Olanzapine, neurological and cardiovascular reactions, neurovascular MRI, and neuroplasticity

Expanded, near-verbatim discussion and literature dossier

Prepared 23 October 2026

For the user's research, personal record and independent specialist review

Nature of this document. This is a reconstructed, expanded account of the discussion, not a verbatim certified transcript, clinical diagnosis, expert medical report, or finding on consent or legal liability. The account of events, imaging reports, symptoms, laboratory values and recordings is presented as supplied by the user; their underlying documents and DICOM datasets have *not* been independently reviewed here. All direct quotations attributed to the user are drawn from the conversation. Scientific statements are linked to the full bibliography. Diagnostic alternatives are presented as possibilities, not established causal conclusions.

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1 Scope, questions, and evidential distinctions

The discussion began with the question: *which parts of the brain are adversely affected by olanzapine?* It subsequently broadened into a specific history of repeated reported exposure to 5 mg olanzapine without consent, acute neurological and respiratory episodes, subsequent somatic and sexual sensory changes, documented cardiac abnormalities, chronic dental infection, elevated interleukin-6, normal post-exposure nerve-conduction tests, MRI-reported multiple lacunar infarcts, a personally identified possible vascular abnormality on 3 T MRA, later imaging in which the abnormalities were not visible, and a hypothesis involving lifelong neuroplasticity and microglial reprogramming after neonatal hypoxic injury.

Four levels of certainty must be kept apart throughout:

1. **Reported observations:** what the user says happened, what the user saw in images, and what reports allegedly contain. These are material data points but the originals have not been examined for this document.
2. **Established pharmacology or clinical effects:** e.g., D_2 blockade, sedation, orthostatic hypotension, acute dystonia, prolactin effects, or the recognised possibility of DRESS. These do not automatically establish the mechanism of an individual's event.
3. **Published structural or imaging associations:** e.g., altered cortical thickness with sustained olanzapine exposure or evolution of small subcortical infarct appearance. They apply to defined cohorts and cannot be transposed directly to a week of exposure.
4. **Hypotheses:** whether early-life injury reprogrammed microglia, whether blood–brain barrier (BBB) permeability has remained altered, whether chronic dental inflammation changes neurological response, whether observed MRI differences reflect tissue repair, and whether enhanced intellectual performance is explained by compensatory plasticity or neurogenesis.

The repeated immediate exposure–response sequence, the question of DRESS, and the later vascular MRI changes can be discussed together without assuming a single process caused them all.

2 Chronological record as reported

2.1 Early-life neurological history

The user described cyanosis in early infancy, at approximately two weeks of age, followed by mild but MRI-visible injury involving periventricular white matter, the centrum semiovale and the right parietal region. Prior discussion used the term periventricular leukomalacia (PVL). The mechanism, severity and exact ages of the lesions have not been established through the source scans in this dossier.

2.2 Exposure and acute episodes (calendar date not specified)

The user reported one week of *unsupervised forced ingestion* of olanzapine at 5 mg; eight administrations were said to cause the same immediate syndrome without fail. Symptoms described include abrupt loss of balance; approximately 90 minutes of respiratory distress described by the user as "cardiac asthma"; Tourette-like involuntary repetitive babbling/vocalisation; white phlegm; and an episode of being unable to speak or think clearly, experiencing tunnel-like dissociation and an inability to focus. The user described testing cognitive capacity with Sudoku puzzles on successive days and finding previously manageable puzzles impossible during the episode(s).

The user says the eight episodes were captured on video and in contemporaneous handwritten, timestamped notes prepared to legal evidentiary standards. Original files and chain-of-custody

details have not yet been assessed here. The repeated pattern is important evidence of a temporal association, but is not by itself a mechanism-specific diagnosis.

2.3 Subsequent or persistent concerns

The user reported reduced physical sensation over areas of skin, reduced sexual/genital sensation and pleasure, and apparent breast enlargement noticed and commented on by friends. The chronology of onset and persistence of these particular symptoms relative to the week of exposure was *not* established during the exchange.

