyte per cent (L) and the per cent of polymorphs + 1 (L/P+1) in the BAL fluid was found to be useful in differentiating these two conditions[46].

Elevated levels of serum ACE have been observed in 40 to 90 per cent of patients with sarcoidosis[3-5,10] and is considered to be a marker of disease activity in reports from the West. Experience with this expensive test in the Indian context has been controversial. Gupta *et al*[5] observed elevated SACE levels in three-fourths of their Indian patients. However, the authors' group[48] did not find higher SACE activity in patients with active sarcoidosis compared to those with inactive disease. Moreover, there was no significant difference in the SACE activity among different stages in patients with active sarcoidosis[47]. One of the reasons for these differences could be differences in the protocols followed for SACE level estimation, lack of uniform laboratory standards, and non-availability of a definitive "gold-standard" for the confirmation of the diagnosis of sarcoidosis. [67]Gallium scanning has been found to be a useful adjunct in diagnosing sarcoidosis[49]. However, a negative gallium scan does not rule out active sarcoidosis. Clinicians should keep these facts in mind before ordering these expensive investigations.

Diagnosis

As there is no definitive "gold standard" for the diagnosis, sarcoidosis is essentially a diagnosis of exclusion. Estimation of serum ACE activity[67], gallium scanning have poor specificity; the antigen required for Kviem-Siltzbach test is not widely available in India, not well standardised, and not approved for general use. Thus, all suspected cases of sarcoidosis must be confirmed by tissue biopsy to exclude mycobacterial, fungal, and other granulomatous infections or malignant conditions. Specimens must be procured for histopathological examination from the most accessible site with the least invasive method. More recently, tran-sbronchial lung biopsy (TBLB) mediastinoscopy, and video-assisted thoracoscopic surgery (VATS) have been found to be useful in procuring lung as well as intra-thoracic lymph node material[3,4,10,50]. Presence of non-caseating epithelioid cell granulomas in tissue biopsy specimens confirms the diagnosis of sarcoidosis. Mere presence of non-caseating granulomas must not be taken as proof of sarcoidosis. A complete and thorough history of occupational and environmental exposure, medication use, and medical history must be obtained and other known causes of granulomatous inflammation must be excluded before a patient is labelled as having sarcoidosis. The difficulty in diagnosis is further compounded by the fact that we have observed necrosis in granulomatous lesions in some of the patients with sarcoidosis at our centre[4] and this renders differentiation from tuberculosis very difficult.

Management

The algorithm for the practical management of patients with sarcoidosis is depicted in Figure 1. Corticosteroids have been the mainstay of therapy in patients with sarcoidosis[51]. Though there are reports of short-term improvement, especially in patients with deteriorating lung function or serious extra-pulmonary disease[167], well controlled clinical trials showing definitive improvement in the long-term outcome with the use of glucocorticoids are lacking. Also, the appropriate dosage, value of daily as opposed to alternate day therapy, and the optimum duration of treatment, are also unclear[52].

