Review Article

PARP Inhibitors-Associated Adverse Effects Across Multiple Organ Systems: A Review of Case Reports from 2022 to 2025

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ABSTRACT

Poly (ADP-ribose) polymerase (PARP) inhibitors have emerged as a groundbreaking therapeutic class for cancers associated with Breast Cancer gene (BRCA)1/2 mutations and other homologous recombination deficiencies. By disrupting Deoxyribonucleic Acid (DNA) repair pathways, these agents demonstrate substantial efficacy in treating breast, ovarian, prostate, and pancreatic cancers. However, PARP inhibitors also pose risks of adverse effects, some of which may be serious or life-threatening. This review synthesizes case reports published between 2022 and 2025 documenting adverse effects associated with PARP inhibitors. Emerging evidence highlights the diverse spectrum of adverse events, including hematologic, dermatologic, gastrointestinal, hepatic, renal, cardiovascular, neurologic, and pulmonary toxicities. Hematologic complications, particularly anemia, are most common, ranging from mild anemia to severe pancytopenia and myelodysplastic syndrome. Dermatologic reactions, such as erythema nodosum, cutaneous vasculitis, and Sweet syndrome, though uncommon, require timely recognition and management. Gastrointestinal, hepatic, and renal toxicities are generally manageable but occasionally severe, especially in older patients. Cardiovascular, neurologic, and pulmonary events remain rare yet clinically significant. Adverse events profiles vary across PARP inhibitors. Olaparib exhibits a broad spectrum of severity and organ systems, Niraparib frequently induces reversible cytopenia, Talazoparib shows favorable long-term tolerability but can affect neurologic, cardiovascular, or pulmonary systems, and Rucaparib is usually associated with mild hematologic effects but can also involve gastrointestinal, hepatic or renal toxicities. As PARP inhibitors are relatively new, continued pharmacovigilance remains essential to identify evolving safety concerns. Personalized risk assessment, vigilant monitoring, timely dose adjustments, and supportive care are essential for optimizing therapeutic outcomes and ensuring patient safety.

Keywords: PARP inhibitors; Olaparib; Niraparib; Talazoparib; Rucaparib; Veliparib; Cancer treatment; Multiple organ systems toxicities; AE; Adverse effect

INTRODUCTION

Poly (ADP-ribose) polymerases (PARPs) are a group of enzymes involved in the detection and repair of DNA strand breaks. Among the seventeen known members of the PARP family, PARP1 and PARP2 are the most well-characterized and play pivotal roles in the base excision repair pathway (1). These enzymes

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contain a conserved catalytic domain, with PARP1 also featuring Deoxyribonucleic Acid (DNA)-binding and automodification domains that regulate its activity in response to DNA damage. In normal physiology, PARPs help maintain genomic integrity by detecting single strand breaks and facilitating their repair. However, in many human cancers, particularly breast, ovarian, lung, and uterine cancers, PARP1 is found to be overexpressed (2). This overexpression is often associated with cancer progression, chemoresistance, and poor prognosis.

Therapeutically, the inhibition of PARP has emerged as a powerful strategy in the treatment of cancers with deficiencies in homologous recombination repair (HRR), especially those with Breast Cancer gene (BRCA)1/2 genes (3). These mutations impair the cell's ability to repair double-strand DNA break via HRR pathway. In such cells, blocking PARP-mediated repair forces reliance on faulty DNA repair mechanisms, resulting in genomic instability and cancer cell death. This concept has been instrumental in the development of targeted therapies called PARP inhibitors. These agents utilize the vulnerabilities of DNA repair of HRR-deficient tumors. Several PARP inhibitors have received regulatory approval for treatment, including Olaparib, Niraparib, Talazoparib, Rucaparib (4), while Veliparib remains investigational.

