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Sequential Neutropenia During Risperidone and Quetiapine Treatment in an Adolescent with Autism Spectrum Disorder: A Case Report

M. Nokrou[1*], A. Abdelhamid[1], I. Bouamoud[1], H. Kisra[1]

[1]Mohammed V University of Rabat, Ar-Razi Psychiatric Hospital, Psychiatry of Children and Adolescents, Salé, Morocco

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***Corresponding author:** M. Nokrou
Mohammed V University of Rabat, Ar-Razi Psychiatric Hospital, Psychiatry of Children and Adolescents, Salé, Morocco

Abstract | **Case Report**

Background: Hematological adverse reactions associated with second-generation antipsychotics (SGAs), other than clozapine, are uncommon. Recurrent neutropenia following sequential exposure to different SGAs is exceptionally rare and has been described almost exclusively in isolated case reports. We report the case of an adolescent with autism spectrum disorder (ASD) who developed neutropenia during risperidone treatment, followed by further deterioration after switching to quetiapine. ***Case presentation:*** A 17-year-old adolescent with ASD experienced worsening behavioral symptoms, leading to gradual titration of risperidone up to 6 mg/day. Routine hematological monitoring revealed leukopenia associated with neutropenia, prompting dose reduction followed by treatment discontinuation because of progressive hematological abnormalities. Neutropenia persisted despite risperidone withdrawal. Owing to persistent severe behavioral symptoms, quetiapine was initiated. Although an initial improvement in neutrophil count was observed, dose escalation was followed by a progressive decline in the absolute neutrophil count, reaching a nadir of 0.51×10^9/L, which required discontinuation of quetiapine. A comprehensive etiological work-up (bone marrow aspirate, bone marrow biopsy, vitamin panel, viral serologies, autoimmune panel) failed to identify an alternative etiology, although pre-existing chronic neutropenia, particularly benign ethnic neutropenia, could not be definitively excluded. Causality assessment using the French Bégaud method (intrinsic imputability: I1–I2) and the Naranjo Adverse Drug Reaction Probability Scale (score = 6) supported a probable causal relationship. ***Conclusion:*** This case highlights the rare occurrence of sequential neutropenia associated with two different second-generation antipsychotics in the same patient. It emphasizes the importance of careful hematological monitoring in patients who develop blood dyscrasias during antipsychotic treatment, particularly when switching to another SGA. It also underscores the need for a comprehensive evaluation of alternative causes before attributing neutropenia solely to drug exposure. Further studies are warranted to clarify the underlying pathophysiological mechanisms and individual susceptibility factors involved in these rare hematological adverse reactions.

Keywords: Autism spectrum disorder; Risperidone; Quetiapine; Neutropenia; Secondgeneration antipsychotics; Pharmacovigilance; Adolescent; Case report.

INTRODUCTION

The use of second-generation antipsychotics (SGAs) in children and adolescents has increased considerably over the past two decades, both in Europe and North America, reflecting the broadening of their therapeutic indications and evolving clinical practice [1–4]. Initially reserved for the treatment of psychotic disorders, these medications are now widely prescribed for various neurodevelopmental and psychiatric conditions, including autism spectrum disorder (ASD), bipolar disorder, disruptive behavior disorders, and attention-deficit/hyperactivity disorder (ADHD) associated with severe aggression, not to mention numerous off-label uses [2,4,5]. A recent systematic review covering the 2013–2023 period confirms that atypical antipsychotics remain among the most frequently prescribed psychotropic medications in children and adolescents across several countries, with variations in prescribing practices according to healthcare systems, but persistent use in neurodevelopmental disorders, psychotic disorders, and numerous off-label indications [5]. This review also highlights growing concerns regarding their long-term safety in young populations. Finally, despite their demonstrated efficacy across several indications, their

increasing use has been accompanied by growing attention to their tolerability profile, particularly owing to metabolic, neurological, endocrine, and, more rarely, hematological adverse effects [4–7].