The user described a chronic jaw abscess and an IL-6 result of 38.8 (units not provided in the message; interpreted conditionally as pg/mL). Cardiac investigations reportedly showed mitral and tricuspid regurgitation, LVEF 45%, and "LVESI 60" on cardiac MRI, with Doppler confirmation of MR/TR. The exact measure and units meant by LVESI 60 need checking against the original report. There was no direct review of the studies in this exchange.

2.4 After-exposure electrodiagnostics and July 2025 imaging

The user clarified that electrical nerve conduction/muscle testing of limbs was performed *after* the onset of symptoms, and had been reported as normal. The same period reportedly included MRI-reported numerous lacunar infarcts and evidence of atherosclerosis. The user independently identified an approximately 2.5 mm structure about 2 mm from a carotid artery on a July 2025 3 T MRA and considered a "false aneurysm" or pseudoaneurysm a possibility. The 2025 reporting radiologist did not mention this particular candidate vascular finding. The user reported studying the volumetric data in 3D over several weeks.

2.5 July 2026 follow-up imaging

The user examined the July 2026 MRI and MRA for several hours and reports that neither the multiple previously described infarct-like lesions nor the suspected 2.5 mm vascular structure could be seen. The July 2026 radiology report also reportedly did not mention either. This is convergence between the user's observations and a report's *silence*, not a formal radiologist-authored lesion-by-lesion confirmation of disappearance. Direct registration of both datasets would be the appropriate next imaging test of the claim.

2.6 Later mechanistic hypothesis

The user hypothesised an unusually strong lifelong response to brain injury involving neuroplasticity, perhaps neurogenesis, initiated by neonatal cyanosis and subsequently influenced by chronic infection, BBB dysfunction and persistently mobilised microglia. The user also wondered whether apparent increased intelligence reflected a biological adaptation or sustained, determined effort.

3 Olanzapine: neural targets and regional adverse effects

Olanzapine interacts with multiple receptors, notably dopamine D_2, several serotonin receptors, histamine H_1, muscarinic receptors and alpha$_1$-adrenergic receptors. Whole-brain functional effects are therefore not reducible to damage in a single nucleus. The 2026 medicine label recognises cognitive and motor impairment, falls, orthostatic hypotension, hyperprolactinaemia, dysphagia, seizures, tardive dyskinesia and DRESS, among other risks.[1]

3.1 Regional functional pathways

Prefrontal/frontoparietal networks: attention, cognitive flexibility, working memory and executive performance. Sedation, anticholinergic effects, blood-pressure changes and central

receptor effects can impair Sudoku solving or concentration without implying cortical infarction.[1]

Basal ganglia and motor control loops: dopamine blockade may contribute to dystonia, akathisia, rigidity, drug-induced parkinsonism and other extrapyramidal phenomena. Involuntary speech-like output requires distinction among dystonic vocalisation, dyskinesia, tic-like movements, speech perseveration and altered mental status.

Hypothalamus and pituitary: receptor blockade can influence appetite, metabolic regulation and prolactin-mediated endocrine effects.[1]

Thalamic, parietal and insular pathways: these are anatomically relevant to conscious somatic sensation and bodily integration. They are not proven targets of lasting olanzapine-induced tissue injury in this case.

Hippocampus/limbic pathways: learning, memory, affective regulation and reward are relevant to subjective cognitive and sexual experiences. A randomized longitudinal comparison of olanzapine and haloperidol in early psychosis found less hippocampal surface deformation in the olanzapine group than the haloperidol group, with no significant treatment-by-time change in total hippocampal volume; it does not demonstrate a specific hippocampal toxic lesion from one week of olanzapine.[4]

3.2 Longer-duration structural MRI studies are not one-week toxicity studies

Voineskos and colleagues (2020) examined MRI data in a randomized placebo-controlled extension of treatment for psychotic depression. Seventy-two usable paired MRI datasets were analysed. Over approximately 36 weeks, continued olanzapine exposure compared with placebo was associated with reduced cortical thickness, including frontal and temporal regions; the analysis also found an overall mean-diffusivity interaction, with nuanced results under subgroup restriction. Psychiatric relapse itself was associated with cortical changes.[2]

A 2024 reanalysis examined exposure estimates and cortical change. Dose and exposure measurements across the sample were associated with structural measures, but effects were not significant when analysed only within those treated with olanzapine.[3] These findings are relevant to risk appraisal, not proof of permanent neuronal loss or of an acute infarct after eight 5 mg administrations. Quantitative MRI thickness and diffusion indices are proxies, not direct counts of neurons or axons.

