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Invited Review

# Relapsing polychondritis

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## Abstract

Relapsing polychondritis (RPC) is a unique and rarely observed autoimmune condition regarded as recurrent extensive chondritis of the auricular, nasal, and tracheal cartilages. Moreover, heart, main arteries, skin, and eyes may be involved. Several forms of clinical manifestations may be seen, and the pathogenesis still remains anonymous. A concomitant disease, particularly myelodysplasia or other systemic autoimmune disease can be detected in one-third of the patients with RPC. The treatment of RPC should be considered on personal basis and classified according to disease activity and severity. This study reviews the available data on clinical manifestations, pathogenesis, diagnosis, and therapeutics of the RPC.

**Keywords:** Relapsing polychondritis, clinics, review

### Relapsing polychondritis

Relapsing polychondritis (RPC) is an autoimmune disease characterized by the inflammation of cartilaginous tissues. The disease can affect several organs, including proteoglycan rich tissues, particularly ears, nose, respiratory tract, eyes, and joints (1-3). Owing to the widespread diversity of clinical manifestations and rarity of the disease, diagnosing it in the early stages may be challenging. Approximately 30% of the patients with RPC appear to have an association with a different disease, frequently several forms of autoimmune rheumatologic disease or myelodysplastic syndrome (MDS) (3). This review reveals clinical manifestations, pathogenesis, diagnosis, and treatment of the rare RPC disease.

### Epidemiology and genetics

The incidence and prevalence of RPC are not exactly known. RPC is seen more common in Caucasians, and a recently study reported that the calculated prevalence is 4.5 cases/million in defense population (4). Although RPC can occur at all age groups, the starting age of the disease is the fifth decade of life. Generally, studies (5, 6) showed no significant gender predilection, but Trentham et al. (7) found a slightly female predominance. Although the studies indicate a possible genetic contribution, RPC is still regarded as a nonfamilial disease.

Genetic studies have identified a relationship only between RPC and human leucocyte antigen (HLA)-DR4. A recent study (8) investigated the frequency of HLA-DR4 in patients with RPC and it was found to be up to 56% in the patient group and 26% in the healthy control group. On the contrary, in case of rheumatoid arthritis, a definite relationship between specific DR4 subtype alleles and RPC was not identified. Similarly, an important link was found between HLA-DR6 positivity and clinical findings of RPC, but the significance of this result is not clearly understood (8).

### Etiopathogenesis

The etiopathogenesis of RPC is still anonymous. Nonetheless, it seems to be a combination of constituents comprising genetic predisposition, a triggering factor, and presence of autoimmunity.

The triggering factors can be chemical, toxic, and infectious agents or direct trauma. Recently, RPC cases have been reported (9) after a trauma of the pinna and (10) intravenous administration of an indefinite substance that may have a direct toxic effect on the cartilage. This suggests that there may be a direct association with trauma and the triggering of autoimmune phenomena. Cañas et al. (11) conducted a study in patients with RPC and showed that patients who had previous cartilage trauma had more autoimmunity features than those who did not have trauma. Possible mechanisms include that cryptogenic antigen release, recognition of pathogenic structure, and metabolic changes generated by trauma (12).

The propensity of RPC to involve cartilaginous structures proposes the occurrence of humoral immunity against this proteoglycan rich tissue. Using the indirect immune fluorescence method, circulating and tis-

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**Table 1.** Systemic diseases associated with relapsing polychondritis* (3, 26, 34)

| Disease Group | Subtype |
|---|---|
| Connective tissue diseases | Systemic lupus erythematosus, Sjögren's syndrome, systemic sclerosis, rheumatoid arthritis, juvenile idiopathic arthritis |
| Spondyloarthritis | Ankylosing spondylitis, reactive arthritis, psoriatic arthritis |
| Systemic vasculitis | ANCA-associated vasculitis, Takayasu arteritis, Behçet's disease, polyarteritis nodosa, cryoglobulinemia |
| Hematologic diseases | Myelodysplastic syndrome, lymphoma |
| Gastrointestinal diseases | Inflammatory bowel disease, primary biliary cirrhosis |
| Dermatologic disease | Sweet syndrome, dermatitis herpetiformis, lichen planus, psoriasis |
| Other inflammatory disease | Retroperitoneal fibrosis, meningoencephalitis, familial Mediterranean fever, sarcoidosis, autoimmune thyroid disease |

