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Independent Clinical and Occupational Predictors of Moderate-to-Severe Occupational Asthma: A Retrospective Cross-Sectional Study from Iraq

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ABSTRACT: Occupational asthma (OA) remains one of the most frequent work-related respiratory disorders, yet the determinants of greater disease severity are poorly characterised, especially in low- and middle-income settings such as Iraq. This retrospective cross-sectional study, conducted at Baghdad Teaching Hospital between August and October 2025, examined 158 adults with a clinical diagnosis of OA to identify clinical and occupational predictors of moderate-to-severe disease. Severity was classified using spirometric indices (FEV1 and FEV1/FVC ratio) as mild or moderate-to-severe, without incorporating symptom control, exacerbation frequency, or treatment intensity. Disease was mild in 52.5% of patients, moderate in 41.8%, and severe in 5.7%, so nearly half (47.5%) of the cohort fell into the moderate-to-severe category. Bivariate comparisons showed that ever smoking, occupational rhinitis, and a longer duration of symptoms before diagnosis were significantly more common among moderate-to-severe cases. Multivariable logistic regression confirmed these three variables as independent predictors: ever smoking (adjusted odds ratio 2.91), occupational rhinitis (adjusted odds ratio 2.35), and each additional year of symptom duration (adjusted odds ratio 1.15). Age, sex, body mass index, atopic status, and the class of causative occupational agent showed no significant association with severity. The regression model showed good calibration (Hosmer-Lemeshow P = 0.667) and acceptable apparent discrimination (AUC 0.812), though this was not externally validated. These findings suggest that smoking behaviour, coexisting occupational rhinitis, and delayed diagnosis are the principal modifiable contributors to OA severity, underscoring the need for earlier workplace screening, smoking-cessation interventions, and proactive management of upper-airway disease among exposed workers in Iraq.

1. INTRODUCTION

Asthma is a chronic inflammatory disease of the airways that affects people of all ages and causes a large burden of symptoms, lost work, and health costs. The workplace is one of the few identifiable causes of adult asthma that can be prevented. Work-related asthma is an umbrella term covering occupational asthma (OA), caused by an agent encountered at work, and work-exacerbated asthma, in which pre-existing asthma worsens at work. OA itself is divided into sensitiser-induced disease, caused by high-molecular-weight (HMW) protein agents such as flour or animal proteins and by low-molecular-weight (LMW) chemicals such as isocyanates or persulphates, and irritant-induced disease, which follows heavy

exposure to irritant gases, fumes, or vapours [1,2]. Population studies suggest that 10% to 16% of adult asthma can be attributed to work [1,3,4].

Despite this large share, OA remains under-recognised. Many patients are treated for years as ordinary asthma because the occupational link is never explored, and the outcome of OA is generally worse than that of non-work-related asthma; prognosis depends mainly on how long and how heavily the worker remains exposed after symptoms begin [3]. National and international statements therefore advise asking every adult with new or worsening asthma about their job, and referring early when a work relationship is suspected [5,6]. Patients diagnosed after a long period of symptomatic exposure, or who already have reduced lung function at diagnosis, are less likely to reach remission [7].

Not all patients with OA are equally affected. Some remain mild, while others develop fixed airflow limitation and disability. Several factors have been linked to more severe or more persistent disease, including occupational rhinitis and its severity [8], the chemical class of the causal agent [4], cigarette smoking, which accelerates the loss of lung function and reduces the response to inhaled corticosteroids [9], obesity [10], and a long delay between the first symptoms and the diagnosis [7]. Most of this evidence originates from specialised centres in Europe and North America, where the diagnosis is confirmed by specific inhalation challenge. These findings cannot be transferred directly to settings where such testing is unavailable and where the diagnosis rests on a careful occupational history together with spirometry and peak-flow monitoring [3,6].

Iraq is one such setting. Asthma is common among Iraqi adults, with a reported prevalence of about 10.5% for physician-diagnosed disease, and smoking, allergic rhinitis, and dusty or fume-laden work are frequent [11]. Occupational exposure to vapours, gases, dust, and fumes is a recognised risk factor for new-onset adult asthma in population-based cohorts [12]. However, occupational health services in Iraq are limited, workplace surveillance schemes are largely absent, and no national register of OA exists. Very few Iraqi studies have described patients with OA, and to our knowledge none has examined the determinants of disease severity using a multivariable model.