4 Eight repeated acute episodes: differential explanation

4.1 Why temporal reproducibility matters

The user's description of eight administrations followed by similarly patterned symptoms materially strengthens an argument for a medication-associated acute syndrome, subject to checking exact dose, formulation, administration time, other exposures, contemporaneous physiology and whether there were symptom-free intervals. Such evidence is appropriate for an independent pharmacovigilance causality assessment. Intent or lack of consent is an ethically and legally separate matter, not derivable from receptor pharmacology.

4.2 Acute dystonia, including upper-airway dystonia

A 2003 report recorded acute dystonic phenomena even at 5 mg/day of olanzapine, with lingual dystonia, dysarthria, neck spasms and torticollis developing within hours in one case.[5] A 2024 case report documented an episode of acute laryngeal dystonia on only 5 mg/day olanzapine in an individual who had also experienced laryngeal dystonia from other antipsychotics.[6] A systematic review identified 45 case reports of antipsychotic-induced acute laryngeal dystonia, a rare but potentially life-threatening event that can be mistaken for primary respiratory disease.[7]

The user's descriptions of acute dysregulated vocalisation, trouble speaking and breathlessness make oromandibular/lingual/laryngeal dystonia important alternatives. These do not conclusively identify the episodes as laryngeal dystonia. Video review by clinicians familiar with movement disorders and airway emergencies would be informative. In a new episode, inability to breathe or to speak requires emergency care, not retrospective interpretation.

4.3 Other pharmacological mechanisms

The label describes orthostatic hypotension associated with dizziness, tachycardia, bradycardia or syncope, especially around treatment initiation. It also warns about cognitive and motor impairment.[1] Other possible mechanisms or coexisting factors include sedation, an anticholinergic confusional effect, hypotension with reduced cerebral perfusion, metabolic effects, seizure, arrhythmia, hypoxaemia, or an independent transient cerebrovascular event. Abrupt inability to produce language should not automatically be labelled "drug-induced speech impairment"; aphasia/TIA/stroke and seizure remain important alternatives.

The phrase "Tourette's" is retained as the user's descriptive analogy for repetitive, uncontrollable speech-like output; Tourette syndrome itself is a specific neurodevelopmental diagnosis and is not established by a brief medication-associated event.

5 Cardiovascular and respiratory context

The reported **LVEF of 45%** is in the mildly reduced systolic function range under common classification schemes. Mitral regurgitation, tricuspid regurgitation and "LVESI 60" require their original reports for interpretation, especially the meaning and units of the last quantity.

The phrase **cardiac asthma** generally refers to wheezing and dyspnoea from heart failure-related pulmonary congestion, not to all episodes of wheezing or dyspnoea in someone with a cardiac history. White sputum/phlegm is not proof of pulmonary oedema. Cardiac decompensation, bronchospasm, aspiration, pulmonary infection, laryngeal dystonia and other causes may overlap clinically.

The medicine label calls for caution with cardiovascular and cerebrovascular disease because haemodynamic responses can be impaired.[1] For retrospective examination, the strongest discriminating data would be symptom-synchronised pulse and blood pressure, oxygen saturation, ECG/rhythm tracings, examination notes, troponin or natriuretic peptides (if measured), chest imaging, and formal interpretation of breath sounds and the original video. A reduced LVEF is a vulnerability factor, not proof that each 90-minute event represented pulmonary oedema.

6 DRESS, IL-6, infection and systemic inflammation

6.1 DRESS is a real olanzapine-associated syndrome

The FDA warned in 2016 that drugs containing olanzapine can rarely cause Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), involving a cutaneous reaction, systemic illness and sometimes hepatitis, nephritis, pneumonitis, myocarditis or pericarditis.[8] The current label continues to include a DRESS warning.[1]

A 2025 review/case study reports a range from 4 to 60 days after olanzapine initiation in previously described cases, with a median of 19 days for the FDA case series.[9] One week is within the reported lower end of onset intervals; *minutes-to-hours recurrent episodes after each dose* are not the classic onset pattern for a delayed T-cell-mediated DRESS syndrome. It is possible for an acute pharmacological reaction and a distinct delayed hypersensitivity syndrome to coexist; neither should be presumed.