Olaparib is the first Food and Drug Administration (FDA) -approved PARP inhibitor, primarily used for patients with BRCA-mutated cancers such as ovarian, breast, pancreatic, and prostate cancers. It specifically inhibits PARP1 and PARP2 and has high oral bioavailability (5). Compared to other PARP inhibitors, Olaparib has the longest clinical track record, making it a well-characterized option. It is often used as monotherapy or in combination and has shown substantial efficacy in maintenance settings. Niraparib is noted for its once-daily dosing and efficacy in both BRCA-mutated and non-mutated tumors. Rucaparib is highlighted for its activity in both germline and somatic BRCA-mutated tumors (4). Talazoparib is distinct for its dual activity: it not only inhibits PARP enzymatic activity but also effectively traps PARP-DNA complexes, enhancing cytotoxicity in cancer cells with BRCA mutations. It is more potent than Olaparib on a molar basis, which can translate to greater efficacy but also increased toxicity in some patients (6).

Numerous clinical trials and post-marketing analyses have validated the efficacy of PARP inhibitors in extending progression-free survival (PFS) in patients with BRCA-mutated or HRR-deficient tumors. However, these agents are not without risk. Hematologic toxicities such as anemia, neutropenia, thrombocytopenia, and even secondary malignancies like myelodysplastic syndrome (MDS) have emerged as concerning adverse effects. Adverse events (AE) on multiple organ systems have also been documented (7).

To capture the most recent evidence on real world side effects, this review evaluates published case reports between 2022 and 2025 that describe adverse reactions associated with PARP inhibitors, across single-agent and combination therapies, with the aim of providing clinical insight into how these adverse effects manifest and are managed. Case reports were the focus because they often document rare, severe, or unexpected toxicities not fully represented in clinical trials, which frequently exclude patients with comorbidities and may underreport long-term events. However, some real-world or clinical trials studies were researched to add additional details, especially for less frequently reported AEs. By aggregating these findings, we aim to offer an additional and practical resource for evaluating the therapeutic benefits of PARP inhibition against the risk of multisystem toxicities.

Sources were identified via Google Scholar and PubMed to identify case reports detailing side effects of PARP inhibitors which were published between 2022 to 2025. Only English-language case reports involving the use of PARP inhibitors were considered. Extracted data included patient demographics, disease, biomarkers, type of PARP inhibitors, treatment duration, and methods of AE assessment and management. Forty-four reports, consisting of 38 individual cases or 6 case series, were analyzed, including Olaparib (23 reports), Niraparib (13 reports), Talazoparib (4 reports), Rucaparib (3 reports), and Veliparib (1 report). Overall, these reports documented 88 AEs in 60 patients. Olaparib accounted for most reports, reflecting its longer clinical history as the first approved PARP inhibitor, while Veliparib was rarely reported, due to its current investigational status. Figure 1 summarizes the findings, categorized by affected organ systems and PARP inhibitors. Details are discussed below.

Hematologic Adverse Effects of PARP Inhibitors

Hematologic adverse effects are the commonly reported events associated with PARP inhibitor use in cancer treatment. In this study, hematologic adverse effects were described in 56.8% (25/44) of reviewed case/case series reports, accounted for 60.0% (36/60)