This study was therefore designed to describe the severity distribution, the occupational exposure profile, and the lung function of adults with a clinical diagnosis of OA attending a tertiary respiratory clinic in Baghdad, and to identify the clinical and occupational factors independently associated with moderate-to-severe disease.

2. MATERIALS AND METHODS

2.1 Study design and setting

This was a retrospective cross-sectional study conducted at the Respiratory Diseases Department, Baghdad Teaching Hospital, Baghdad, Iraq, between August and October 2025. The source population consisted of adults who attended the department and were assessed for suspected occupational asthma during the study period. Patients were identified through medical records and departmental registries and were evaluated according to established clinical, occupational, and spirometric criteria for the diagnosis of OA. All eligible patients seen during the study period were included using consecutive sampling. The report follows the STROBE recommendations for cross-sectional studies.

2.2 Eligibility criteria

The inclusion criterion was adult patients (older than 18 years) with a clinical diagnosis of occupational asthma. OA was operationally distinguished from work-exacerbated asthma as follows: OA was defined as new-onset asthma, or an objectively documented substantial worsening of previously quiescent asthma, beginning after the start of the current job and showing a consistent temporal relationship with workplace exposure — namely, onset or worsening of respiratory symptoms during periods at work with improvement on days off, weekends, or holidays away from work. Patients whose asthma was already active and unchanged before starting the relevant job, and who did not show this work-related temporal pattern, were regarded as having pre-existing asthma without a demonstrable occupational component and were not enrolled as OA cases.

Patients were excluded if they had any of the following: (1) asthma-like symptoms due to another clear cause, such as chronic obstructive pulmonary disease or asthma-COPD overlap; (2) an active respiratory infection at the time of assessment; (3) significant structural lung disease or another chronic respiratory disorder that could confound interpretation; (4) known heart failure, particularly moderate-to-severe or unstable disease, or another significant cardiac disease that could confound respiratory symptoms or spirometry; or (5) pregnancy, because dyspnoea and reflux during pregnancy may overlap with work-related asthma symptoms and complicate interpretation.

2.3 Sample size and sampling

No formal sample size calculation was performed because all eligible patients seen during the study period were enrolled consecutively. Of 182 patients assessed for suspected OA, 24 were excluded because they did not meet the eligibility criteria, leaving 158 patients for the final analysis. Complete data were available for all primary study variables, so no case was excluded for missing data and no imputation was performed.

2.4 Data collection and definitions

Baseline sociodemographic data included age, sex, occupation, smoking status, and smoking pack-years. Clinical variables included occupational rhinitis, atopic status, and the duration of symptoms before diagnosis in years. BMI was calculated as weight in kilograms divided by height in metres squared. Smoking pack-years were calculated as (cigarettes per day ÷ 20) × number of years smoked. Patients were classified as never smokers or ever smokers, the latter including current and former smokers. Occupational rhinitis was diagnosed clinically on the basis of nasal symptoms related to workplace exposure that improved during time away from work and recurred after re-exposure. Atopic status was determined clinically and, when available, was supported by a history of allergic disease and by positive allergen sensitisation tests (skin prick testing or specific IgE). A detailed history was obtained from each patient, including the timing of symptoms in relation to work exposure, any change in symptoms during weekends or holidays, and associated upper-airway symptoms. Causal agents were grouped as HMW agents, LMW agents, or irritants according to the reported workplace exposure.

OA was diagnosed on the basis of asthma symptoms related to workplace exposure, a work-related pattern in the history, spirometry with bronchodilator reversibility, and peak-flow variability. Objective evidence of variable or reversible airflow obstruction was defined as an increase in FEV1 of at least 12% and 200 mL after an inhaled short-acting bronchodilator and/or a peak expiratory flow (PEF) variability of at least 20%, assessed from single-visit spirometry and patient-recorded peak-flow charts. Serial peak-flow monitoring with paired at-work and away-from-work recordings, and specific inhalation challenge — the reference-standard objective tests for OA in specialised centres [13] — were not performed in this cohort. This constraint, and the resulting possibility of diagnostic misclassification, is stated explicitly in the Limitations section.

