

Review

Omalizumab for atopic dermatitis: case series and a systematic review of the literature

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Abstract

Omalizumab is a recombinant humanized monoclonal antibody targeting the high-affinity Fc receptor of IgE, registered for the treatment of chronic spontaneous urticaria and severe allergic asthma. We present a case series of nine patients with atopic dermatitis (AD) treated off-label with omalizumab and a systematic review of the existing literature. Patients were selected consecutively from a tertiary dermatological referral center during a 5-year period. All patients were treated with omalizumab at a starting dose of 300 mg subcutaneously every 4 weeks. Systematic literature searches were performed in PubMed, Web of Science, EMBASE, and ClinicalTrials.gov to identify any study (case reports, case series, and controlled trials) evaluating the effect of treatment with omalizumab in AD. Based on physicians' assessment, 50% of our patients had a good or excellent response to treatment with omalizumab; a further 12.5% had a moderate response, while 37.5% experienced no response or deterioration of symptoms during treatment. Treatment was generally well tolerated. Twenty-six studies with a median of four patients each (range 1–21), comprising 174 patients, were included in the systematic review. Summed over all studies, a total of 129 patients (74.1%) experienced a beneficial effect of treatment ranging from little to complete response. Omalizumab appears to be a safe and well tolerated, however expensive, treatment with some clinical benefit in patients with severe recalcitrant AD. Recommendation for use in clinical practice awaits evidence from larger randomized controlled trials.

Introduction

Atopic dermatitis (AD) is a chronic inflammatory pruritic skin disease,¹ which is part of the atopic triad involving also asthma and allergic rhinitis.^{2,3} AD has a lifetime prevalence of 10–20% and primarily affects young children,⁴ although it can present at any age.⁵ AD impacts negatively on quality of life,⁶ and optimal disease control is therefore paramount. Current management of severe recalcitrant AD includes systemic treatment with immunosuppressant drugs such as azathioprine, prednisolone, cyclosporine, or mycophenolate mofetil, as well as methotrexate and ultraviolet radiation. Treatment with biologic agents is in the pipeline but not yet registered for treatment of AD.

Omalizumab is a recombinant humanized monoclonal antibody targeting the high-affinity Fc receptor of immunoglobulin E (IgE) approved for the treatment of moderate to severe asthma and chronic spontaneous urticaria.⁷ Additionally, omalizumab has been used off-label for various other diseases involving IgE such as AD with

varying results.^{8–10} We present a case series of patients with AD treated with omalizumab in a dermatological university department. Furthermore, we systematically review the existing literature identifying studies of AD treated with omalizumab.

Methods

The patients reported herein were selected consecutively from the Department of Dermatology, Bispebjerg University Hospital (Copenhagen, Denmark), in the period between June 2010 and July 2015. All patients were treated with omalizumab for recalcitrant AD, defined as persisting AD despite treatment with ≥ 4 different drugs included in the Danish guidelines for AD treatment. Patients were treated with an initial dose of 300 mg every 4 weeks, based on the dosage recommended for the treatment of chronic spontaneous urticaria. The clinical response to treatment with omalizumab was evaluated based on the physician's assessment as well as the change in the patient's symptomatic complaints reported in each

patient file representing three grades:¹¹ grade 1, failure of treatment; grade 2, moderate improvement; and grade 3, successful treatment. In addition, the duration of treatment, as well as any adverse effects of omalizumab, was recorded. Furthermore, the following variables were retrieved from patient records: presence of atopy, systemic comorbidities, duration of and previous treatment of AD, serum total IgE, and filaggrin gene (*FLG*) mutation status.

The response to treatment in our patients was compared to reports from the existing literature retrieved from PubMed, Web of Science, EMBASE, and ClinicalTrials.gov using the search terms: eczema, dermatitis, atopic, omalizumab, and anti-IgE. Cross-references were retrieved, but this did not identify additional studies. Studies published by August 2015 were included.

Results

Present study

Nine patients (five males) with a mean age of 49.1 years were identified (Table 1). Seven patients had asthma, and all patients had atopy verified by skin prick test or RAST. The mean baseline IgE was 2750 IU/mL (range 1540–12,500). All patients received topical corticosteroids, whereas six patients (77.8%) had received systemic corticosteroids, and seven (77.8%) had received tacrolimus ointment and/or pimecrolimus cream. All patients had previously been treated with cyclosporine, methotrexate, and/or azathioprine. The average number of earlier topical and systemic treatments was 7.4 (range 3–11).