DRESS assessment is based on multiple clinical and laboratory domains (including rash, fever, eosinophils, lymphadenopathy, liver/kidney involvement and alternative causes), not IL-6 by itself. RegiSCAR scoring requires actual contemporaneous data. Skin testing or intentional re-exposure is *not* something to undertake without appropriate specialist assessment, particularly following severe suspected reactions.

6.2 IL-6 38.8 and chronic jaw abscess

Assuming the reported IL-6 value of 38.8 is in pg/mL, it exceeds one Mayo Clinic Laboratories test reference interval of less than 6.4 pg/mL. Different assays and reference ranges may differ. Importantly, IL-6 is *nonspecific*; elevated serum IL-6 does not establish DRESS, BBB breakdown, microglial activation, or a "cytokine storm".[10]

A 2023 longitudinal study of patients with acute apical dental abscesses identified higher circulating IL-6, TNF-alpha and IL-10 than controls; IL-6 and IL-10 fell following treatment.[11] This makes odontogenic inflammation a biologically plausible source of elevated serum IL-6, although the user's chronic infection need not match that study's acute cohort and no individual attribution can be made without chronology and other markers.

7 Sexual and cutaneous sensory changes; breast enlargement

A reported reduction of *sexual pleasure* must be distinguished from loss of *tactile sensitivity*. Reduced subjective reward can be related to dopamine-mediated function, endocrine changes, mood, other medication effects or context. Loss of genital touch sensation implicates peripheral cutaneous afferents, sacral pathways, spinal cord and brain sensory processing; it is not simply a decrease in libido.

The current label states that D_2 receptor antagonism can elevate prolactin and can suppress hypothalamic GnRH and downstream gonadal function; it includes gynecomastia, impotence and sexual-function adverse-event descriptions.[1] Apparent breast enlargement could be glandular gynecomastia, adipose tissue (lipomastia), medication-associated weight gain, or other causes. The European Academy of Andrology guideline recommends clinical examination to differentiate these and, where appropriate, hormonal testing including testosterone, estradiol, SHBG, LH, FSH, TSH, prolactin, hCG and associated tests.[12]

One week of exposure, by itself, cannot establish the cause of persistent glandular changes. An endocrine assessment remains important even if a medication is a possible contributor. Nor does apparent breast enlargement establish that serum prolactin was elevated at the time: this requires measurement and clinical correlation.

For cutaneous sensory loss, possible anatomical sites include small sensory fibres, larger peripheral sensory fibres, dorsal roots, spinal cord tracts, thalamus, internal capsule, corona radiata, parietal cortex and higher-level bodily integration circuits. A targeted sensory examination can distinguish light touch, vibration, temperature, pinprick, proprioception and allodynia, and map left–right patterns and dermatomal boundaries.

8 Normal post-exposure nerve conduction and EMG

The timing clarification is key: the user says tests of limbs were *normal after symptoms developed*. Such results argue against substantial abnormalities of *tested large-fibre peripheral motor or sensory pathways*, conditional on the particular studies performed. EMG of muscles alone does not establish that sensory nerve action potentials were tested. Standard EMG/NCS do not assess thalamic or parietal sensory processing and generally cannot exclude isolated small-fibre neuropathy.

The 2026 AANEM evidence review specifically addresses small-fibre neuropathy with normal conventional nerve conduction and found skin biopsy with intraepidermal nerve-fibre density the best-validated direct diagnostic test among reviewed modalities, albeit with imperfect sensitivity and specificity.[13] Quantitative sensory testing, autonomic evaluation, and where appropriate SSEPs or MRI address different anatomical questions. Negative electrodiagnostics are helpful *negative evidence*, not a proof that the deficit must be in the brain.

A clinically important qualification is that neurological findings can be mixed: old focal central lesions, small-fibre dysfunction, medication effects and endocrine changes can co-occur. The distribution of objective sensory loss would determine the most appropriate diagnostic pathway.