Among the adverse effects of SGAs, hematological complications are rare compared with metabolic, endocrine, and neurological effects, but they can have potentially serious clinical consequences when they progress to severe neutropenia or agranulocytosis [8,9]. Clozapine remains the antipsychotic most frequently associated with these hematological complications, with a risk high enough to justify systematic hematological monitoring throughout treatment [9,10]. In contrast, neutropenia induced by other SGAs, particularly risperidone, quetiapine, olanzapine, and aripiprazole, remains exceptional and is mainly documented through isolated case reports or small case series published in the literature [8,11–14]. The pathophysiological mechanisms underlying these blood dyscrasias remain incompletely understood. Several hypotheses have been proposed, including direct bone marrow toxicity, idiosyncratic immunological mechanisms, the formation of reactive metabolites responsible for destruction of granulocyte precursors, and individual genetic susceptibility that may predispose to hematological toxicity across different antipsychotics [8,9,15].

Reports of neutropenia recurring upon sequential exposure to multiple atypical antipsychotics remain exceptionally rare [9,11,12,16]. To date, available data rely essentially on isolated case reports, with no cohort study having systematically assessed this phenomenon [11,12,16]. The case described by Lim *et al.,*remains the principal observation reporting successive neutropenia under four different atypical antipsychotics (olanzapine, risperidone, quetiapine, then aripiprazole), suggesting the existence of individual susceptibility to hematological toxicity induced by this therapeutic class [12]. More recent reviews focusing on risperidone-induced neutropenia confirm that patients who have developed a blood dyscrasia under a first antipsychotic may be at increased risk of recurrence when another antipsychotic is introduced, warranting enhanced hematological monitoring, although this hypothesis still rests on a low level of evidence [8,9,11,16].

In this context, we report the case of a 17-year-old adolescent with autism spectrum disorder (ASD) who developed persistent neutropenia during treatment with risperidone, followed by worsening of this abnormality after switching to quetiapine. Chronological analysis of the clinical and biological data revealed a temporal and dose-dependent relationship compatible with hematological toxicity induced by these two antipsychotics, although causality assessment remains limited by the absence of complete normalization of the absolute neutrophil count (ANC) after their discontinuation. This case highlights the rarity of sequential neutropenia under two atypical antipsychotics in the same patient, a phenomenon still very poorly documented, as well as the diagnostic difficulties involved in the causal attribution of such an abnormality, requiring rigorous assessment of differential diagnoses and the use of standardized causality assessment tools. It also underscores the importance of attentive hematological monitoring whenever an antipsychotic is changed in a patient who has experienced a blood dyscrasia, and calls for further research into the pathophysiological mechanisms and individual susceptibility factors involved.

Case Presentation

Patient Information

This was a 17-year-old adolescent with autism spectrum disorder diagnosed at age 4 years, the elder of two brothers, living with his mother since his father's death in 2023. He was born to non-consanguineous parents after an uncomplicated pregnancy and a cesarean section for dystocia. His development was marked by delayed language acquisition (first words around age 2 years), whereas walking was acquired early, at 8 months.

History

The father reportedly had a history of psychiatric follow-up for a depressive disorder, although no medical documentation was available. The patient had been receiving regular child and adolescent psychiatric follow-up since the diagnosis of ASD; at his last consultation before the current episode, in May 2025, he was receiving risperidone at a dose of 4 mg/day. No other relevant medical or surgical history was identified.

Presenting Concerns and Clinical Course

The clinical picture was dominated by worsening behavioral disturbances characterized by marked motor hyperactivity, irritability with repeated episodes of aggression toward others, marked oppositional behavior, school refusal, repeated elopement, motor and vocal stereotypies, and major impairment of social interaction. The patient also reported arthralgias and chest pain without other associated clinical signs, as well as sleep disturbance and increased appetite.

Clinical examination on admission: the patient was alert and conscious, weighing 41 kg. The remainder of the examination was unremarkable.

Given the worsening irritability and aggressive behavior related to ASD, risperidone was progressively increased from 4 mg/day (May 2025) to 6 mg/day (December 2025). In our clinical practice, a complete blood count (CBC) was obtained before any dose increase.

Follow-up of the patient within our department began on December 12, 2025, when the risperidone dose was increased to 6 mg/day; earlier data (April 2021 and November 2023) were obtained from the patient's prior medical records and were collected retrospectively.