Follow-up data on treatment effectiveness was only available for eight of the nine patients, because treatment startup for the ninth patient was too recent to involve evaluation. Four patients (50%) had a good or excellent response (successful) to treatment with omalizumab; one (12.5%) had some effect (moderate), while three (37.5%) experienced no response or deterioration of symptoms (failure) during treatment. In general, treatment was tolerated very well, and no patients reported any adverse effects except episodic local flare-ups of AD, which is believed to be a natural part of active AD. One patient reported shivering and subfebrilia (flu-like symptoms), which is a known side effect of omalizumab.

Literature search

Our literature search identified 26 studies (five case studies, 18 case series, one prospective single-center investigation, and two randomized, double-blinded, placebo-controlled pilot studies) comprising 185 (results reported on 174) patients, with a median of four, ranging from 1 to 21 patients exploring omalizumab treatment for recalcitrant AD. A total of 129 (74.1%) patients experienced

a beneficial effect of treatment ranging from little to complete response (Table 2).^{8,10,12–28} Forty-five patients (25.9%) from seven different studies reported no or negative effect off omalizumab treatment.^{9,29–32} Ongoing trials (ClinicalTrials.gov) exploring the use of omalizumab in the treatment of AD are being conducted.³³ We identified seven trials with two currently posting results (not included).

Discussion

This case series of nine adult patients with recalcitrant AD treated with omalizumab showed that 62.5% experienced some benefit of the treatment, whereas 37.5% had no effect or experienced deterioration of symptoms during treatment. This adds to the growing body of literature supporting the use of omalizumab in the treatment of AD, refractory to more conventional treatments.

Two randomized placebo-controlled trials (RCTs) were identified^{17,18} with a combined total of 17 patients treated with omalizumab. Notably, Iyengar *et al.*¹⁸ showed an overall positive effect of the treatment corresponding to a 20–50% reduction in the scoring of atopic dermatitis (SCORAD) that, however, did not exceed the effect observed in the placebo group (approximately 45–80% reduction). Likewise, Heil *et al.*¹⁷ did not show any statistically significant difference in clinical improvement between the treatment and placebo groups, evaluated on investigator's global assessment. Both studies reported a decrease in IgE levels during treatment. Furthermore, one prospective single-center investigation of 11 patients by del Pozo¹⁶ reported a statistically significant decrease in mean SCORAD of 31.5 points from a baseline average of 71.2 (range 43–101) and a reduction in mean dermatology life quality index (DLQI) from 9.36 to 3.72 after treatment. In addition, of the 18 retrospective case series, with a range of 3–21 patients, 110 of 141 patients (78%) showed a clinically positive effect from treatment with omalizumab. Eleven patients experienced a deterioration of disease, while 20 had no response to treatment. Finally, five case studies of single patients were reviewed, with two studies reporting excellent^{19,20} and two good^{15,22} response to treatment. In particular, Forman and Garrett²² observed a deterioration of disease during a 4-week interruption of treatment but a positive clinical effect during treatment periods. The study showing no response²⁹ reported improvement when dosage was increased to 375 mg/2 weeks (from an initial dose of 150 mg/4 weeks).

A report on 73 patients by Hijnen *et al.*¹¹ found improvement ranging from moderate (16%) to successful (77%) in 93% of their study population treated with cyclosporine. Compared to omalizumab treatment, our results showed a positive effect in 62.5% of patients

