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CASE STUDY

APPLIED PSYCHOLOGY

Idiopathic Environmental Intolerance (Multiple Chemical Sensitivity): A Psychiatric Case Report with Psychodynamic Formulation

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ABSTRACT

Patients labelled with "Multiple Chemical Sensitivity" (MCS) frequently present with multi-system complaints, high avoidance, and substantial psychiatric burden. Etiology remains disputed. Psychiatric care must validate suffering while remaining agnostic about toxic causality. A 67-year-old woman with lifelong somatic vulnerability, chronic tobacco use, and extensive occupational exposure to pesticides and solvents reported odor-linked autonomic surges, fatigue, pain, and cognitive fog. External physicians documented GSTM1 null genotype; abnormal red-ox/mitochondrial markers (elevated lactate–pyruvate ratio, high SOD, low GPx), disturbed vitamin-D metabolism (low 25-OH with high 1,25-di-OH), and MRI/MRS (2013) interpreted by neuroradiology as diffuse toxic leukoencephalopathy "compatible with chronic solvent exposure." Autonomic testing reproduced paroxysmal tachycardia. Psychiatric evaluation identified depressive and anxiety symptoms, illness-focused ruminations, high environmental vigilance, and moderate structural vulnerabilities in affect regulation and mentalization. Psychodynamic counseling (OPD-guided focus on self-esteem regulation, affect tolerance, and relational patterns), paced functional restoration, and liaison with medical care. No etiologic assertions were made. Improved affect regulation and role function with a reduced avoidance radius; persistent sensitivity to strong odors. Psychodynamic treatment can reduce distress and disability in IEI/MCS-labelled presentations while remaining causally neutral. Transparent attribution of external medical findings and CARE-standard reporting enable constructive interdisciplinary dialogue.

Keywords: Idiopathic Environmental Intolerance, Multiple Chemical Sensitivity, Psychodynamic counseling, OPD-2, Somatic symptom–related distress, Health anxiety

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Multiple Chemical Sensitivity (MCS) denotes patient-reported symptom exacerbations attributed to low-level chemical exposures. Major allergy and occupational bodies often prefer the term *Idiopathic Environmental Intolerance* (IEI) to emphasize uncertain causation. Presentations are clinically heterogeneous and carry high psychiatric comorbidity and functional burden. This report provides a psychiatry-centered, psychodynamic formulation and clinical course, while attributing medical findings to the original physician documents without causal endorsement. The goal is to demonstrate a pragmatic, interdisciplinary frame that neither dismisses symptoms nor over-interprets mechanisms. Patient information for the case study are as follows—

- **Demographics:** 67-year-old woman.
- **Early vulnerability:** Marked low weight in youth; recurrent food/environmental intolerances; repeated hospitalizations; persistent physical frailty without anorexia history.
- **Occupational history:** 2000–2012 management role in agricultural supply with chronic exposure to pesticides (including organophosphates such as dichlorvos) and pyrethroids; at least two severe indoor fumigations reportedly at five-fold dose with early re-entry.
- **Lifestyle:** ~10 cigarettes/day; BMI 17–19 kg/m²; deconditioned.
- **Presenting concerns:** Odor-linked tachycardia and blood-pressure surges, dizziness, fatigue, cognitive fog, paresthesias, skin microbleeds/erythroderma after solvent contact; high environmental vigilance; fear of decline; social withdrawal.

De-identification: Names and identifiers removed. All medical data reproduced verbatim from physician reports; no independent re-analysis of images/specimens.

Time-line

Date	Event / Exposure	Symptoms / Course	Psychiatric status / intervention	Documentation
2000–2012	Agricultural chemicals at work; two overdosed foggings with next-day re-entry	Progressive fatigue, headaches, neurologic complaints	Growing health anxiety and avoidance	Toxicology expert opinion; occupational history
2009	Depot neuroleptic during psychiatry-led admission	Acute dystonic reaction; emergency care	Heightened mistrust of care	Psychiatric record
10/2011	Lab panel	L/P ratio 76; pyruvate low; LDH pattern change	Bodily vigilance rises	Lab report
04/2012	Lab panel	25-OH vitamin D 14.9 µg/l (low) with 1,25-di-OH 69.8 ng/l (high); SOD high; GPx low; M2-PK elevated; Mg low	Persistent fatigue; avoidance expands	Lab report
04/2013	Brain MRI + MRS	Per report: patchy confluent WM FLAIR hyperintensities; ↓NAA; impression “diffuse toxic leukoencephalopathy compatible with solvent exposure”	Anxiety about brain injury	Neuroradiology report