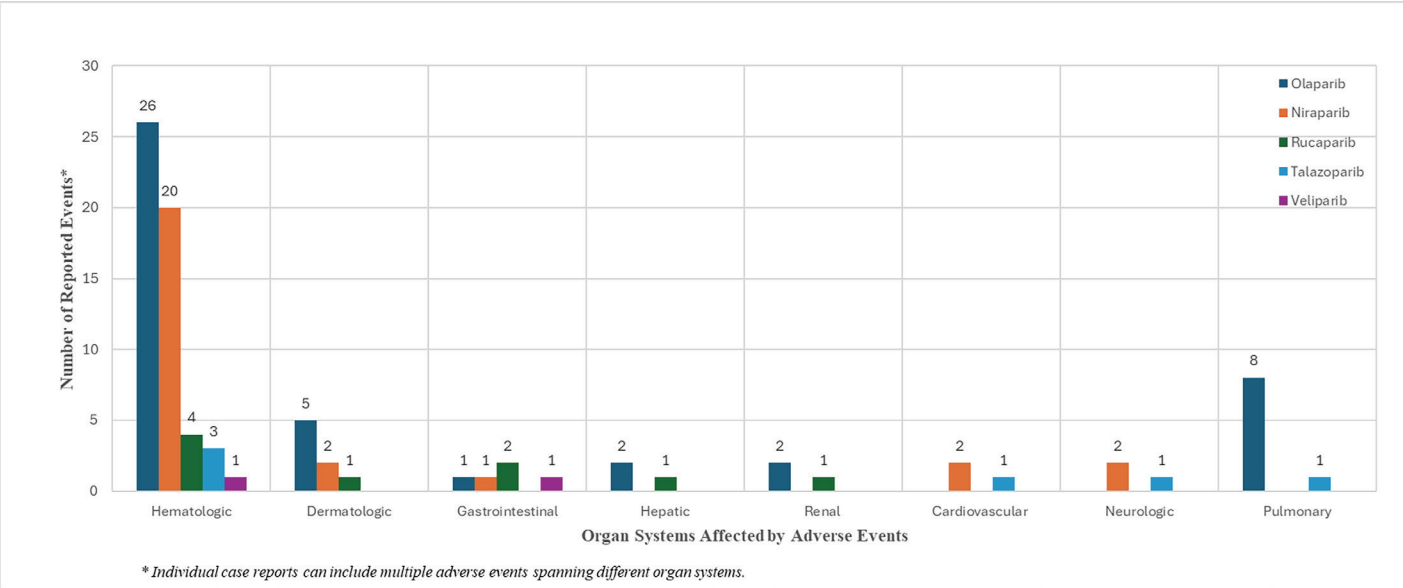

Figure 1. Number of Reported Adverse Events by Affected Organ Systems and PARP Inhibitors from Case Reports between 2022 and 2025.

patients, and represented 61.4% (54/88) of all reported AEs.

Anemia

Anemia is the most frequently reported hematologic toxicity and represents the most common category of AEs associated with all PARP inhibitors.

Several case reports published between 2022 and 2025 illustrate a various range of anemia severity with Olaparib. Mild, self-resolving anemia was reported in a 53-year-old male (YOM) patient with metastatic cholangiocarcinoma (BRCA2+, high Programmed Death-Ligand-1 (PD-L1)) after a dose reduction (8), and a 48-year-old female (YOF) with triple-negative breast cancer previously received adjuvant chemotherapy (9), both receiving Olaparib in combination with pembrolizumab. A grade 1 anemia was also reported during Olaparib treatment in a 64YOM with variant prostate cancer with BRCA2, Phosphatase and Tensin Homolog, Ataxia Telangiectasia Mutated (ATM), Retinoblastoma-1, Catenin Beta-1, Fanconi Anemia Complementation Group A alterations, which was well tolerated and manageable (10). Additionally, case of grade 2 anemia was reported in a 70-year-old woman with platinum-sensitive recurrent ovarian cancer receiving Olaparib monotherapy (600 mg daily). Although the anemia was tolerated, treatment was ultimately discontinued due to concurrent dermatologic toxicity (11). In a six-case series, one patient, a 52-year-old postmenopausal female with high-grade endometrioid ovarian cancer with BRCA1 mutation developed moderate anemia during Olaparib treatment. Before PARP inhibitor therapy, the patient previously underwent primary debulking surgery, partial frontline platinum-based chemotherapy, carboplatin, ands pegylated liposomal doxorubicin due to a para-aortic lymph node relapse. The anemia prompted a dose reduction of Olaparib from 600mg daily to 450 mg, after which the patient maintained disease control for 32 months without further significant toxicity (12).

More severe anemia has also been reported. A 56YOF patient diagnosed with stage IIA breast cancer in the left breast, which was Estrogen Receptor (ER) negative, Progesterone (PR) negative, and Human Epidermal Growth Factor Receptor-2 (HER2) positive, developed severe grade 3 anemia after four months of Olaparib therapy, despite multiple dose adjustments and supportive therapy; resulting in treatment being discontinued after six months, and hormone therapy with exemestane being initiated (13). A retrospective analysis found that 59.1% of patients experienced Olaparib-induced anemia, with 36.4% having grade 3 or higher anemia, with patient-specific factors such as low baseline hemoglobin (Hgb), hematocrit, and red blood cell count significantly increasing risk, highlighting its dose-limiting nature (14). Another case involved a

71YOF BRCA-positive patient with high-grade serous ovarian cancer who developed prolonged, severe anemia despite dose reductions and multiple transfusions. Bone marrow evaluation revealed a clonal T-large granular lymphocyte population (20% cellularity) and a DNA (cytosine-5)-methyltransferase 3 alpha (Variant allele frequency 32%) without myelodysplasia. Subsequent warm autoimmune hemolysis responded rapidly to high-dose corticosteroids, illustrating mixed-origin anemia from prolonged Olaparib toxicity and secondary autoimmune complications (15). In another notable case, a 30-year-old woman with advanced triple-negative breast cancer treated with pembrolizumab in combination with Olaparib initially at 300 mg twice a day developed persistent grade 3 anemia, further developed into pancytopenia, which necessitates dose reduction, and eventually suspension (16).