*Adopted from reference 28

ANCA: anti-neutrophilic cytoplasmic antibody

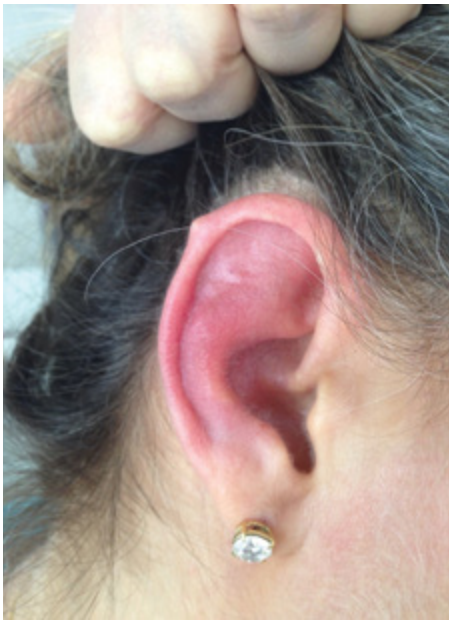

**Figure 1.** Auricular involvement characterized by redness, swelling of the pinna, and sparing of the lobule

sue antibodies against collagen type II, IX, and XI were revealed in patients with RPC (13-15). Studies showed that an increased serum level of the cartilage-specific protein matrilin-1 can be found in patients with RPC, particularly in the active phase (16, 17). However, neither anti-collagen type II nor anti-matrilin-1 antibodies have considerable sensitivity and specificity to be used for diagnostic purposes.

Besides antibodies and humoral immunity, which play a role in disease pathogenesis, cellular immunity may maintain cartilage inflammation (18-20). It has been shown that the activation of T cells lead to the production of helper T cells 1 (Th1) cytokines containing tumor necrosis factor alpha, interferon-γ, Interleukin-8, and macrophage inflammatory protein 1 in RPC cases (21, 22). It can be hypothesized that a damage to the cartilage containing chondrocyte epitopes leads to cytokine release and local inflammation following autoantibody production in an inherently susceptible host (22).

## Clinical features

The clinical picture of the RPC changes with severity and duration of the disease. The most common and characteristic feature is auricular involvement, but other sites of the body and tissues may be involved.

Auricular involvement is present in 90% of the patients with RPC, and inflammation is restricted to the cartilaginous fragment of the auricle. Earlobes are typically protected from inflammation. (Figure 1). Most patients refer to medical attention with auricular discoloration, tenderness, or pain (23, 24). Attacks frequently occur in a relapsing–remitting pattern and may cause deformities of the pinna. In conjunction with common symptoms, there may be infrequently seen findings in approximately one-third of the patients with RPC with ear involvement. For instance, inner ear inflammation may cause impaired hearing, tinnitus, and vertigo (25).

Ocular inflammation in RPC can affect any part of the eye and may affect 20%–60% of the RPC cases. Most common ocular manifestations are episcleritis, peripheral ulcerative keratitis, scleritis, and uveitis (24, 26).

Nasal cartilage inflammation can cause crusting, epistaxis, and rhinorrhea. Nasal chondritis is present in approximately 20% of the patients at presentation and 60% over the course of the illness. Cartilage destruction associated with relapsing attacks of inflammation can result in a characteristic saddle nose deformity (3, 27).

Respiratory tract manifestations of active RPC include coughing, roughness, aphonia, wheezing, dyspnea, or sensitivity above the trachea (5, 28). Approximately 50% of the patients with RPC complicated by laryngo-tracheobronchial disease, which may lead to airway obstruction resulting in even death. Tracheostomy or a stent implantation may be compulsory in severe obstructions (29).