Date	Event / Exposure	Symptoms / Course	Psychiatric status / intervention	Documentation
2014	Nerve conduction	Mild length-dependent mixed sensory-motor axonopathy	Worry about progressive neuropathy	Neurophysiology report
2024	Chemistry/CBC	Cholesterol 283 mg/dl; platelets 441×10³/µl; RDW 15.2%	Liaison for cardiovascular risk	Lab report
2025	Psychiatric intake	Odor-linked autonomic surges; depressive/anxious symptoms; high avoidance	OPD-guided psychodynamic therapy begins	Clinical notes

Clinical findings (psychiatry)

- **Mental status at intake:** Cooperative; fatigued; constricted affect; anxious apprehension about health; illness-focused ruminations; intact reality testing; partial insight; sleep fragmentation.
- **Risk:** No current suicidal ideation, intent, or plan.
- **Function:** Reduced activities outside home; avoidance of public spaces and perceived triggers; strained relationships.
- **Relational/psychodynamic:** Help-seeking/help-rejecting oscillation; fear of dismissal; validation–withdrawal cycles; therapist counterpressure to "explain" somatic findings managed with a neutral, affiliative stance.

Diagnostic assessment (attempted)

Psychiatric/psychodynamic formulation (OPD-2)

- **Axis I (illness experience / prerequisites):** High illness preoccupation with fluctuating trust; prerequisites for psychotherapy present.
- **Axis II (relations):** Ambivalent attachment patterns with alternating proximity seeking and distancing; sensitivity to invalidation.
- **Axis III (conflicts):** Self-esteem regulation vs. perfectionistic strivings; autonomy vs. dependency; care vs. self-neglect.
- **Axis IV (structure):** Moderate vulnerabilities in affect regulation and differentiation of bodily signals; mentalization decreases under stress; identity largely coherent.
- **Axis V (syndrome level):** Depressive and anxiety-spectrum features; somatic symptom–related distress; sleep disturbance.

Attributed medical findings (non-endorsing, verbatim sourcing)

- **Genetics:** Homozygous GSTM1 deletion reported.

- **Imaging:** 04/2013 MRI/MRS described above with neuroradiology impression of diffuse toxic leukoencephalopathy "compatible with chronic solvent exposure."
- **Neurophysiology:** 2014 mild length-dependent mixed sensory-motor axonopathy.
- **Autonomic:** Tilt-table reproduced paroxysmal tachycardia; baseline ECG/echo unremarkable.
- **Selected laboratory patterns**
  - 10/2011–04/2012: Elevated lactate–pyruvate ratio (76), LDH-isoenzyme shift (↓LDH-2, ↑LDH-4/5), low 25-OH vitamin D with elevated 1,25-di-OH vitamin D, high SOD, low GPx, elevated M2-PK, low magnesium.
  - 2024: Hypercholesterolemia, thrombocytosis, RDW 15.2%; other chemistries within reference.

IEI/MCS criteria mapping (descriptive)

Criterion	Case facts	Source
Chronicity	Multiyear course with persistent symptoms	Patient history; records
Reproducible low-level triggers	Odor-linked tachycardia/BP spikes	Patient report; tilt-table episode
Multiple unrelated chemicals	Workplace pesticides/solvents; household odors	Occupational history; patient report
Multi-system involvement	Autonomic, neurological, dermatologic, fatigue/pain	Mixed records
Improvement with avoidance	Partial relief in low-odor settings	Patient report

Therapeutic interventions (psychiatry, in-patient)

- **Modality/frequency:** Psychotherapeutic-interventions, once weekly.
- **Focus:** Self-esteem regulation; affect tolerance; cognitive reappraisal of illness beliefs without invalidation; reduction of catastrophic ideation; relational patterns driving validation–withdrawal cycles.
- **Behavioral program:** Pacing and graded re-engagement with valued activities and low-odor environments; sleep hygiene; gentle conditioning.
- **Liaison:** Communication with primary care on cardiometabolic risk, underweight/sarcopenia, and smoking cessation offers.
- **Medications:** External physicians documented mirtazapine 15 mg/day with moderate mood/energy benefit; low-dose bisoprolol for episodic hypertension; past neuroleptic exposure in 2009 caused acute dystonia.

Follow-up and outcomes

- **Objective:** Attendance stable; expanded time outside home; decreased unscheduled medical contacts.

- **Subjective:** Lower catastrophic ideation; improved affect regulation; reduced avoidance radius; persistent sensitivity in high-odor contexts.
- **Adverse events:** None reported in counseling course.

Patient perspective

"I have learned to separate what I feel from what I fear. Being heard without being told it is 'all in my head' helped me try small steps again. Strong smells still set me off, but I no longer plan my whole day around them. I feel less alone and more in control of my reactions."