Table 1 Case series

ID	Sex	Age	Duration (years)	FLG	Atopy	Asthma	IgE IU/mL	Smoking	Comorbidities	Previous treatment	Dose (mg/month)	Time on Therapy	Effect	Side effects
1	M	62	53		Yes	No		Yes	COPD, osteoporosis, urticaria, keratoconjunctivitis	AR, MFM, CsA, PL, SS, TL, TS	300	3 years ^a	Successful	None
2	F	49	49		Yes	Yes	1540	No	Osteoporosis	AB, Aza, CsA, EZ, H1, MFM, MTX, PL, SS, TS	600	5 years ^a	Moderate	Flare-up
3	F	19	3		No	No	> 2000		None	Aza, CsA, MFM, SS, TS	300	10 months ^a	Successful	None
4	M	68	64		Yes	Yes	6130	Former, with 45 patient years	Brain surgery, following apoplexy and now aphasia.	Aza, CsA, MFM, MTX, SS, TS, TL	300	5 years 2 months ^a	Successful	None
5	F	68	67	Homo, 3	Yes	Yes	12,500	No	COPD	AR, Aza, H1, PL, TL, TS	300	5 days	Failure	Worsening of AD
6	M	67	Many	Normal	No	Yes	> 2000	No	Osteoporosis	AB, AR, Aza, CsA, H1, MFM, MTX, PL, SS, TL, TS	300	6 weeks	Failure	Flu-like symptoms erythroderma, desmoplastic lymphadenopathy.
7	M	36	32	Normal	Yes	Yes	> 2500	Yes	None	AB, AR, Aza, H1, MFM, MTX, PL, SS, TL, TS	300	24 days	Failure	Flare-up of AD with prurigo nodules and eczema on hands and feet
8	F	42	37	Hetero, 1	Yes	Yes	> 2000		IBS, generalized anxiety	Aza, CsA, H1, MFM, PL, SS, TL, TS	300	9 months ^a	Successful	None
9	M	31	31	Hetero, 1 + 3	Yes	Yes	1830	No	None	Aza, H1, TS	300	1 months ^a	-	

AD, atopic dermatitis; AR, aliretinoin; Aza, azathioprine; COPD, chronic obstructive pulmonary disease; CsA, cyclosporine; EZ, efalizumab; F, female; FLG, flaggrin; H1, second-generation antihistamines; IBS, irritable bowel syndrome; IU, international units; M, male; MFM, mycophenolate mofetil (CellCept); MTX, methotrexate; PL, pimecrolimus; PY, pack years (20 cigarettes a day for one year); SS, systemic steroids (prednisolone); TL, tacrolimus; TS, topical steroids.

^aOngoing treatment.

Table 2 Reviewed cases

1st author	Year	Pt.	Men	Age (years)	IgE mean	BL doses (s.c.)	Time on Therapy	Eczema (baseline)	Atopy	Comorbidities	Effect	Side effects	Comments
Heil ¹⁷	2010	13	5/13	30 (median)	372.78	0.016 mg/kg/IgE	16 wk	Hanifin and Rajka criteria met for inclusion ³⁶ Mean baseline IGA 2.92	Yes	AA, AR	→ Overall clinical effect	Inj. site reaction, vertigo, headache, migraine, pruritus, infection, simple labial herpes, pharyngolaryngeal pain, muscle strain, hematoma	Effect on IgE levels. No significant difference in improvement between treatment groups.
Iyengar ¹⁸	2013	4		7.4	1065.75	150-375 mg every 2-4 wk	24 wk	Mean SCORAD 73.50		AA and AR	↑↑ Overall effect	None	Effect only based on IgE level decrease.
del Pozo ¹⁶	2010	11	1/11	12-52			10mo	Mean SCORAD 71.20			11 × ↑↑	None	Gradual improvement in all pts.
Krathen ⁹	2005	3	2/3	39.3	17,613.30	450 mg/2 wk	4mo	Chronic severe AD		Asthma, allergic rhinitis, hypertension	3 × →	None	AD with periodic flares of acute disease
Lane ⁸	2006	3	2/3	11.7	3666.70	150 mg/2 wk	6mo	80% skin affection.	Yes	Multiple allergies, asthma.	2 × ↑↑↑	Headache, dizziness, acute urticaria	Dosage increase to 450 mg/2 wk. One pt. pos. MRSA skin swap
Vigo ¹⁰	2006	7	1/7	31.6	1062.90	375 mg/2 wk	7mo	IGA: 4 × 3 2 × 4 1 × 2	Yes	Asthma and AR	7 × ↑↑		Oldest pt. 300 mg/2 wk.
Belloni ³¹	2007	11		–	>>1000	150 mg/2 wk	20 wk			ARC, AA	2 × ↑↑↑ 4 × ↑↑ 3 × → 2 × ↓	None	3 pts reported under Andres ³²
Lim <i>et al.</i> ³⁴	2007	3	2/3	41	20,867	150 mg/2 wk	10 cycles = 20 wk	SCORAD	Yes	11/14 had asthmatic bronchitis and 11/14 ARC	→ 2 × ↑↑↑		14 pts treated, only 3 reported and compared to a 4th pt receiving Cyclosporine with a SCORAD reduction of 25.
Sheinkopf ²⁷ (only abstract available)	2008	21		14-64	1521		9mo				Overall ↑↑		All pts showed clinical and statistical significant improvement.
Andres ³²	2008	11		–	> 1000	150 mg/2 wk	20 wk			All AA and ARC	6 × ↑ 3 × → 2 × ↓	None	
Amroff ²¹	2010	3	2/3	18.7	14,083.70	375 mg/2 wk (1pt got 300 mg/2 wk)	Mean 10.7mo	Mean SCORAD 74.23	Yes	Asthma, AR	↑ ↑↑ ↑↑↑	Single eczema flare	Discontinuation of 1 pt (work and economy) Effect after 12 mo of treatment