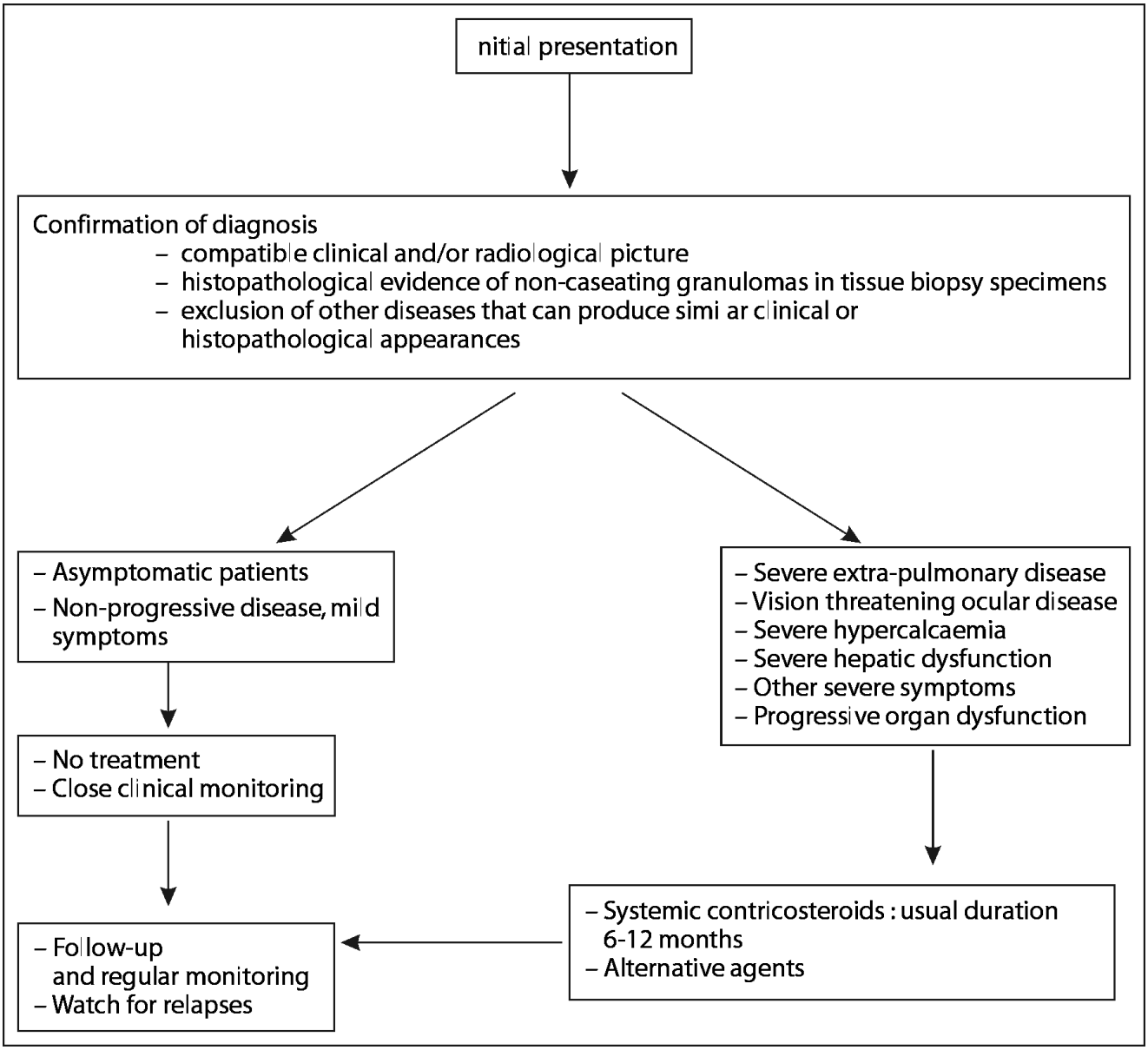

Fig. 1 : *Algorithm for the evaluation and management of patients with sarcoidosis.*

Asymptomatic patients with pulmonary sarcoidosis should not receive corticosteroids. Once treatment is initiated with corticosteroids, and an adequate interval of time has elapsed, the patient should be critically reviewed to assess the need for further continuing the treatment. If the response is considered appropriate, corticosteroids should be gradually tapered while following the patients clinically, radiologically, and physiologically. The patients should be very carefully monitored for the rest of their lifetime for any recurrences, as reactivation is not uncommon even after prolonged periods of remission.

We have observed that prednisolone treatment resulted in significant improvement in pulmonary functions, but the values returned to the baseline once the corticosteroid treatment was stopped[3]. Prednisolone treatment also resulted in a significant reduction in peripheral blood and BAL fluid complement and immunoglobulin levels. These findings suggest peripheral blood lymphopenia and lymphocytic alveolitis which improves with corticosteroid treatment[43-46].

Even steroid responsive sarcoidosis, particularly stage II or stage III disease, has been reported to relapse in about

25 per cent patients after stopping steroid treatment[50]. Our experience with corticosteroid treatment has been similar. Nineteen of the 21 patients (90.4%) who relapsed in the series we reported[3,4] had stage II or III disease. The following correlated with the development of relapse: history of malaise; presence of crepitations; wheezing; peripheral blood eosinophilia; pre-treatment FEV_1/FVC (%) less than 65 per cent predicted. Muller[53] observed that eosinophilia was a favourable prognostic sign in patients with tuberculosis. We observed the exact opposite in patients with sarcoidosis[3]. To the best of our knowledge, the observation that eosinophilia is a poor prognostic marker in patients with sarcoidosis has never been reported till date.

Several cytotoxic agents such as methotrexate, azathioprine, and non-cytotoxic agents such as hydroxyl chloroquine, thalidomide, ketoconazole among others, have been explored to treat patients with sarcoidosis[51]. These agents can cause severe adverse drug reactions which may limit their use. Definitive benefits with these agents is also not established. Further work is required to clarify these issues. Till a consensus is evolved, these agents should be used only by an experienced clinician. Treatment should be tailored to the needs of the individual case.

Learning points

- In India sarcoidosis is an under-diagnosed disease. All ethnic groups in the Indian sub-continent are affected by sarcoidosis.
- Most of the Indian patients with sarcoidosis are males; the majority presenting late in the fourth or fifth decade of their life.
- Constitutional symptoms such as fever, fatigue, malaise, and weight loss have been observed to occur more often in Indian patients.
- In Indian patients with sarcoidosis, it has been observed that while the chest radiographs may look startling, patients may manifest minimal symptoms (*clinico-radiographic dissociation*).
- Depending on the organ systems involved, patients with sarcoidosis present themselves to various specialities; pulmonary involvement is by far the commonest. Acute presentation in the form of "Löfgren's syndrome" and "Heerfordt's syndrome" is uncommon in Indian patients.
- Pulmonary function abnormalities were suggestive of a mixed obstructive and restrictive ventilatory defect; however, an obstructive defect was predominantly observed.
- For asymptomatic patients with pulmonary sarcoidosis, no therapy is required. In symptomatic patients, or those with complications, corticosteroids remain the mainstay of treatment. Corticosteroid treatment usually results in significant symptom resolution and improvement in pulmonary functions, but the values return to the baseline once the treatment is stopped. Occasionally, steroid-sparing alternative treatments may be beneficial. Treatment should be tailored to suit the needs of the individual patient under close clinical monitoring.

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