9 Lacunar infarcts, atherosclerosis and clinical–imaging correlation

9.1 The terminology needs source-image verification

A **recent small subcortical infarct** on diffusion imaging is not precisely the same thing as a **chronic lacune** (a small CSF-like cavity) or a white-matter hyperintensity. Enlarged perivascular spaces and other changes may resemble lacunes. The 2023 STRIVE-2 consensus outlines the relevant distinctions and acquisition considerations.[14]

According to the conversation, the 2025 report described many lacunar infarcts without individually assigning precise coordinates. A lesion-by-lesion audit could determine whether each candidate is a lacune, noncavitated ischaemic lesion, perivascular space, nonspecific white matter hyperintensity or another entity. Atherosclerotic disease should be separately described by vessel and imaging modality; it must not be inferred from parenchymal spots alone.

9.2 Locations that may explain symptoms

Deep thalamic lesions can cause sensory stroke; internal capsule, corona radiata and brainstem injury may disrupt motor or ascending sensory signals; frontal–subcortical networks may alter processing speed/executive function. These are examples of anatomy, not a statement that the user's lesions occupy those locations. Normal peripheral electrodiagnostics do not contradict a central vascular cause.

If multiple genuine infarcts are present, their age, morphology, laterality and vascular distribution matter. Not all small deep lesions share the same source: small vessel pathology, proximal artery disease and cardioembolism can require distinct investigations. Reported cardiac abnormalities are an additional reason for a stroke specialist to consider appropriate cardiac evaluation rather than assuming a single mechanism.

10 The 2025–2026 apparent disappearance of lesions

10.1 What the follow-up evidence currently consists of

The user personally examined both datasets for prolonged periods and says the 2025 findings are not present on the July 2026 MRI/MRA. The second radiology report also reportedly omits them. However, **absence of a finding from a written report is not equivalent to a direct comparison statement** specifying that previously named lesions have disappeared. Imaging sequences, spatial resolution, slice thickness, contrast, flow conditions and reconstruction algorithms must be compared.

10.2 Some small infarcts do become invisible on later MRI

A study released online 29 July 2026 evaluated 173 patients with a documented recent small subcortical infarct and follow-up MRI at approximately one year. The reported lesion outcomes

included 150/173 (87%) showing some cavitation, 10/173 (6%) becoming invisible, and 4/173 (2%) partially cavitated lesions regressing; importantly the 2% regression description partly overlaps the outcome pathways rather than representing a free-standing population to add to 6%. Some lesions became smaller than 3 mm.[15]

This directly supports the limited statement that *MRI nonvisibility can occur after real small subcortical infarction*. It does *not* demonstrate full histological restoration, widespread neuron replacement, or unusually efficient repair in the user. Having *numerous* previously reported chronic lacunes all disappear would be a more unusual pattern, making lesion definitions and protocol matching especially important.

10.3 Concrete DICOM-based comparison protocol

1. Inventory each study: scanner, field strength, TR/TE, voxel size, slice thickness, orientation, acquisition dates and whether angiography was TOF or contrast enhanced.
2. Preserve original DICOM files and metadata; work from read-only copies and record checksums.
3. Examine 2025 and 2026 source images in original planes and reformat orthogonally; do not rely on a maximum intensity projection alone.
4. Register structural volumes rigidly and (if justified) nonrigidly, checking that registration has not hidden abnormalities; inspect same anatomical coordinates on native images.
5. Review candidate infarcts on T1, T2, FLAIR, DWI, ADC and susceptibility-weighted imaging where available. Note which sequences are absent and any time-dependent limitations (e.g., diffusion signal normalisation).
6. For each 2025 candidate record anatomical label, hemisphere, voxel/world coordinate, sequence, estimated size, source image/frame, classification confidence, and the corresponding 2026 finding.
7. Document discordances separately from interpretive diagnoses and request an independent specialist comparative report.

A longitudinal disappearance should be claimed only for lesions reproducibly matched across comparable source data. An honest audit could also discover instances of unchanged lesions, acquisition differences or previous overcalling.