Timeline and Diagnostic Assessment

Retrospective analysis of laboratory tests showed that hematological abnormalities had in fact already appeared earlier, at a much lower dose: in April 2021, while receiving risperidone 1.5 mg/day, the complete blood count (CBC) showed leukopenia (3.65×10^9/L) associated with neutropenia (ANC 1.04×10^9/L). In November 2023, on 3 mg/day, the parameters had returned to normal (leukocytes 4.50×10^9/L; ANC 2.06×10^9/L).

After the dose increase to 6 mg/day (December 2025), a control performed ten days later confirmed recurrence of leukopenia (3.05×10^9/L) and neutropenia (ANC 1.36×10^9/L). The dose was then reduced to 4 mg/day, but a further control showed worsening (leukocytes 2.66×10^9/L; ANC 0.87×10^9/L), leading to permanent discontinuation of risperidone on January 7, 2026. Despite discontinuation, neutropenia persisted, with an ANC of 0.94×10^9/L on January 15, 2026, and 0.66×10^9/L on February 24, 2026.

Given the persistence of severe behavioral disturbances, quetiapine was introduced on February 25, 2026 (100 mg/day). The first control showed partial improvement (ANC 1.06×10^9/L). Owing to an insufficient clinical response, the dose was increased to 200 mg/day on April 13, 2026; subsequent laboratory controls then showed a progressive decline in ANC ($1.01 \rightarrow 0.87 \rightarrow 0.79 \rightarrow 0.75 \times 10^9$/L), down to a nadir of 0.51×10^9/L on June 3, 2026, while the leukocyte count remained relatively stable. Quetiapine was then discontinued on June 8, 2026.

This chronology is depicted in Figure 1 and summarized in Table 1.

Top panel: periods of exposure to risperidone and quetiapine with corresponding doses. Bottom panel: evolution of the absolute neutrophil count (ANC), with severity thresholds and drug exposure periods shown as colored bands

Table 1: Summary of Treatments, Doses, and Complete Blood Count Results

Date	Medication / Event	Dose	Leukocytes ($\times 10^9$/L)	ANC ($\times 10^9$/L)	Severity	Comment / Action
04/2021	Risperidone	1.5 mg/day	3.65	1.04	Mild	Incidental finding, not acted upon at the time
11/2023	Risperidone	3 mg/day	4.50	2.06	Normal	—
05/2025	Risperidone	4 mg/day	–	–	–	Last consultation before current episode
12/12/2025	↑ Risperidone	6 mg/day	–	–	–	Dose increase for irritability/aggression
22/12/2025	Risperidone	6 mg/day	3.05	1.36	Mild	Routine control at day 10
25/12/2025	↓ Risperidone	4 mg/day	–	–	–	Dose reduction due to neutropenia
03/01/2026	Risperidone	4 mg/day	2.66	0.87	Moderate	Worsening despite dose reduction
07/01/2026	Risperidone discontinued	—	–	–	–	Permanent discontinuation
15/01/2026	Drug-free interval	—	3.14	0.94	Moderate	Day 8 post-discontinuation
24/02/2026	Drug-free interval	—	2.36	0.66	Moderate	Day 48 post-discontinuation — paradoxical worsening
25/02/2026	Quetiapine	100 mg/day	–	–	–	Introduced for persistent severe symptoms
02/04/2026	Quetiapine	100 mg/day	3.02	1.06	Mild	Partial improvement
13/04/2026	↑ Quetiapine	200 mg/day	3.47	1.01	Mild	Dose increase for insufficient clinical response
20/04/2026	Quetiapine	200 mg/day	2.89	0.87	Moderate	—
27/04/2026	Quetiapine	200 mg/day	2.86	0.79	Moderate	—
05/05/2026	Quetiapine	200 mg/day	3.04	0.75	Moderate	—
13/05/2026	Quetiapine	200 mg/day	2.80	0.87	Moderate	—
03/06/2026	Quetiapine	200 mg/day	2.82	0.51	Moderate (nadir)	Nadir — decision to discontinue
08/06/2026	Quetiapine discontinued	—	–	–	–	Permanent discontinuation
15/06/2026	Drug-free interval	—	3.46	0.81	Moderate	Day 7 post-discontinuation
29/06/2026	Drug-free interval	—	2.90	0.72	Moderate	Day 21 post-discontinuation — incomplete recovery

Severity thresholds: normal ≥ 1.5 ×10 ^{9}/L; mild 1.0–1.49 ×10 ^{9}/L; moderate 0.5–0.99 ×10 ^{9}/L; severe < 0.5 ×10 ^{9}/L. ANC: absolute neutrophil count.