Treatment with Niraparib has also been associated with development of anemia in patients with various cancers, including tonsillar carcinoma, ovarian carcinoma, and brain metastases. Reported cases demonstrate that anemia can range from moderate (grade 2) to severe (grade 3 or higher). For example, a 62-year-old man with metastatic undifferentiated tonsillar carcinoma developed grade 2 anemia (Hgb 8.3 g/dL) within the first month of niraparib therapy, accompanied by mild fatigue and leukopenia; these AEs resolved after reducing the dose to 200 mg once daily, allowing continued treatment (17). An 82-year-old woman with high-grade serous ovarian adenocarcinoma and peritoneal carcinomatosis developed pancytopenia, including grade 2 anemia (Hgb 9.2 g/dL), following co-administration of fusidic acid. She presented with petechial purpura and oral bleeding, requiring hospitalization, platelet transfusions, filgrastim, and vitamin supplementation; niraparib was temporarily stopped and later resumed at 200 mg daily, eventually returning to her original 300 mg daily dose after normalization of blood counts (18). Similarly, a 48-year-old woman with BRCA wild-type ovarian cancer and brain metastases developed grade 3 anemia after eleven weeks of niraparib therapy at the dose of 200 mg once daily. Treatment was suspended for four weeks, then resumed at 100 mg once daily, increased to 200 mg, and later reduced again to 100 mg daily. The anemia resolved, and the patient maintained stable disease with PFS of 29 months (19). However, dose-adjustment is not always effective. A case was reported in a 78-year-old patient with a history of serous ovarian carcinoma and prior diffuse large B-cell lymphoma, who developed moderate anemia (Hgb 9.6 g/dL) while receiving maintenance Niraparib following a response to platinum-based chemotherapy. Bone marrow evaluation revealed infiltration by high-grade B-cell lymphoma, indicating leukemic relapses, resulting in PARP inhibitor discontinuation (20). These cases highlight that although anemia associated with Niraparib is often reversible with temporary treatment interruption, dose adjustment, or supportive care, complications might arise and close monitoring for more severe side effects are essential.

Talazoparib appears to be better tolerated in selected patients, but tolerability can vary. In a case of BRCA2-positive metastatic breast cancer, a 38YOF patient received Talazoparib for six years and experienced only mild grade 1 anemia (21). In contrast, a phase III Talazoparib Prostate Cancer Trials-2 trial involving Talazoparib in combination with enzalutamide for HRR-deficient metastatic castration-resistant prostate cancer (mCRPC) reported that 41% of patients developed anemia at grade 3 or higher, which led to dose adjustments (43.2% of cases) or discontinuation (8.3% of cases) (22).

Rucaparib typically produces mild hematologic toxicity, including anemia, in patients with BRCA-mutated metastatic breast cancer. In two reported cases (23), a 37-year-old woman with germline BRCA1–mutated and a 35-year-old woman with somatic BRCA2–mutated triple-negative breast cancer developed mild anemia and asthenia during Rucaparib therapy. Both patients were effectively managed with dose reductions (from 600 mg twice daily to 300 mg twice daily), and continued treatment allowed prolonged disease control and complete response.

While anemia is the most frequently observed hematologic toxicity, occurring with all types of PARP inhibitors and exhibiting a wide range of severity – as outlined above – PARP inhibitors can also impact other blood cell lineages. Patients may develop leukopenia, neutropenia, thrombocytopenia, or even pancytopenia, reflecting broader myelotoxic effects. The following section highlights these additional cytopenias, their severity, and management considerations across different PARP inhibitors.