Arthritis is the first presenting symptom in 33% of the patients with RPC and is eventually observed in 50%–75% of the patients with RPC (30). RPC characteristically affects the manubriosternal, sternoclavicular, and costochondral joints. Any joint may be involved, but the frequently affected joints are the metacarpophalangeal, proximal interphalangeal, knees, and wrist joints. Usually, arthritis is seen as polyarthritis or oligoarthritis with or without synovitis and may last from weeks to months. It has an episodic, self-remitting, asymmetric, nonerosive, and nondeforming course (31).

Approximately 10% of the patients with RPC have clinically remarkable valvular disease mostly affecting aortic or mitral valves. Additionally, cardiac involvement may be associated with pericarditis, heart blocks, and myocardial infarction. Valvular involvement can occur any time during the disease course (32). Progression of the disease is often insidious; as a result, echocardiography should be periodically performed to determine valvular dysfunction (33).

Renal disease arises in a minority of RPC cases. Various kidney pathologies can occur in RPC cases, including immunoglobulin (Ig) A nephropathy, tubulointerstitial nephritis, and glomerulonephritis (27, 34).

In addition to audiovestibular manifestations, neurological involvement in RPC appears in approximately 3% of the cases. The most common neurologic features are cranial neuropathies of the second, sixth, seventh, and eighth nerves. However, miscellaneous neurological conditions can occur such as hemiplegia, seizures, organic brain syndrome, dementia, and cerebral dysfunction (3, 35).

Several nonspecific skin lesions may develop in patients with RPC. Almost 36% of the patients

**Table 2.** Criteria for diagnosing relapsing polychondritis* (5, 37)

| Author | Criteria |
|---|---|
| Mc Adam et al. | Recurrent chondritis of both auricles |
| | Nonerosive inflammatory polyarthritis |
| | Chondritis of nasal cartilages |
| | Inflammation of ocular structures |
| | conjunctivitis/keratitis/scleritis/uveitis |
| | Chondritis of respiratory tract |
| | laryngeal/tracheal cartilages |
| | Cochlear and/or vestibular damage |
| | neurosensory hearing loss/tinnitus/vertigo |
| McAdam et al. (5) | Requires 3 of 6 criteria to diagnose |
| Damiani et al. (37) | 3 of 6 McAdam et al. (5) criteria or |
| | 1 of 6 McAdam et al. (5) criteria and a positive histologic confirmation or |
| | 2 of 6 McAdam et al. (5) criteria and a response to corticosteroid or dapsone |

*Adopted from reference 31.

have skin lesions such as oral or skin ulcers, papules, purpura, and nodules. Skin biopsy studies show nonspecific findings, including cutaneous leucocytoclastic vasculitis, small vessel thrombosis, and panniculitis (33, 36).

### Associated diseases

As many as one-third of the patients with RPC have a concomitant disease including systemic vasculitis, dermatologic or hematologic disease, or other systemic rheumatic disease (Table 1) (3, 28, 36). An intercurrent disease may precede RPC, occur after the diagnosis of RPC, or present simultaneously with RPC. In the last setting, RPC symptoms and signs may be the cause for hospital admission.

### Diagnosis

Relapsing polychondritis has no pathognomonic clinical, radiological, and histopathological features. The diagnosis is established by the constellation of clinical manifestations, complementary laboratory data, radiological procedures, and biopsy of a cartilaginous site.

Well-known diagnostic criteria are the original "so-called" McAdam's criteria, which needs the existence of three or more of the clinical findings summarized in Table 2 for diagnoses (5). Damiani et al. (37) suggested a new set of criteria including histological features and therapeutic responses (Table 2).

There is no specific laboratory test for RPC, but measurement of acute phase reactants can be helpful similar to that in other autoimmune diseases. Approximately 10% of the patients may have peripheral eosinophilia that suggests vasculitis as a potential differential diagnosis. Particularly when there is an overlap with other rheumatologic disorders, patients with RPC may have a positive serology such as rheumatoid factor, anti-neutrophil cytoplasmic antibody (ANCA), anti-nuclear antibody (ANA), and false-positive venereal disease research laboratory (VDRL) tests (27, 38, 39).