2.5 Lung function measurement

Spirometry was performed with a calibrated spirometer by trained staff, following accepted technical standards for the performance and interpretation of routine lung function tests, and results were expressed as a percentage of predicted values [14]. FEV1, forced vital capacity (FVC), the FEV1/FVC ratio, PEF, and FEV1 reversibility after a short-acting bronchodilator were recorded.

2.6 Classification of asthma severity

Asthma severity was classified from spirometry using FEV1 and the FEV1/FVC ratio: mild (FEV1 >80% predicted, normal FEV1/FVC), moderate (FEV1 60–80% predicted, FEV1/FVC 75–80%), and severe (FEV1 <60% predicted, FEV1/FVC <75%). Discordant cases were classified according to the spirometric criterion (FEV1 or FEV1/FVC) indicating the more severe category, consistent with the original clinical records. For comparative analysis, patients were grouped as mild OA or moderate-to-severe OA; because FEV1 and FEV1/FVC define these categories, differences in these two parameters between groups are inherent to the classification and were treated as descriptive rather than as independent outcomes (Table 3). Current guidelines define asthma severity multidimensionally, incorporating symptom control, exacerbation history, and treatment requirements [15]. The spirometric approach was chosen for consistent application to retrospective records — a limitation acknowledged below, since treatment step, inhaled corticosteroid dose, symptom-control scores, exacerbation frequency, and adherence were not systematically recorded and could not be incorporated. Accordingly, "moderate-to-severe OA" throughout this manuscript denotes a spirometry-defined physiological classification, not the full multidimensional severity construct recommended by current guidelines.

2.7 Statistical analysis

Data were entered in Microsoft Excel 2019 and analysed with IBM SPSS Statistics version 26. Continuous variables were assessed for normality (Shapiro-Wilk test, histograms) and presented as mean ± SD (compared with the independent-samples t test) or median (interquartile range, IQR) (compared with the Mann-Whitney U test), as appropriate; categorical variables were presented as frequencies and percentages and compared with the Fisher exact test. Crude odds ratios (ORs) with 95%

confidence intervals (CIs) were obtained from univariable logistic regression. A multivariable binary logistic regression model was fitted with moderate-to-severe OA as the dependent variable, including variables associated with severity at $P < 0.10$ in univariable analysis (ever smoking, occupational rhinitis, symptom duration before diagnosis, agent class), plus age and BMI for clinical relevance. Agent class was entered as a three-level variable (HMW agents as reference, LMW agents, irritants), yielding two indicator terms so that irritant-induced disease could be assessed independently rather than collapsed into or excluded from the HMW/LMW comparison. With 75 events and seven covariate terms, the model had an events-per-variable ratio (EPV) of approximately 10.7, meeting the conventional EPV ≥10 rule proposed for stable logistic regression estimates; multicollinearity was checked and was not significant. Calibration was assessed with the Hosmer-Lemeshow test, explained variation with Nagelkerke R^2, and discrimination with the AUC. Sensitivity and specificity were calculated at a predicted-probability cut-off of 0.50, fixed a priori rather than derived from the data. As the AUC, sensitivity, specificity, and classification accuracy were derived in-sample, without internal (bootstrap or k-fold) or external validation, these represent apparent rather than validated predictive performance. All tests were two-sided, with $P < 0.05$ considered statistically significant.

3. RESULTS AND DISCUSSION

A total of 182 patients were assessed for suspected OA during the study period. Twenty-four patients did not meet the eligibility criteria and were excluded, leaving 158 patients for analysis. The mean age of the study group was approximately 35.6 years, and 43 patients (27.2%) were men.

Mild disease was the most common category (83 patients, 52.5%), followed by moderate disease (66 patients, 41.8%) and severe disease (9 patients, 5.7%). The moderate and severe categories were combined for the comparative analysis, giving 75 patients (47.5%) with moderate-to-severe OA.

The two severity groups were similar in age, sex, and BMI (Table 1). Ever smoking was almost twice as frequent in the moderate-to-severe group as in the mild group (61.3% versus 33.7%; crude OR 3.12, 95% CI 1.63–5.97; $P < 0.001$), and smokers in the moderate-to-severe group had a higher cumulative consumption (median 13 versus 7 pack-years; $P < 0.001$). Occupational rhinitis was also more common in moderate-to-severe disease (66.7% versus 42.2%; crude OR 2.74, 95% CI 1.43–5.25; $P = 0.002$). The median duration of symptoms before diagnosis was more than twice as long in the moderate-to-severe group (5 versus 2 years; $P < 0.001$). Atopy was similarly distributed between the groups (48.0% versus 45.8%; $P = 0.780$). LMW agents were relatively more frequent, and HMW agents relatively less frequent, in moderate-to-severe disease, but the overall distribution of agent categories was only borderline, not statistically significant ($P = 0.096$).