Table 2 Continued

1st author	Year	Pt.	Men	Age (years)	IgE mean	BL doses (s.c.)	Time on Therapy	Eczema (baseline)	Atopy	Comorbidities	Effect	Side effects	Comments
Velling ²³	2011	9	3/9	38.1	3528	150 mg/2 wk	12 mo	DLQI average 19 at baseline.	Yes	Allergic asthma	Overall ↑↑	Headache, nausea, local reaction at point of inj.	Pts with concomitant AA and AD. Effect on AD evaluated. Max dose of 450 mg/2 wk.
Thaiwat ¹³	2011	3	1/3	32.7	489.80	300 mg/2 or 4 wk	4 mo	Trunk, face and flexural extremities	Yes	AR, asthma, and nodular prurigo	3 × ↑↑	None	
Toledo ¹⁴	2012	4	3/4	26.5	13,796.50	150 mg/2 wk	7.5 mo (6–12)	SCORAD ≥ 50		Allergic asthma	3 × ↑↑↑ →	Inj. site irritation hypotension after the first dose, herpetic eczema.	Combined therapy with IVIG. 2 pts increased to 300 mg/2 wk Xolair 5 pts had IgE > 5000 IU/mL.
Kim ³⁷	2012	10	6/10	26.2	4060.40	300 mg/2 wk	16 wk	Mean SCORAD 57.54	Yes	AR, asthma, ARC	2 × ↑↑↑ 5 × ↑↑ 3 × →	None	
Martinez ²⁸	2012	9	4/9	31	8464.40	450 mg/3 wk	Average 11 inj. = 33 wk (2–22 inj.)	Severe AD, refractory to minimum 2 systemic drugs		3/9 had asthma, 1 got ductal breast cancer	1 × → 1 × ↑ 4 × ↑↑ 3 × ↑↑↑	Inj. site reaction, vasovagal syncope, recurrence of lesions	One patient received 600 mg/3 wk and one 300 mg/2 wk. Eczema, pruritus and QoL were evaluated.
Sánchez-Ramón ¹²	2013	4	1/4	36	12,300	450 mg/2 wk	3 mo	Mainly head and neck.	Yes	Asthma, ARC, depression, food allergy, emphysema (α ₁ -antitrypsin deficiency)	2 × ↑↑↑ 2 × ↑↑	Urinary and respiratory tract infections.	Combination therapy study with Rituximab. Response from 1st cycle in 4 pts receiving only omalizumab.
Barrios ²⁶	2013	7	4/7	10.6	16,007.10	375 mg/2 wk	29.30mo (12–68)	Initial SCORAD average 75.40	Yes	Allergic rhinitis, asthma	Overall ↑↑↑	None	2 pts were treated with 225 mg/2 wk and 1 with 300 mg/2 wk
Hotze ³⁰	2014	20	9/20	23–76	–	150 mg/2 wk	28 wk		Yes		4 × ↑↑↑ 4 × ↑↑ 5 × → 7 × ↓		SCORAD reduction ≤ 25% from visit 8 resulted in an increase in dosage to 300 mg/2 wk. Deterioration believed to be flare-ups rather than drug-induced negative effects