In addition to the clinical evaluation and laboratory tests, the work-up should include imaging studies such as dynamic expiratory computed tomography (CT), magnetic resonance imaging (MRI), positron emission tomography (PET), and Doppler echocardiography along with lung function tests. When patients are diagnosed with RPC, large airways should be scanned by CT to evaluate laryngo-tracheal bronchial luminal narrowing, wall thickening, and calcification of cartilaginous structures (27, 40). MRI is more advantageous than CT to discriminate among fibrosis, inflammation, and edema (41, 42). PET/CT is used for the early diagnosis of RPC as well as for the assessment of activity and extent of disease (43, 44). Doppler echocardiography is used to evaluate the involvement of cardiac valves.

### Treatment

There are not many clinical trials to estimate treatment modalities for RPC because of the rarity of the disease. The pharmacological approach is mainly based on large series of single or multiple case reports. Despite successful suppression of the clinical features, no therapy has been proved to change the natural course of the disease.

Medical treatment of RPC depends on disease severity and extension of the disease. Active large airway, cardiac, and main artery involvement usually demonstrate the need for aggressive therapy. Recently, an activity scoring system has been developed for RPC. RPC Disease Activity Index (RPDAI) can be used for the assessment of activity in routine clinical practice.

For patients with nasal, auricular, and articular chondritis but no visceral involvement, anti-inflammatory drugs, colchicine, or dapsone can be attempted with limited efficacy (6, 7, 33). Low-dose glucocorticoid therapy is often required. For patients with large airway, ocular, cardiovascular, neurologic, or renal disease, the initial treatment is determined by the assessment of disease severity. For those with relatively mild involvement, oral glucocorticoids can be initially used. In patients with potentially severe manifestations (severe laryngeal or tracheobronchial chondritis, very recent and abrupt onset of sensorineural hearing loss, or systemic vasculitis with poor prognosis factors), methylprednisolone bolus therapy (15 mg/kg/day) combined with an immunosuppressive or immunomodulatory agent (4, 5, 6, 34) can be beneficial as an initial therapy.

Several immunosuppressive and anti-inflammatory drugs were used to lessen the duration and doses of corticosteroid treatment. The frequently used immunomodulatory drugs are cyclophosphamide, methotrexate, azathioprine, and cyclosporine. Cyclophosphamide is a commonly used immunosuppressive drug based on experiences in other systemic rheumatic diseases, particularly systemic vasculitis (45, 46). It can be used at a quantity of 1–2 mg/kg (Maximum dose 150 mg/day) per oral until a clinical response is obtained. After achieving remission, the dose should be reduced to a maintenance level. (46).

The efficacy of biological agents in the treatment of RPC has been recently reviewed (47). This study showed that infliximab has been the most frequently used biological agent so far with variable results that are difficult to interpret. Nearly half of the patients benefited from infliximab therapy but several experienced

infections, of which a few were fatal (47). Rituximab usually has no treatment effect. Abatacept was used in a pilot study of four patients and for exploratory purposes in three patients with variable results (47). Other biological agents attempted in case of RPC include anakinra, tocilizumab, etanercept, adalimumab, and certolizumab, but the numbers of treated patients are too small to draw definitive conclusions (47).

Surgery may be needed in selected patients owing to some of the visceral manifestations such as the segmental collapse of airways (48, 49). Surgical management may also be necessary for intractable heart failure because of valvular regurgitation and for aortic aneurysms.

### Prognosis

Improvements in the outcomes of patients with RPC have been achieved in recent years, probably because of better comprehension of the disease, thereby allowing the diagnosis of mild forms and development of better treatment strategies. Survival rates were reported to be increased from 70% at 5 years (5, 6) to 91% at 10 years in a recent study (33, 50). Main causes of death in patients with RPC are specific organ involvements such as airways, blood vessels, concomitant MDS, and infections in which development is facilitated by the treatment itself (5, 6). Despite the course of RPC being rapidly lethal, the more common patterns reproduce a relatively benign disease. Some disabilities can be seen in the chronic phase of RPC, including visual impairment, audio-vestibular dysfunction, and cardiopulmonary disease (7, 33, 50).

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