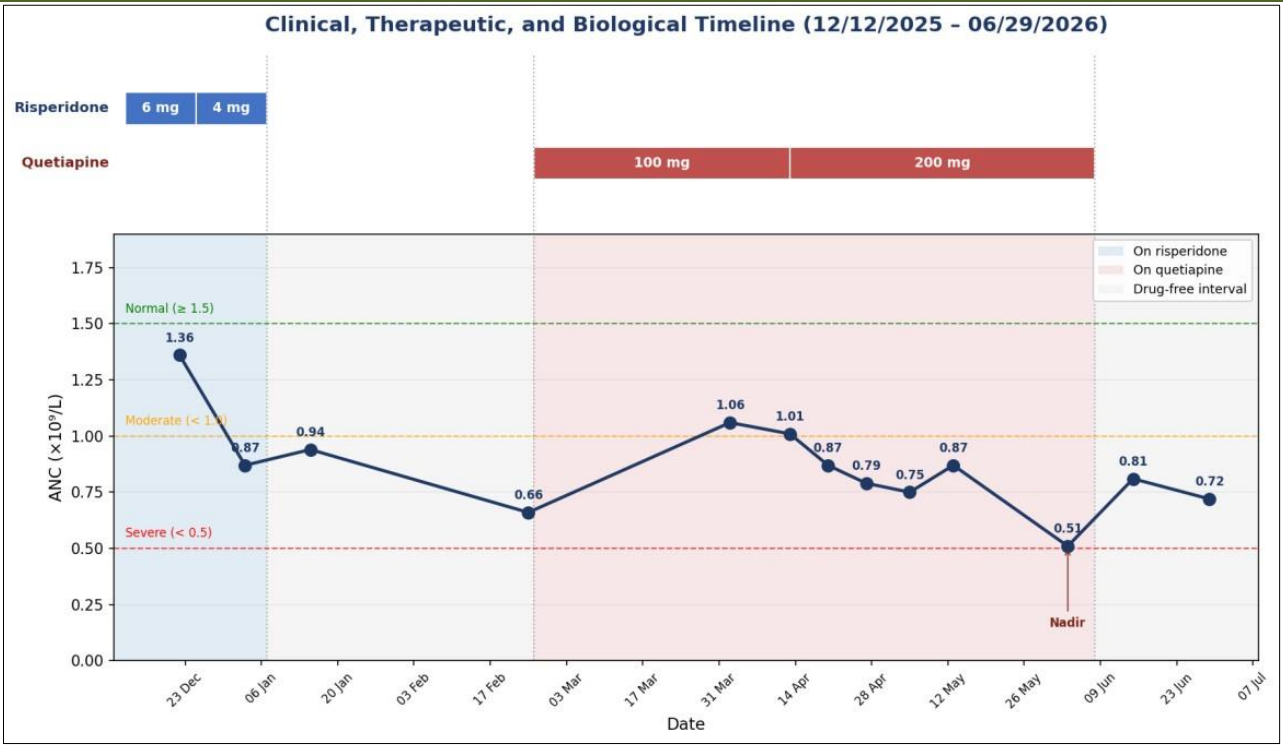

Figure 1: Clinical, Therapeutic, and Biological Timeline

No other medication known to induce neutropenia was administered during this period.

An etiological work-up was undertaken to rule out the main causes of chronic neutropenia. On pathological examination, bone marrow biopsy was borderline normal, with no fibrosis and no suspicious cellular infiltrate. Bone marrow aspirate (myelogram) showed a highly cellular smear with numerous megakaryocytes; all three cell lines were represented, with features of multilineage myelodysplasia, without blast excess or atypical cells. Serum protein electrophoresis was normal.

On laboratory testing, vitamin B12 was 389.60 pg/mL, folate 8.72 ng/mL, and ferritin 42 ng/mL, all within normal limits. Serologies for hepatitis B, hepatitis C, and HIV were negative, as was testing for anti-extractable nuclear antigen (anti-ENA) antibodies. A specialist hematology opinion concluded that the clinical picture was compatible, as a first hypothesis, with grade 3 drug-induced neutropenia (CTCAE classification) related to psychotropic treatment. An internal medicine opinion, sought in parallel, likewise favored an iatrogenic origin.