Other types of cytopenia and Myelotoxicity

Severe hematologic complications affecting multiple blood lineages have also been reported with PARP inhibitors.

Leukopenia and neutropenia have been observed

during PARP inhibitor therapy, with both Olaparib and Niraparib. These reductions in white blood cells (WBC) increase the risk of infection and may necessitate dose modification or treatment interruption. A case reported that a 66YOF patient, with BRCA2-mutated metastatic ovarian adenocarcinoma and primary hepatocellular carcinoma, developed moderate leukopenia (WBC: 2.47×10^9/L) and asthenia during Olaparib therapy. Olaparib was discontinued after 2.5 months, and the patient subsequently recovered (24). In another report, a 64YOM patient with BRCA2-mutated aggressive variant prostate cancer receiving Olaparib (300 mg twice daily) after chemotherapy and radiotherapy experienced occasional leukopenia (grade 1-2), which were manageable during treatment (10). Similarly, grade 3 neutropenia occurred in a 70-year-old woman with platinum-sensitive recurrent ovarian cancer after eleven months of Olaparib maintenance therapy. Although treatment was temporarily suspended and subsequently resumed, it was ultimately discontinued due to non-hematologic adverse effects (11). Niraparib recipients are also reported to develop leukopenia and neutropenia. One patient with metastatic undifferentiated tonsillar carcinoma developed leukopenia and anemia during Niraparib plus tislelizumab, which was successfully managed with dose adjustment (17). Another report described an 82-year-old woman with high-grade serous ovarian adenocarcinoma who developed grade 2 leukopenia and neutropenia after 2.5 years of Niraparib therapy combined with fusidic acid. Her blood counts improved after discontinuation of Niraparib, and she was later rechallenged without recurrence (18). Hence, while Olaparib was associated with a wider range of leukopenia severity and tolerability, Niraparib appeared to be comparatively more tolerable.

Persistent thrombocytopenia has been reported mostly in patients receiving Niraparib. A 50-year-old woman with metastatic clear cell renal cell carcinoma and CDK12/RAD51C mutations developed mild thrombocytopenia one week after starting Niraparib 200 mg daily, which resolved after reducing the dose to 100 mg/day (25). Another case reported that a 56YOF patient with advanced ovarian cancer developed persistent grade 1 thrombocytopenia after 4 years of Niraparib treatment, leading to Niraparib discontinuation and stem cell transplantation. She was later switched to Olaparib 200 mg daily, where thrombocytopenia persisted but remained tolerable, allowing continued therapy (26). In a reverse switch from Olaparib to Niraparib, a 69-year-old BRCA1-mutated female with stage IIIC high-grade serous ovarian carcinoma initially received Olaparib maintenance but relapsed after five months. Following secondary cytoreductive surgery and nedaplatin, Niraparib (200 mg/day) was started, leading to normalization of CA-125 and radiographic regression. The patient developed grade 2 thrombocytopenia (platelets 50,000–75,000/μL) during Niraparib, which resolved after a two-week pause, allowing continuation. The patient achieved complete response and remained progression-free for 15 months (27). In a six-case series, a 65-year-old postmenopausal female with high-grade serous ovarian cancer (BRCA wild-type) developed recurrent grade 2 thrombocytopenia while receiving Niraparib 300 mg once daily as maintenance therapy following neoadjuvant platinum-based chemotherapy and interval debulking surgery. The thrombocytopenia required a three-week treatment interruption and dose reduction to 200 mg daily. Despite these hematologic toxicities, Niraparib was safely continued without further dose adjustments (12). More severe thrombocytopenia is also linked to Niraparib. A case report described that a 48-year-old woman with BRCA wild-type ovarian cancer and brain metastases developed grade 2 thrombocytopenia alongside grade 3 anemia during Niraparib therapy, which was resolved through multiple dose adjustments and care (19). An additional case reported Niraparib-associated pancytopenia, including grade 4 thrombocytopenia (platelet count of 1×10^9/L) and anemia, in an 82-year-old woman with high-grade serous ovarian carcinoma and peritoneal carcinomatosis. Following oncologic treatment, Niraparib was resumed without recurrence of adverse effects (18).