11 The separate 2.5 mm MRA finding: candidate pseudoaneurysm

The user's 2025 3 T MRA observation was an approximately 2.5 mm focus around 2 mm from a carotid artery, not called by the radiologist. The term **pseudoaneurysm** means a contained arterial wall disruption with communication to the arterial lumen; an **embolus** is travelling intravascular material, and a true aneurysm is different again. A nearby signal focus alone is insufficient to identify any of these.

3 T time-of-flight MRA is useful for small intracranial aneurysms but is affected by flow and reconstruction artefacts; evaluating structures at approximately 2–3 mm requires care, especially on source slices. A 2012 technical review discusses both sensitivity gains at 3 T and pitfalls.[16]

A 2023 published neuroimaging case documents a 2 mm perforator pseudoaneurysm that was clearly demonstrated on catheter angiography and no longer visible on repeat catheter angiography six weeks later.[17] This proves that *some* genuine pseudoaneurysms resolve spontaneously, not that the user's carotid-adjacent structure was one. Moreover, the published lesion followed haemorrhage and affected a different artery.

Useful discriminators are whether the 2025 focus connects to the lumen on sequential source images, whether it persists across phases/sequences, whether the apparent neck has anatomically plausible

continuity, whether vessels overlap in projection, and whether the repeat study had matching flow sensitivity. A neurovascular specialist can judge whether repeat MRA, CTA or (in selected cases) invasive angiography is warranted. Not every equivocal 2.5 mm image feature justifies catheter angiography.

12 Neonatal hypoxia, BBB, microglia and a possible long-term adaptive phenotype

12.1 Restating the user's hypothesis faithfully

The user proposed that neonatal cyanosis and associated brain injury may have produced unusually strong lifelong compensatory neuroplasticity, perhaps neurogenesis. Chronic infection and a damaged or altered BBB might have changed immune–brain signalling, leaving microglia in a repeatedly mobilised or unusually responsive state. The user suggested this could help explain the observed MRI changes and perhaps increasing intellectual capacity, while noting the alternative of persistence and intensive learning.

12.2 Mechanistic pathway that has experimental support

Microglia are resident immune cells that survey and regulate CNS tissue, prune synapses, clear debris and engage in injury responses. They are not simply "always on" or "off". A 2026 *Nature Reviews Neuroscience* review describes diverse transcriptomic states and "hidden" reprogrammed states reflecting earlier exposures, including innate immune training or tolerance.[18]

In a 2020 rat neonatal hypoxia–ischaemia study, subventricular-zone microglia showed a prolonged region-specific response and expressed both inflammatory and neurotrophic genes; isolated SVZ microglia supported neurosphere generation in vitro.[19] This supports a *biological possibility*: early-life insult can affect the developmental neuroimmune environment, sometimes including neurotrophic pathways. It does not directly establish lifelong enhancement of tissue regeneration in adult humans.

A separate 2024 rat study associated acute neonatal hypoxia–ischaemia with BBB permeability disruption and changing microglial states over the first 72 hours.[20] This is acute postinjury evidence, *not* a demonstration that neonatal BBB leakage necessarily persists for decades. Whether the user's BBB remains abnormal would require independent evidence.

12.3 Placing IL-6 and chronic infection into the model

Systemic infections may influence CNS immunity by cytokine signalling, vascular and endothelial pathways, autonomic routes, and in some circumstances impaired BBB regulation. IL-6 38.8, if pg/mL, supports systemic inflammation but is not an assay of microglial activity or BBB leakiness. A dental abscess can raise IL-6 without proving ongoing neuroimmune priming.[10][11]

Continuous microglial activation is not necessarily a desirable regenerative condition: chronic neuroinflammation can impair synapses, cognition, and vascular health as well as participating in repair. The hypothesis should therefore predict *altered reactivity or homeostatic set-point*, not automatically beneficial permanent "deployment".

12.4 Neuroplasticity and neurogenesis must be separated

Neuroplasticity is reweighting, strengthening, recruiting and reorganising existing connections and functional networks; it operates throughout life and is compatible with substantial cognitive adaptation after early injury. **Neurogenesis** denotes birth of new neurons from progenitor cells and their successful integration into functionally relevant circuits. These are not synonyms.