Table 1: Baseline clinical and occupational characteristics by occupational asthma severity

Variable	Mild (n = 83)	Moderate-to-severe (n = 75)	Crude OR (95% CI)	P value
Age (years), mean ± SD[a]	35.2 ± 8.5	36.1 ± 9.2	1.01 (0.98–1.05)	0.420
Male sex, n (%)	22 (26.5)	21 (28.0)	1.08 (0.53–2.17)	0.830
BMI (kg/m²), mean ± SD[a]	28.5 ± 4.2	29.1 ± 4.8	1.03 (0.97–1.10)	0.380
Smoking, never, n (%)	55 (66.3)	29 (38.7)	Reference	
Smoking, ever, n (%)	28 (33.7)	46 (61.3)	3.12 (1.63–5.97)	< 0.001
Pack-years, median (IQR)[b]	7 (4–11)	13 (9–19)	Not modelled[d]	< 0.001
Occupational rhinitis, n (%)	35 (42.2)	50 (66.7)	2.74 (1.43–5.25)	0.002
Atopy, n (%)	38 (45.8)	36 (48.0)	1.09 (0.58–2.04)	0.780
Exposure agent, n (%)[c]				**0.096***
High-molecular-weight agent	44 (53.0)	29 (38.7)	Reference	

Low-molecular-weight agent	26 (31.3)	36 (48.0)	2.10 (1.06–4.18)	—
Irritant exposure	13 (15.7)	10 (13.3)	1.17 (0.45–3.01)	—
Symptom duration, years, median (IQR)[b]	2 (1–5)	5 (3–9)	1.19 (1.08–1.31)	< 0.001

BMI, body mass index; CI, confidence interval; IQR, interquartile range; OR, odds ratio; SD, standard deviation. aT test. bMann-Whitney U test. cFisher exact test (3-group comparison). dNot modelled: collinear with smoking status.

Bakers formed the largest occupational group overall (40 patients, 25.3%) and were the most frequent group among patients with mild disease, whereas painters were relatively more frequent among patients with moderate-to-severe disease (26.7% versus 15.7%). Textile workers, laboratory workers, healthcare workers, and construction workers were distributed almost equally between the two groups. The overall distribution of occupational categories did not differ significantly between the severity groups (P = 0.300, Table 2).

Table 2: Occupational exposure profile by occupational asthma severity

Occupation	Mild (n = 83), n (%)	Moderate-to-severe (n = 75), n (%)	Total (N = 158), n (%)	P value*
Bakers	24 (28.9)	16 (21.3)	40 (25.3)	
Painters	13 (15.7)	20 (26.7)	33 (20.9)	
Textile workers	11 (13.3)	10 (13.3)	21 (13.3)	
Laboratory workers	9 (10.8)	7 (9.3)	16 (10.1)	
Healthcare workers	8 (9.6)	7 (9.3)	15 (9.5)	
Construction workers	7 (8.4)	6 (8.0)	13 (8.2)	
Others	11 (13.3)	9 (12.0)	20 (12.7)	
Total	**83 (100.0)**	**75 (100.0)**	**158 (100.0)**	**0.300**

**Fisher-Freeman-Halton exact test (7×2 comparison), reported at the variable header.*

As expected from the classification scheme, FEV1 percentage predicted and the FEV1/FVC ratio were lower in the moderate-to-severe group, and these differences are definitional. Among the parameters not used to define severity, FVC percentage predicted, FEV1 reversibility, and PEF were all significantly lower in moderate-to-severe disease (P < 0.001 for each, Table 3). The lower reversibility in the more severe group suggests a component of fixed airflow limitation.