However, Niraparib-related thrombocytopenia is not always reversible. In one case, a 59-year-old woman on Niraparib maintenance therapy for stage IIIC high-grade serous ovarian cancer developed severe thrombocytopenia (grade 4, platelets 7×10^9/L), requiring hospitalization, platelet transfusions, thrombopoietin agonists, and close monitoring. Despite these interventions, thrombocytopenia persisted and the patient condition deteroriated (28). Most cases, however, as outlined above, respond to careful dose adjustments, sometimes multiple modifications, allowing treatment continuation while managing hematologic toxicity. With careful monitoring and dose modification, patients achieved prolonged disease control, and their thrombocytopenia was effectively managed.

Olaparib has also been associated with thrombocytopenia. One case described 30-year-old woman with advanced triple-negative breast cancer

who initially received Olaparib 300 mg twice a day but required dose reduction due to thrombocytopenia and other hematologic toxicities. The patient's condition later worsened, ultimately necessitating complete suspension of PARP therapy (16). It is important to note that in patients with complex oncologic conditions, attributing thrombocytopenia can be challenging, as it may result from PARP inhibitor therapy, progression of the underlying disease, or an interaction between both. For example, a case report documented a 78YOF patient with a history of serous ovarian carcinoma and prior diffuse large B-cell lymphoma who developed severe thrombocytopenia (platelets 28 × 10^9/L) and anemia (Hgb 9.6 g/dL) while receiving maintenance Niraparib post platinum-based chemotherapy. Bone marrow evaluation revealed infiltration by high-grade B-cell lymphoma, suggesting leukemic relapse; however, it remains unclear whether the thrombocytopenia was directly related to PARP inhibitor therapy or to underlying disease progression (20).

A more severe hematologic adverse effect is pancytopenia, which involves simultaneous reductions in red blood cells, white blood cells, and platelets, making it particularly serious due to the combined risks of anemia, infection, and bleeding. Reported cases include a 58-year-old woman with breast cancer developed pancytopenia progressing to MDS after five to six months of combined Olaparib and Programmed Cell Death Protein 1 (PD-1) inhibitor therapy, requiring discontinuation and hospice care (29). A more severe case was reported in a 75YOM patient with castrate-resistant prostate cancer who developed aplastic anemia after initiating Olaparib therapy, with reductions in hemoglobin, white blood cells, and platelets, consistent with pancytopenia. Discontinuation of Olaparib and supportive care led to gradual recovery of blood counts, highlighting the potentially severe myelosuppressive effects of PARP inhibitors even in patients without prior hematologic disorders (30). In another notable case, a 30YOF patient with advanced triple-negative breast cancer treated with pembrolizumab in combination with Olaparib (initially at 300 mg twice daily) developed other hematologic toxicities that required dose reduction, followed by the suspension of PARP inhibitor therapy. One week after treatment suspension, she developed pancytopenia, which requires transfusions (16). Niraparib has also been associated with grade 3 pancytopenia following treatment, as reported in a patient with platinum-sensitive ovarian cancer. In this case, pancytopenia was exacerbated by the use of fusidic acid but resolved with supportive care and dose reduction (18). In another case from a six-patient series, a 65-year-old postmenopausal female with high-grade serous ovarian cancer (BRCA wild-type), while receiving Niraparib maintenance at 300 mg once daily following neoadjuvant platinum-based chemotherapy and interval debulking surgery, developed severe pancytopenia (Hgb 7.41 g/dL, neutrophils 1.40 × 10^3/μL, and platelets 6.77 × 10^3/μL) and hypocellularity detected from bone marrow biopsy, accompanied by spontaneous hemorrhages. Niraparib was continued at a reduced dose of 200mg per day until later in her disease course when neurologic complications developed (12).