A 2025 *Science* study identified proliferating progenitor cells in the adult human hippocampus, strengthening the case for at least some adult hippocampal neurogenesis.[21] It does not demonstrate replacement of arbitrary damaged thalamic, parietal or white-matter tissue, nor explain the invisibility of an MRI lesion by itself. Glial remodelling, inflammation resolution, cavity contraction and image detectability changes are different mechanisms.

12.5 What could explain cognitive gains?

Improved intellectual performance may reflect learning, preserved or rerouted networks, compensatory developmental plasticity, enriched cognitive activity, effort and persistence. To assert objectively "increased intelligence" would require comparable standardised tests longitudinally, accounting for practice effects, age and testing context. The user's Sudoku observations during acute episodes may be valuable *within-person function monitoring*, but are not a standardized measurement of IQ or neuron generation.

12.6 A falsifiable research formulation

> **Candidate hypothesis:** Neonatal hypoxic–ischaemic injury produces durable changes in neuroimmune response thresholds and network organisation. Later systemic inflammatory episodes may interact with this altered state to affect cognition, sensation, cerebrovascular vulnerability and recovery. Variation in adult neural compensation may be driven predominantly by network plasticity rather than large-scale neurogenesis.

This formulation is biologically reasonable as a *research hypothesis* but unproven in this individual. Predictions must be separated: altered BBB permeability, a primed microglial phenotype, anatomical resolution of ischaemic lesions, and above-baseline cognition are four separate empirical claims.

13 Integrated interpretation and outstanding tests

No single account currently explains all observations. At least five parallel hypotheses deserve independent appraisal:

1. An acute olanzapine-associated autonomic, motor, cognitive or dystonic reaction with high temporal reproducibility.
2. A separate delayed DRESS-like syndrome, requiring objective rash/eosinophil/organ findings and temporal compatibility, rather than IL-6 alone.
3. Cerebrovascular disease or other central nervous system lesions contributing to balance, sensory and language dysfunction, independent of the medication; relative dates and locations remain unknown.
4. Endocrine, metabolic, small-fibre or central processing causes for persistent altered genital/body sensation and breast appearance.
5. Long-term developmental adaptation and neuroimmune reprogramming after early hypoxic injury, a plausible programme for research but not yet a specific diagnosis.

13.1 Potential high-value documentary evidence

- The exact administration timeline: date/time, medication identity/formulation, route, dose, setting, witnesses, prior drugs and withdrawal date.
- Unedited original video/audio, handwritten contemporaneous logs, camera-file metadata and chain-of-custody notes.

- Primary cardiac MRI quantitative report and echocardiographic Doppler including interpretation of “LVESI 60” and MR/TR severity.
- Complete blood counts with absolute eosinophils, inflammatory markers and IL-6 sample dates/units, hepatic and renal chemistry, fever/rash records.
- Original detailed sensory nerve conduction and EMG measurements, including whether sensory nerve action potentials were measured.
- July 2025 and July 2026 complete DICOM datasets, scanner protocols, and both unaltered radiology reports.
- Any pre-exposure neuroimaging, prior cognitive testing, and independent specialist clinical examinations.

An adverse-drug-reaction review could use a structured causality framework, while a neuroradiological second opinion should explicitly mark lesion type, confidence, exact location and cross-year visibility. These analyses can converge without presupposing causation. Any new stroke-like deficit, acute respiratory obstruction, or chest pain merits immediate emergency evaluation.

14 Near-verbatim user statements, in discussion order

This appendix preserves the salient user phrasing that materially shaped the evolving analysis. It is an *extract*, not a complete machine-exported transcript. Minor punctuation is preserved where feasible; explanatory clarifications outside quotation marks are editorial.