Table 3: Respiratory function tests by occupational asthma severity

Variable	Mild (n = 83)	Moderate-to-severe (n = 75)	P value
Defining (classification) variables			
FEV1 (% predicted), mean ± SD[a]	85.5 ± 8.2	67.3 ± 8.0	—
FEV1/FVC ratio, mean ± SD[a]	0.78 ± 0.05	0.71 ± 0.06	—
Independent comparisons			
FVC (% predicted), mean ± SD	92.5 ± 9.1	87.9 ± 8.4	< 0.001
FEV1 reversibility (%), mean ± SD	15.2 ± 8.5	12.4 ± 7.8	< 0.001
PEF (L/s), mean ± SD	6.8 ± 1.2	5.3 ± 1.1	< 0.001

FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; PEF, peak expiratory flow; SD, standard deviation. aNot tested: used a priori to define severity groups (incorporation bias).

In the multivariable model, three variables remained independently associated with moderate-to-severe OA (Table 4). Ever smokers had almost three times the odds of moderate-to-severe disease compared with never smokers (aOR 2.91, 95% CI 1.44–5.86; P = 0.003). Occupational rhinitis more than doubled the odds (aOR 2.35, 95% CI 1.17–4.71; P = 0.016). Each additional year of symptoms before diagnosis increased the odds by about 15% (aOR 1.15, 95% CI 1.04–1.27; P = 0.005). The association with LMW agents became weaker and lost statistical significance after adjustment (aOR 1.74, 95% CI 0.83–3.64; P = 0.141). Compared with HMW agent exposure, irritant exposure was not significantly associated with moderate-to-severe OA (aOR 1.56, 95% CI 0.72–3.39; P = 0.259). Neither age nor BMI was independently associated with severity.

Table 4: Multivariable logistic regression analysis for predictors of moderate-to-severe occupational asthma

Predictor	Crude OR (95% CI)	Adjusted OR (95% CI)	P value*
Ever smoking (versus never)	3.12 (1.63–5.97)	2.91 (1.44–5.86)	0.003
Occupational rhinitis (versus no)	2.74 (1.43–5.25)	2.35 (1.17–4.71)	0.016
Symptom duration (per 1-year increase)	1.19 (1.08–1.31)	1.15 (1.04–1.27)	0.005
Low- versus high-molecular-weight agent	2.10 (1.06–4.18)	1.74 (0.83–3.64)	0.141
Irritant exposure (versus HMW)	1.17 (0.45–3.01)	1.56 (0.72–3.39)	0.259
Age (per 1-year increase)	1.01 (0.98–1.05)	1.02 (0.98–1.06)	0.348
BMI (per 1 kg/m² increase)	1.03 (0.97–1.10)	1.04 (0.98–1.11)	0.246

**P values for the adjusted model. Crude ORs from univariable logistic regression; adjusted ORs from the multivariable model (HMW = reference). Outcome: moderate-to-severe OA = 1, mild = 0. EPV = 75/7 = 10.7, meeting the ≥10 rule. BMI, body mass index; CI, confidence interval; HMW, high-molecular-weight; OR, odds ratio*

The model was well calibrated (Hosmer-Lemeshow chi-square 5.82, 8 degrees of freedom, P = 0.667) and explained approximately one third of the variation in severity (Nagelkerke R^2 0.342). Discrimination was acceptable, with an AUC of 0.812 (95% CI 0.746–0.878), a sensitivity of 78.7%, a specificity of 74.6%, and an overall classification accuracy of 76.6% (Table 5).

Table 5: Performance of the multivariable logistic regression model

Parameter	Value
Hosmer-Lemeshow chi-square	5.82
Degrees of freedom	8
Hosmer-Lemeshow P value	0.667
Nagelkerke R^2	0.342
Area under the ROC curve (AUC)	0.812
95% CI of the AUC	0.746–0.878
Sensitivity	78.7%
Specificity	74.6%
Overall classification accuracy	76.6%

AUC, area under the curve; CI, confidence interval; ROC, receiver operating characteristic. Cut-off = 0.50, fixed a priori rather than derived via Youden's index, avoiding added optimism from data-driven threshold selection. Values reflect apparent (derivation-sample) performance; no internal or external validation was performed, so estimates are likely optimistic.

4. DISCUSION

In this hospital-based study of 158 Iraqi adults with a clinical diagnosis of occupational asthma, 47.5% had moderate-to-severe disease. Three factors were independently associated with severity: ever smoking, occupational rhinitis, and a longer symptom-to-diagnosis interval. Age, sex, BMI, atopy, and the molecular-weight class of the causal agent were not.