DISCUSSION

This case shows that psychiatry can offer tangible benefit in IEI/MCS-labelled presentations without endorsing disputed etiologies. The stance balances validation and uncertainty, uses OPD to select a focused psychodynamic perspective, and targets functional restoration. Smoking despite odor sensitivity illustrates self-regulatory use of substances under stress and complicates simplistic exposure narratives. Structural vulnerabilities in affect regulation and mentalization plausibly amplify symptom attention and avoidance. The therapeutic alliance depended on transparent attribution of medical data, a neutral stance toward causality, and steady work on avoidance, mood, and relationships. Premature psychiatric labeling and the routine discounting of patients' reports function as iatrogenic stressors. They shift attention inward, amplify vigilance to bodily signals, and increase safety-seeking and avoidance. This dynamic is documented across contested illnesses where patients' testimony is downgraded in credibility (cf. epistemic injustice), producing demoralization and disengagement from care. In this case, repeated non-validation plausibly increased illness preoccupation and social withdrawal, independent of any toxic etiology. (Carel & Kidd, 2014; Blease *et al.* 2017). Diagnostic overshadowing compounds the harm: once a psychiatric label is applied, new physical complaints are more likely to be attributed to that label, delaying appropriate medical evaluation and reinforcing the patient's belief that only self-surveillance protects against further injury. Consequently, continued invalidation supports somatic salience and increases feelings of isolation. What emerges is a vicious cycle of psychological distress and symptom-vigilance (Eisenberger, 2012; Kross *et al.* 2011; Holt-Lunstad *et al.* 2010; Naito *et al.* 2023). The psychiatric dimensions require careful consideration without dismissive reductionism. Simon (1994) documented high prevalence of psychiatric symptoms in MCS patients, finding correlations between psychological factors and symptoms that exceed those with measurable chemical exposures. However, the clinical perspective developed by Sparks *et al.* (1994a, 1994b) emphasized the need for systematic case definitions and evidence-based evaluation methods while addressing diagnostic testing approaches and multidisciplinary care considerations, rather than premature psychological attribution.

Critical perspectives, however, must also be acknowledged. Barrett and Gots (1998) argued that environmental illness attributions often lack scientific validation and that psychological factors may play more significant roles than chemical exposure in symptom manifestation. Reid (1999) similarly questioned whether environmental factors truly cause MCS symptoms, proposing that psychological mechanisms may better explain diverse symptom presentations. These perspectives highlight the ongoing scientific debate while underscoring the importance of maintaining clinical neutrality Communication itself modulates symptoms and thus, a validating and tolerant stance can significantly reduce distress, regardless of

disputed medical mechanisms. Across sessions, shifts from invalidation to consistent acknowledgment of symptom burden coincided with reduced catastrophic ideation and a smaller avoidance radius, despite unchanged sensitivity to strong odors. This pattern supports the view that prior non-validation and social withdrawal were maintaining factors in overall suffering rather than mere epiphenomena. The complexity of this case is underscored by emerging research on stress-related pathophysiology and environmental sensitivity mechanisms. Algamal *et al.* (2021) demonstrated that repeated unpredictable stress combined with social isolation induces chronic HPA axis dysfunction and persistent abnormal fear memory, providing neurobiological context for how environmental stressors might contribute to lasting symptom patterns through dysregulated stress response systems. This aligns with Juruena's (2014) findings that early-life stress serves as a critical trigger for recurrent adulthood depression through persistent HPA axis dysregulation, suggesting that childhood vulnerabilities may predispose individuals to both psychiatric symptoms and heightened environmental sensitivity later in life.

The patient's documented GSTM1 null genotype represents a significant genetic vulnerability that warrants careful consideration. Baranova *et al.* (1997) established that glutathione S-transferase M1 gene polymorphisms significantly influence susceptibility to environmental health conditions, while Bolt and Thier (2006) comprehensively demonstrated the clinical relevance of GSTT1 and GSTM1 deletion polymorphisms in individual susceptibility to environmental toxin exposure and metabolic stress. Rebbeck (1997) further confirmed through molecular epidemiological analysis that these genotypes serve as significant modifiers of health vulnerability, with null genotypes conferring increased risk due to impaired detoxification capacity. These genetic findings provide biological plausibility for individual differences in chemical sensitivity without necessarily validating specific causal mechanisms.

The neurobiological basis for multiple chemical sensitivity still continues to evolve to this day, with Molot *et al.* (2023) presenting updated neuroscience evidence supporting MCS as a legitimate medical condition through neurobiological mechanisms including central sensitization and neuroinflammation. Sorg (1999) earlier proposed neural sensitization as a mechanistic explanation, suggesting that neuroplasticity changes following chemical exposure could create persistent hypersensitivity through central nervous system alterations. Winder (2002) provided a comprehensive toxicological review of proposed mechanisms, examining hypotheses from immune dysfunction to neurological sensitization, while Zucco and Doty (2021) synthesized current brain science research, further integrating neuroimaging findings and cognitive processing alterations to provide neurobiological foundations for chemical sensitivity syndromes.