Another serious hematologic adverse effect associated with PARP inhibitors is the development of MDS and acute myeloid leukemia (AML), particularly after long-term use. One report described a 58YOF patient with stage IIIA left breast cancer (ER+, PR-, BRCA1, BRCA2 mutations) who received Olaparib in combination with Keytruda after several prolonged but poorly tolerable treatments. During Olaparib therapy, the patient developed therapy-related MDS with sign of AML, including tumor protein 53 (TP53) depletion. All treatments were later stopped as the patient elected to go to hospice (29). Another 56YOF patient with advanced ovarian cancer and a BRCA1 mutation developed recurring MDS after four years of maintenance therapy with Niraparib. Following autologous stem cell transplantation, the patient was re-treated with Olaparib and achieved a partial response, remaining on therapy for 11 months (26). Another case reported a 67YOF patient with progressive ovarian cancer treated with Talazoparib (1mg/day), who developed acute pancytopenia, circulating blasts, and near-total bone marrow replacement by malignant blasts. The patient was diagnosed with therapy-related MDS progressing into secondary AML, with disseminated intravascular coagulation contributing to thrombocytopenia. Given her poor fitness for intensive chemotherapy, supportive care and hypomethylating therapy were considered, but due to rapid disease progression, the patient opted to palliative care and eventually died (31). Additionally, a case series reported nine patients receiving PARP inhibitors (Olaparib, Niraparib, and Rucaparib), all developed myeloid malignancies. Among these, four patients, two treated with Olaparib and two with Niraparib, were diagnosed with MDS, while five developed AML, including four on Olaparib and one on Rucaparib. Molecular analyses frequently revealed complex karyotypes and TP53 mutations, features

that distinguish PARP inhibitor–associated myeloid neoplasms from their de novo counterparts (32).

Lymphocyte abnormalities, specifically lymphocytopenia, have been observed during the treatment of PARP inhibitors. In a case of Rucaparib-associated Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome, a 63-year-old woman developed lymphocytopenia (lymphocytes 0.27 k/μL) along with systemic symptoms and diffuse rash. Rucaparib was discontinued, and corticosteroid therapy resulted in rapid clinical improvement (33). In another patient with advanced prostate cancer treated with Olaparib, continuous grade 1- and one-time grade 3 lymphocytopenia was observed (10). This patient experienced grade 1 lymphocytopenia during chemotherapy, occasional grade 1–2 lymphocytopenia during radiotherapy combined with Olaparib, and a single episode of grade 3 lymphocytopenia later in treatment. These cases demonstrate that PARP inhibitors can induce lymphocyte suppression, even in patients without pre-existing bone marrow disorders, highlighting lymphocytopenia as a relevant but often mild-to-moderate hematologic adverse effect.

These reports indicate that cytopenias beyond anemia represent serious but less common AEs associated with PARP inhibitor therapy. Olaparib carries a notable risk of progression to MDS or AML, whereas Niraparib-induced cytopenias are often reversible with supportive care or dose modifications.

Hemorrhage in other organs

Veliparib has been associated with intracranial hemorrhage in an FDA Adverse Event Reporting System (FAERS) case 46 days after initiation (34), indicating potential delayed hemorrhagic risk. Another report detailed a 65YOF patient with high-grade serous ovarian cancer who received Niraparib maintenance at 300 mg daily after neoadjuvant platinum-based chemotherapy and interval debulking surgery. Early in treatment, the patient developed recurrent grade 2 thrombocytopenia requiring a 3-week interruption and dose reduction to 200 mg daily. Later, she experienced severe hemorrhagic events, including spontaneous hematomas and conjunctival/eyelid bleeding, with bone marrow biopsy showing 25% CLL/SLL infiltration and hypocellularity. The bleeding events were clinically significant, reflecting severe PARP inhibitor-related hematologic toxicity, and were managed in the context of ongoing disease progression, which ultimately led to rapid clinical decline and death 12 months after brain metastasis diagnosis (12). The most severe case was reported in a 49YOF with metastatic triple-negative breast cancer, a BRCA2 pathogenic variant, and a family history of breast and prostate cancer. While receiving Talazoparib as fourth line PARP inhibitor therapy, the patient developed severe risk of hematologic complications, including disseminated intravascular coagulation (DIC). The DIC prevented continuation of systemic therapy, despite prior management of deep vein thrombosis (DVT) with anticoagulation and drug-induced pneumonia with high-dose steroids (35).

In summary, PARP inhibitors are predominantly associated with hematologic toxicities. Figure 2 illustrates the relative proportion of each hematologic adverse effect reported in case studies from 2022 to 2025, in comparison to adverse events in other organ systems, emphasizing the importance of careful monitoring for these events.