Ever smokers had almost threefold odds of moderate-to-severe disease. This is biologically plausible: smoking accelerates FEV1 decline, promotes fixed airflow limitation, and reduces the response to inhaled corticosteroids, with part of this excess decline persisting after cessation [9], and it is also a major factor associated with asthma in Iraqi adults generally [11]. In occupational settings the picture is more complex, as smoking increases IgE sensitisation to some HMW agents while its effect on severity has been inconsistent across studies [4]. The higher pack-years in the moderate-to-severe group suggest a cumulative effect, although smoking may partly mark longer and heavier workplace exposure. Because moderate-to-severe disease was defined using FEV1, and smoking independently and dose-dependently lowers FEV1 irrespective of asthma [9], part of this association may reflect a direct spirometric effect of smoking on lung function rather than a specific effect on occupational-asthma severity. This definitional circularity could not be resolved analytically in the present dataset and is discussed below as the principal limitation of the study.

Occupational rhinitis was present in two thirds of moderate-to-severe cases and remained independent after adjustment. The upper and lower airways behave largely as one organ, and occupational rhinitis is two to three times more common than OA, usually precedes it, and is most closely linked to HMW agents [4,8]. Because rhinitis is often dismissed as a minor nuisance, it is a frequently missed warning sign: any worker with work-related nasal symptoms should be assessed for lower-airway disease, and treating rhinitis forms part of OA management [8].

Each additional year of symptom duration before diagnosis was associated with an approximately 15% higher odds of moderate-to-severe disease (median duration 5 years versus 2 years in mild disease). Because this study is cross-sectional, symptom duration before diagnosis was measured at the same time point as severity, so this association cannot be interpreted as evidence that diagnostic delay causes greater severity. Reverse or bidirectional explanations cannot be excluded: patients with more severe or more disabling symptoms may seek medical attention sooner, while patients with milder, more tolerable symptoms may remain undiagnosed for longer, which would generate the same cross-sectional pattern without a causal delay effect. This is consistent with prognostic data showing that the duration and level of exposure after symptom onset are the main determinants of outcome [3], and that shorter symptomatic exposure with better lung function predicts remission [7]. This is the most readily modifiable factor: because there is no workplace asthma surveillance scheme in Iraq, systematically asking every adult with new or worsening asthma about their job, as recommended internationally, would shorten the diagnostic pathway at little or no cost [5,6].

There was a crude association between LMW agents and the moderate-to-severe group, but this weakened after adjustment. The evidence on agent class and severity is mixed: LMW agents are heterogeneous and several behave like HMW agents, so pooling them may be misleading, and reactive LMW chemicals can also injure the airway epithelium directly [4,16]. Our occupational profile matches international patterns: bakers were the largest group, reflecting flour dust and enzymes as a leading cause [17]; painters were relatively more frequent in severe disease, consistent with isocyanates remaining a major cause of OA [18]; and healthcare and laboratory workers together formed about one fifth of the sample, in line with disinfectants and cleaning products as established causes of work-related asthma and rhinitis, particularly high-level, quaternary-ammonium, or chlorine-based agents [19-21]. Such exposure is probably under-recorded in Iraqi hospitals, where disinfection practices intensified after the COVID-19 pandemic. Job distribution did not differ between severity groups ($P = 0.300$), most likely because individual occupational categories were small.

Other variables examined were not related to severity. BMI was slightly higher in moderate-to-severe disease but did not independently correlate with it, despite reported associations between asthma control and BMI in other cohorts [10]. Atopy was similarly distributed between groups, supporting the concept that atopy predicts sensitisation to HMW agents rather than disease severity [4]. Women outnumbered men in this sample compared with many European series; this more likely reflects

local referral patterns than true differences in risk, and sex was not correlated with severity. FEV1 and FEV1/FVC were used to define the severity categories and were therefore not formally tested, while the remaining lung-function parameters (FVC percentage predicted, PEF, FEV1 reversibility) were all lower in moderate-to-severe disease. The lower reversibility observed in the more severe group is of interest with respect to airway remodelling and partially fixed obstruction, which can determine residual airway impairment once a worker is no longer exposed to the causative job [22], and because a late diagnosis may reduce the potential benefit of intervention.