The clinical heterogeneity observed in this case reflects broader patterns documented in literature. Hojo *et al.* (2018) characterized idiopathic environmental intolerance through clinical assessments, identifying specific symptom patterns that distinguish chemically sensitive individuals from healthy controls, while Lago Blanco *et al.* (2016) developed assessment frameworks integrating both physical symptoms and psychological factors in comprehensive patient evaluation. The high psychiatric comorbidity observed aligns with Bornschein *et al.*'s (2002) findings of significant psychiatric and somatic disorders in environmental patients, and Bornschein et al.'s (2000) documentation of psychiatric morbidity patterns that often preceded chemical exposure complaints. As for the assessment of biographical factors, Algamal *et al.* (2021) demonstrated that repeated unpredictable stress combined with social isolation induces chronic HPA axis dysfunction and persistent abnormal fear memory, providing neurobiological context for how environmental stressors might contribute to lasting symptom patterns through dysregulated stress response systems. This aligns with Juruena's (2014) findings that early-life stress serves as a

critical trigger for recurrent adulthood depression through persistent HPA axis dysregulation, suggesting that childhood vulnerabilities may predispose individuals to both psychiatric symptoms and heightened environmental sensitivity later in life.

Clinical implications

1. Attribute external medical findings precisely and avoid causal leaps.
2. Treat avoidance and health anxiety with psychodynamic and graded behavioral methods.
3. Track function and distress longitudinally; improvement does not require etiologic certainty.
4. Address lifestyle risks via liaison care.
5. Provide testimonial validation without causal endorsement; separate symptom reality from etiologic certainty.

Limitations

Single-patient report; reliance on external documents; no blinded re-read of neuroimaging; incomplete autonomic profiling.

CONCLUSION

Psychodynamic counseling within an attribution-transparent, interdisciplinary frame reduced distress and disability in this IEI/MCS-labelled presentation. Such cases are publishable when reported to CARE standards and framed without overinterpretation.

Transparency Remark

Preparation of this manuscript was supported by the use of GPT-5 (OpenAI). The system was used solely to assist in aligning the case report with the CARE reporting standard, improving formatting and structural clarity. All clinical data, laboratory values, imaging findings, and references derive from the actual patient record and published sources and were not generated, altered, or fabricated by the system. The case is real, and the author remains fully responsible for the accuracy, integrity, and interpretation of all content.

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Appendix: Data provenance and tables

(A) Source documents (redacted identifiers)

Type	Date	Issuer	Key content
Lab report	10/2011	Clinical laboratory	L/P 76; pyruvate low; LDH isoenzyme shift
Lab report	04/2012	Clinical laboratory	25-OH Vit D 14.9 µg/l; 1,25-di-OH 69.8 ng/l; GPx 5.0 U/ml; SOD 189 U/ml; M2-PK 17.2 E/ml; Mg 1.3 mMol/l
MRI/MRS report	04/2013	Neuroradiology	Patchy confluent WM FLAIR hyperintensities; ↓NAA; impression "diffuse toxic leukoencephalopathy compatible with solvent exposure"
Neurophysiology	2014	EMG/NCS lab	Mild length-dependent mixed sensory-motor axonopathy
Lab report	2024	Clinical laboratory	CBC/Chem incl. cholesterol 283 mg/dl; platelets 441×10³/µl; RDW 15.2%
Genetics	—	Molecular lab	GSTM1 null genotype

(B) Selected laboratory values (verbatim)

Analyte	Date	Value	Reference
Lactate–pyruvate ratio	10/2011	76	<20
Pyruvic acid	10/2011	1.6 mg/l	3.6–5.9 mg/l
25-OH vitamin D	10/2011	11.1 µg/l	30–100 µg/l
25-OH vitamin D	04/2012	14.9 µg/l	30–100 µg/l
1,25-di-OH vitamin D	04/2012	69.8 ng/l	20–65.5 ng/l
M2-pyruvate kinase	04/2012	17.2 E/ml	<15.0 E/ml
GPx	04/2012	5.0 U/ml	>6.0 U/ml
SOD	04/2012	189 U/ml	120–150 U/ml
Magnesium (ionized, serum)	10/2011	1.3 mMol/l	1.5–2.0 mMol/l
Cholesterol	2024	283 mg/dl	≤200 mg/dl
Platelets	2024	441×10³/µl	176–391
RDW	2024	15.2%	<15%