Table 2 Continued

1st author	Year	Pt.	Men	Age (years)	IgE mean	BL doses (s.c.)	Time on Therapy	Eczema (baseline)	Atopy	Comorbidities	Effect	Side effects	Comments
Zink ²⁴	2015	10	8/10	43.7	18,094.20	450 mg/2 wk	6 mo	Mean SCORAD 59.89 And mean VAS (0–100) 56		Asthma and AR	Overall ↑↑	Exacerbations, headache, elevated liver enzymes, abdominal pain, axillary lymph node swelling.	Omalizumab combined with immune absorption therapy. Improvement of SCORAD and VAS during 6 m treatment.
Romano ²⁵	2015	3	2/3	20.3	3961.70	300 mg/2 wk	22.3 mo average (7–42)	Initial SCORAD average 46.30	Yes		↑↑ 2 × ↑↑↑	Pain at inj. site upon administration	1 pt received 450 mg/2 wk. This study included 6 CU pls.
Foman ²²	2007	1	1	41	7340	375 mg/2 wk	16 wk	Hypertrophic scar-like lichen, areas of depig. and atrophy.		None	↑↑	None	4 wk break Patient discontinued after 16 wk due to cost.
Incorvaia ²⁰	2008	1	0	39	1304	375 mg/2 wk	5mo	SCORAD 75	Yes	Asthma	↑↑↑	None	80% reduction of SCORAD over 5 months. Patient still treated when published.
Caruso ¹⁹	2009	1	0	15	107	300 mg/4 wk	7mo	SCORAD 55	Yes	AA, ARC	↑↑↑	None	Dose decreased to 300 mg/2 wk after 2mo.
Park ¹⁵	2010	1	1	34	9360	600 mg/2 wk	8mo	SCORAD 48		Herpetic eczema, secondary infection, AR, asthma, cataract.	↑↑	None	Improvement when dosage was increased to 375 mg/2 wk
Quist Ambach ²⁹	2013	1	1	53	15,000	150 mg/4wk ^a	15mo	Extremities, face, chest and upper back.	Yes	Lymphadenopathy, frequent bacterial and viral skin superinfections, ARC	→	None	

↑, mild; ↑↑, partial; ↑↑↑, complete; →, none; ↓, deterioration; AA, allergic asthma; age, mean age or range; AR, allergic rhinitis; ARC, allergic rhinoconjunctivitis; BL, baseline; CU, chronic urticaria; depig, depigmentation; DLQI, dermatology life quality index; hyperpig., hyperpigmentation; IGA, investigator's global assessment; IgE, baseline mean serum total immunoglobulin level (IU/mL); inj., injection; IVIG, intravenous immunoglobulin G; lichen, lichenification; mo, months; Pt(s), patient(s); QoL, quality of life; s.c., subcutaneous; SCORAD, scoring atopic dermatitis; VAS, visual analogue scale; w/k, weeks.

^aDosage was increased to 300 mg s.c. every 2 weeks, when extracorporeal photopheresis was stopped after 15 months of treatment, and was further increased to 375 mg/s.c. biweekly 5 months later.

treated, whereas the reviewed studies showed effect in 74.1% of the patients treated with omalizumab. Lim *et al.*³⁴ reported a comparable effect (SCORAD decreased by 25 points) between three patients treated with omalizumab and one treated with cyclosporine (3 mg/kg body-weight).

The observational studies of omalizumab for AD, in general, report more positive results compared to the RCTs, which found no difference in effect from placebo. This could indicate publication bias. Both RCTs and four of the case series^{23,24,26,27} use overall effect as a simplified measure for clinical outcome. In these studies, disease severity was usually stratified to outline different patient groups assessed at baseline, involving numerous validated scoring systems including IGA,^{10,17} visual analogue scale (VAS)²⁴, DLQI,²³ or SCORAD,^{14,16,18–21,24–26} while others report main anatomical skin areas of involvement.^{12,13,22,29} SCORAD was the most widely used, in 13 studies. This could argue SCORAD as the recommended standardized measure for disease severity. However, without a stringent definition of clinical outcome, based on a validated disease severity measure, results are difficult to compare and validate. As an example, Caruso *et al.*¹⁹ presented a case study with clinical benefit, based on an 80% SCORAD reduction over 5 months, validated as an excellent response. Contrarily, Zink *et al.*²⁴ report the results of their case series of 10 patients as an overall good effect, referred to SCORAD and VAS improvement during treatment, without any exact measures. No trend has been noted between baseline severity measured by SCORAD and clinical outcome. Furthermore, it is not explicit to what extent patient interviews have been the basis of treatment evaluation in the reviewed studies. The source of evaluation is therefore inconsistent between studies. We did not use a validated scoring system to assess objectively the treatment effect of omalizumab, which is a limitation of our study. However, physician global assessment has previously been used as an informative marker of treatment response to systemic immunomodulatory drugs in AD.¹¹

Our results agree with Amrol,²¹ reporting variation in the clinical improvement between patients. This difference in effect may reflect the clinical heterogeneity of AD. Notably the best effect was seen among the youngest patients, suggesting a contribution of the treatment to the natural outgrowing of AD.²¹ As seen in the case series with the lowest mean age^{8,19,21,26} (<20 years), which reports good or excellent clinical effect on all but one patient, who still showed mild improvement. The interpretation of improvement as an effect of treatment should accommodate the fact that most patients with AD (75%) have a spontaneous remission of disease before adolescence.⁴ It is therefore impossible to determine whether

the effects reported are a combination of the two, or solely due to the natural history of AD.