The practical implications for Iraq are straightforward. A work history should be a routine part of the assessment of every adult with asthma. Spirometry combined with a short questionnaire on work-related nasal and chest symptoms would benefit workers in bakeries, paint shops, textile plants, laboratories, and hospitals, since occupational exposure is a recognised trigger for the onset of adult asthma [12]. Smoking-cessation support should be actively offered, as smoking was the strongest determinant identified here. New cases can be reduced through workplace exposure control, such as local exhaust ventilation, closed handling of flour and paints, and safer disinfection practices [3]. Finally, establishing a simple national notification process would make the true magnitude of the problem evident.

4.1 Strengths and limitations

Strengths include consecutive enrolment of all eligible patients, complete data for the key study variables, and a multivariable model with an adequate events-per-variable ratio together with formal calibration and discrimination testing. To our knowledge, this is the first Iraqi study to examine determinants of OA severity rather than simply describe cases. Several limitations should be considered, foremost among them a definitional circularity affecting the principal finding on smoking. Because moderate-to-severe disease was defined using FEV1, and cigarette smoking independently and dose-dependently lowers FEV1 irrespective of asthma, part of the observed association between smoking and moderate-to-severe OA may reflect a direct spirometric effect of smoking on lung function rather than a smoking-specific effect on occupational-asthma severity. This circularity could not be resolved analytically within the present retrospective dataset; a sensitivity analysis using an alternative, non-spirometric severity definition (for example, based on treatment step or symptom control) was not possible with the data available and should be undertaken in future prospective studies. The retrospective cross-sectional design also precludes causal inference more generally, particularly for the symptom-duration association. Single-centre recruitment in Baghdad raises the possibility of referral bias, limiting generalisability. Severity itself was defined by spirometry alone, capturing only the physiological dimension of the multidimensional construct used in current guidelines. Diagnosis relied on history, reversibility, and peak-flow variability without specific inhalation challenge, methacholine testing, exhaled nitric oxide, or complete serology, permitting some misclassification of OA and of agent class; exposure itself was classified without quantitative measurement or a job-exposure matrix. Smoking status and symptom duration were self-reported and subject to recall bias, and treatment and adherence data were unavailable. Only nine patients had severe disease, precluding separate analysis of this subgroup, and residual confounding — for example, from air pollution, socioeconomic status, rhinosinusitis, or gastro-oesophageal reflux — cannot be excluded. Finally, model discrimination and classification statistics were derived in-sample, without internal (for example, bootstrap) or external validation, and should be interpreted as apparent rather than validated predictive performance.

5. CONCLUSION

Nearly half of the adults with a clinical diagnosis of occupational asthma seen at a tertiary respiratory clinic in Baghdad had moderate-to-severe disease, as graded by a spirometry-based severity definition in this cross-sectional, single-centre sample; these design features limit causal inference, and the findings should be regarded as hypothesis-generating rather than confirmatory. Ever smoking, occupational rhinitis, and a longer interval between the first symptoms and the diagnosis were independently associated with moderate-to-severe disease, while age, sex, BMI, atopy, and agent class were not. Assuming these associations are causal, all three predictors are potentially modifiable, although part of the smoking association may additionally reflect the spirometric definition of the outcome rather than a specific effect on OA severity. Earlier recognition of work-related asthma, systematic assessment and treatment of work-related nasal symptoms, and active smoking-cessation support, combined with better workplace exposure control, represent the most realistic strategies to reduce the burden of severe occupational asthma among Iraqi workers. Prospective multicentre studies using guideline-based, non-spirometric severity definitions, objective exposure assessment, and internal or external model validation are needed to confirm these findings.

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AUTHOR CONTRIBUTIONS

NFA: Conceptualization, methodology, data collection, statistical analysis, and writing – original draft preparation. HMA: Conceptualization, study supervision, methodology validation, and critical review of the manuscript. SJA: Study supervision, data interpretation, manuscript editing, and final approval of the submission. All authors read and approved the final manuscript.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

ETHICS STATEMENT

Ethical approval was granted by the Arab Board of Health Specializations at the Iraqi Ministry of Health (Protocol No. 227; approved 15 June 2025). Because anonymised records were used retrospectively, the requirement for individual informed consent was waived. All data were coded before analysis and no personal identifiers were retained. The study was conducted in accordance with the Declaration of Helsinki.

DATA AVAILABILITY STATEMENT

Lorem ipsum dolor sit amet, consectetur adipiscing elit. Data supporting the findings of this study are available from the corresponding author upon reasonable request.

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