Management

Given the persistence of neutropenia, a specialist opinion from internal medicine was sought, and close clinical and biological monitoring was implemented. No infectious episode was observed during follow-up, and no antibiotic therapy, granulocyte colony-stimulating factor, or hospitalization was required.

Follow-up and Outcome

After discontinuation of quetiapine, subsequent laboratory controls showed partial improvement in neutropenia, without complete normalization of the absolute neutrophil count by the end of follow-up (ANC 0.81×10^9/L, then 0.72×10^9/L).

Causality Assessment

Drug causality was assessed using the French method of Bégaud and the Naranjo algorithm. The chronological relationship was compatible with responsibility of both antipsychotics (C1), while the etiological work-up, guided by a specialist hematology opinion, allowed reasonable exclusion of the main alternative causes without formally ruling out an underlying constitutional susceptibility (S2). Intrinsic causality was estimated at I1 to I2, and the Naranjo score was 6, corresponding to a "probable" causal relationship. These results should be interpreted with caution given the absence of a pretreatment blood count, the incomplete dechallenge, and the possibility of an underlying, non-genotyped chronic neutropenia.

Consent

Written informed consent was obtained from the patient's legal guardian for publication of this case and the associated data, in accordance with the CARE guidelines.

DISCUSSION

Our case illustrates neutropenia occurring sequentially under two second-generation antipsychotics, risperidone followed by quetiapine, in an

adolescent followed for autism spectrum disorder. Such recurrence under a second molecule of the same therapeutic class remains exceptionally rare in the literature, the closest comparable observation being that reported by Lim *et al*., who described successive neutropenia under four different atypical antipsychotics. Our observation thus provides additional evidence supporting possible individual susceptibility to hematological toxicity within this drug class.

Chronological analysis reveals a plausible temporal relationship between dose increases and the decline in ANC for both molecules: risperidone 3→6 mg/day (ANC 2.06→1.36 $\times 10^9$/L) and quetiapine 100→200 mg/day (ANC 1.06→1.01→0.87→0.79 $\times 10^9$/L), a dose-dependent gradient that supports a direct pharmacological effect. However, the course following discontinuation of each molecule remains the most problematic aspect of this case: after risperidone discontinuation, the ANC evolved from 0.87 to 0.94 $\times 10^9$/L at day 8, then paradoxically fell to 0.66 $\times 10^9$/L at day 48; after quetiapine discontinuation, it rose from 0.51 to 0.81 $\times 10^9$/L at day 7, then declined again to 0.72 $\times 10^9$/L at day 21. In both instances, the ANC never returned to the normal range (> 1.5 $\times 10^9$/L) during the available observation windows, which limits the robustness of a classically positive dechallenge. No formal rechallenge was performed; however, the switch from risperidone to quetiapine may be regarded as an informal form of "class rechallenge," and the recurrence of the abnormality on a background that had not fully normalized is clinically noteworthy despite its limited methodological probative value.

Several differential diagnoses of chronic neutropenia were systematically investigated.

Serologies for hepatitis B, hepatitis C, and HIV were negative; EBV, CMV, and parvovirus B19 serologies were not performed, which represents a limitation of this work-up. Vitamin B12 (389.60 pg/mL), folate (8.72 ng/mL), and ferritin (42 ng/mL) levels were all normal. Autoimmune testing (anti-ENA antibodies) was negative, and serum protein electrophoresis showed no abnormality. Bone marrow evaluation (myelogram and bone marrow biopsy) showed a highly cellular smear with all three cell lines represented; features of multilineage myelodysplasia were noted, without blast excess or atypical cells a finding more consistent with a reactive drug-induced effect than with a primary myelodysplastic syndrome. A specialist hematology opinion concluded that the picture was compatible, as a first hypothesis, with grade 3 drug-induced neutropenia according to the CTCAE classification, a conclusion shared by the internal medicine opinion sought in parallel. The absence of any infectious episode despite moderate-to-severe chronic neutropenia further supports this etiological orientation. The hypothesis of benign ethnic neutropenia common in certain populations and potentially explaining precisely the absence of complete normalization after discontinuation of both treatments nonetheless remains an alternative hypothesis that was not formally excluded, as the work-up performed did not include specific genotyping (DARC/ACKR1 gene). The absence of a pretreatment blood count constitutes an additional limitation: the first available result (April 2021, ANC 1.04 $\times 10^9$/L) was already obtained while the patient was receiving risperidone, precluding exclusion of pre-existing neutropenia prior to any antipsychotic exposure.