The role of *FLG* mutation status was explored by Hotze *et al.*³⁰, where none of the seven patients carrying an *FLG* mutation responded to treatment. All eight responders were non-mutation carriers, indicating that patients with primary skin barrier deficiency are less likely to benefit from immunomodulatory treatment with omalizumab. This did not agree with our results, where none of the two non-mutation carriers had a response to treatment, whereas one mutation carrier had a positive response and one experienced failure of treatment.¹¹

In the reports reviewed, eight different baseline treatment regimens were identified, with the initial dosage ranging from 150 mg/2 weeks (Quist *et al.*²⁹ every 4 weeks)^{8,13,14,18,23,29–32} to 600 mg/2 weeks,¹⁵ and with different strategies for increasing dosage throughout treatment^{8,14,29,30} (see comments in Table 2). The average duration of treatment was 8.1 months, ranging from 3 to 29.2 months, with two studies far exceeding average treatment duration.^{25,26} The regimen 150 mg/2 weeks (or 300 mg/4 weeks) was used in eight studies and hereby the most widely used. One study increased dosage until an improvement in dermatological status was seen, with a maximum of 450 mg/2 weeks.²³ Yet another followed the guidelines for treatment of asthma and dosed after bodyweight and IgE level (0.016 mg/kg/IU).¹⁷ Some studies treated with lower than recommended dose and reported clinical benefit from treatment.^{8,31} Of the five studies reporting negative or uncertain positive effects, four were of minimal starting dose,^{29–32} while Krathen and Hsu⁹ were at 450 mg/2 weeks.

Several studies address the effect of omalizumab in binding free IgE. Heil *et al.* reported no significant clinical improvement despite great changes in IgE levels.¹⁷ In contrast Zink *et al.*²⁴ found a relationship between reduction in free IgE levels and clinical improvement. Treatment discontinuation led to an increase in IgE within weeks. The same aggravating pattern was seen for both SCORAD and VAS.²⁴ No observational pattern was seen between baseline IgE and clinical outcome in any other studies or among our patients.

The studies including the longest follow-up periods also tended to report more positive results. Amrol²¹ observed improvements in DLQI, SCORAD, and VAS, 3 months after initiation of treatment, whereas Velling *et al.*²³ treated patients for 12 months despite showing an improvement in AD already after 2 months with a far lower dosage. This accounts for the possible placebo-induced clinical improvement in AD suggesting follow-up periods sufficiently long to allow an effect on clinical symptoms from treatment with omalizumab to be seen. Iyengar *et al.*¹⁸ observed no clinical difference between treatment

and placebo, and Belloni *et al.*³¹ described improvement in up to 40% of placebo-treated cases, which raises the question as to whether positive clinical outcome is caused by omalizumab or spontaneous improvement, just as observed flare-ups in AD are not a deterioration of disease due to treatment but part of the disease itself.

We and most other studies report no or very few side effects of treatment with omalizumab. Only eight studies reported side effects,^{8,12,14,17,23–25,28} and none of these were severe. The most common side effects were injection site reactions^{14,17,25,28} and headache, dizziness, and nausea.^{8,17,23,24} The most severe reaction was a vasovagal syncope²⁸ and a hypotensive episode after the first dose.¹⁴ No studies reported dose-dependent side effects. Compared to, for example, cyclosporine¹¹ or azathioprine,³⁵ where a sizable proportion experience short- and long-term side effects, omalizumab is a safe and well-tolerated, however expensive, alternative.

In conclusion, omalizumab appears to be a safe and well-tolerated treatment, with some clinical benefit in patients with severe recalcitrant AD. However, recommendation for use in clinical practice awaits evidence from larger randomized controlled trials, using a validated disease severity measure such as SCORAD, though a starting dose of 300 mg/month for no less than 6 months appears sufficient for the evaluation of treatment efficacy.

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