Question initiating the discussion

> *hello which parts of the brain are adversely affected by olanzapine*

Exposure and reproducible acute symptoms

> *it was one week's unsupervised forced ingestion, causing immediate loss of balance, 90 minutes of cardiac asthma and tourette's involuntary repetetive babbling, spitting up white fleghm, and on one occasion stopping me entirely from being able to speak or think clearly, like i had tunnel vision / disassociation like i was in a box, completely unable to focus (i was deliberately testing by doing sudoku every day and found i couldn't do it)*
>
> *with the chronic jaw abcess and supporting evidence of IL-6 levels of 38.8 i have a sneaking suspicion that the forced ingestion of 5mg of olanzapine caused a massive D.R.O.S.S reaction*

Clarification of DRESS, cardiac status, sensation and breast appearance

> *yes D.R.E.S.S and the tourette's and Cardiac Asthma (i have MR TR, LVEF of 45% and LVESI of 60 from a Cardiac MRI, and confirmation of the MR TR by doppler) occurred immediately every single time without fail eight times and was captured on video and documented in hand-written note form with timestamps to Legal standards.*
>
> *i also have loss of physical sensation including reduction of sexual pleasure and sensation, as well as having lost sensation on other parts of my skin, and was told by my friends when sunbathing to put my shirt back on as they were disturbed by the fact that it looked like i had female breasts*

Post-exposure electrodiagnostics

> *"peripheral nerve, dermatomal, spinal or central distribution." i've had electrical nerve impulse testing done on my legs and arm muscles and the conductivity was measured and reported as normal.*
>
> *after. this was at the time when the MRI also showed multiple lacunar infarcts, evidence of atherosclerosis, and (although not on the actual Radiographer report) a false embolism approx 2.5mm in diameter, approx 2mm away from one of the cartoid arteries*

2025 vascular observation and 2026 comparison

> *"pseudoaneurysm" yes that sounds right. yes it was my own observation. the radiographer had not commented on it: i spent several weeks (not 15 minutes) studying the images in 3D.*
>
> *the Radiographer didn't pinpoint the exact areas with lacunar infarcts, as there were so many.*
>
> *visible on a 3T MRA, july 2025. MRI and MRA July 2026 shows that neither the (suspected) pseudoaneurism nor the lacunar infarcts are present. they've healed/repaired, basically.*

> *both. the lacunar infarcts and the suspected pseudoaneurism were clearly not visible to me after spending several hours reviewing the 3T MRA and MRI, and the Radiographer's own report makes no mention of either*

Proposed microglial and neuroplasticity mechanism

> *my personal suspicion / hypothesis is an unusual life-long neuroplasticity and neurogenesis, triggered by the birth cyanosis. the hypothesis is that the chronic infection and damaged blood-brain barrier has altered my body's response in some way that microglia are pretty much continuously deployed. this would also partly explain the increased intelligence although it could also be attributed to sheer bloody-minded persistence*

Document requirement and rendering problem

The user repeatedly reported truncation of the on-screen responses, requested full ASCII URLs/DOIs, and ultimately requested this near-verbatim expanded PDF created with `pdflatex` and uploaded into File Library under `lkcl`. The current dossier is intended to preserve a stable, searchable copy of the discussion.

15 Full source ledger: DOI, PMID, and exact URLs

Every bibliographic item below was checked against a discoverable publication record or recognised clinical source during preparation, except for a *legacy FDA link* separately identified in the note after the ledger. Full URLs are printed exactly, without shortened tracking links. Inline reference markers throughout this dossier map to this ledger.

[1] Olanzapine prescribing information (U.S. DailyMed; updated 15 September 2026).

Includes DRESS, orthostatic hypotension, cognitive/motor impairment, endocrine and metabolic warnings.

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Source-audit note: historical links and earlier compression

An earlier assistant response supplied the FDA URL https://www.fda.gov/downloads/Drugs/DrugSafety/UCM499603.pdf. The URL is retained here for completeness as a **historical/legacy link**, not asserted to remain functional. A separately located FDA media URL [8] was also returned by search; access to that PDF from the available search interface was unreliable. The present DailyMed label [1] and peer-reviewed DRESS review [9] are the independently usable sources for the underlying warning. A previously mentioned site, https://harness-neuroimaging.org/strive-standards, was not necessary for bibliographic verification; the verified STRIVE-2 DOI, PubMed and UCL links are provided under [14].

One correction to an overly compressed earlier reply: the 2026 lacunar-infarct study's 6% disappearance and 2% partial-cavitation regression categories should *not* be casually added as if independent rates. The primary paper provides the exact denominators and transitions.[15]

End of expanded discussion dossier. All links intentionally printed as full ASCII URLs. Generated from LaTeX using `pdflatex`; no generated images.