Regarding causality assessment, application of the French method of Bégaud yielded a chronology score of C1 (compatible, not suggestive, owing to the incomplete and paradoxical dechallenge) and a semiology score of S2, the comprehensive etiological work-up (viral, nutritional, autoimmune, and bone marrow) together with the specialist hematology opinion having allowed reasonable exclusion of the main alternative causes, corresponding to an overall intrinsic causality of I1 to I2 for each of the two molecules. The Naranjo score, recalculated in light of this work-up, was 6, corresponding to a causal relationship classified as "probable." There is thus a notable discrepancy between the two tools: the French method remains conservative owing to the weight it places on the incomplete and paradoxical dechallenge, whereas the additive Naranjo algorithm is less penalized by this ambiguity and is more strongly influenced by the dose-response relationship and the exclusion of alternative causes. This divergence illustrates the inherent limitations of any causality scoring system and warrants a cautious interpretation, short of concluding definite causality.

This work has several limitations that should be acknowledged. Biological follow-up by our team began only at the time of the dose increase to 6 mg/day (December 2025); the 2021 and 2023 data were obtained from the patient's prior medical records and do not benefit from the same level of direct verification. A gap of more than two years (2021–2023) without documented biological monitoring also limits analysis of the initial kinetics of the abnormality. The absence of laboratory testing at the exact time of each dose change precludes precise determination of the true interval between the therapeutic modification and the change in ANC. Finally, although the etiological work-up performed was extensive, it did not include EBV, CMV, or parvovirus B19 serologies, nor genotyping for benign ethnic neutropenia, two elements that could have further strengthened the specificity of the diagnostic attribution.

Although causality cannot be established with certainty, the sequential recurrence observed in our patient reinforces the need for careful hematological monitoring whenever switching between second-generation antipsychotics after a previous blood dyscrasia, as well as the need for a rigorous assessment of differential diagnoses before drawing any conclusion regarding drug causality. Further studies, particularly on

individual genetic susceptibility factors, would be useful to better characterize this rare phenomenon.

CONCLUSION

This report describes a rare case of neutropenia occurring successively under two secondgeneration antipsychotics, risperidone followed by quetiapine, in an adolescent with autism spectrum disorder. Although the chronological relationship, the partially dose-dependent nature of the biological course, and the recurrence of neutropenia under a second molecule support a drug-induced origin, the absence of complete normalization of the absolute neutrophil count after treatment discontinuation and the possibility of pre-existing chronic neutropenia preclude a definitive conclusion regarding causality. This observation therefore highlights the need for cautious interpretation of causality, based on rigorous chronological analysis and exclusion of the main alternative causes.

Clinically, this case underscores the importance of attentive hematological monitoring in patients who develop a blood dyscrasia under an antipsychotic, particularly when switching to another second-generation antipsychotic. It also highlights the value of multidisciplinary collaboration involving psychiatrists, internists, and laboratory specialists to optimize the diagnostic and therapeutic management of these rare situations.

Finally, the rarity of reports of recurrent neutropenia under multiple atypical antipsychotics underscores the need for additional data, particularly from multicenter studies and research into genetic and immunological susceptibility factors, to better understand the mechanisms underlying these adverse effects and to identify patients at greatest risk.

Declarations

Ethics approval and consent to participate: Not applicable. This case report did not require formal ethics committee approval at the authors' institution, in accordance with local policy for single-patient case reports; written informed consent for participation was nonetheless obtained from the patient's legal guardian.

Consent for publication: Written informed consent was obtained from the patient's legal guardian for publication of this case report, in accordance with the CARE guidelines.

Availability of data and materials: The datasets generated and/or analyzed during the current case (laboratory results and clinical timeline) are available from the corresponding author on reasonable request, subject to patient confidentiality restrictions.

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