Dermatologic/Cutaneous Adverse Effects of PARP Inhibitors

Cutaneous reactions have been reported with PARP inhibitor therapy, most commonly erythema nodosum and immune-mediated rashes, particularly with Olaparib. Dermatologic adverse effects can occur anytime from within hours of treatment initiation to as late as one year after starting therapy. For example, a 70YOF patient with stage IVB high-grade adenocarcinoma breast cancer developed a rapid-onset hypersensitivity reaction within two hours of receiving Olaparib, manifesting as a diffused rash over the face, neck, upper trunk, back, and abdomen, without systemic symptoms such as hypotension, tachycardia, or respiratory compromise. The patient successfully underwent a desensitization protocol and was able to continue Olaparib therapy without recurrence of the reaction (36). A 69-year-old woman with recurrent BRCA2-mutated breast cancer developed erythema nodosum during Olaparib monotherapy, which resolved within one week following its discontinuation and corticosteroid treatment (37). More recently, a 41-year-old woman with high-grade serous ovarian carcinoma developed cutaneous vasculitis with pain and mobility limitation after several months of Olaparib maintenance therapy at 300 mg twice daily. The skin lesions resolved completely within one week after Olaparib was discontinued, with no recurrence observed during a six-month follow-up (38). Similarly, an 80YOF patient with recurrent stage IVB ovarian cancer developed purpura, swelling, and subcutaneous induration of both

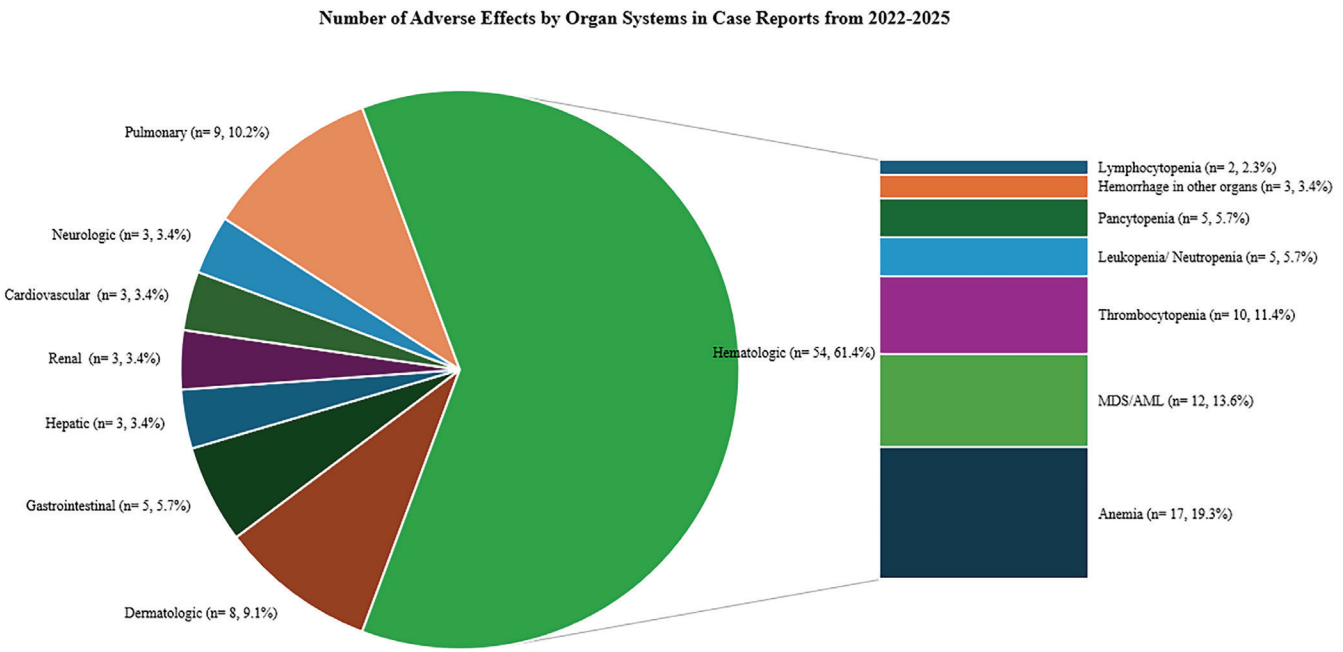

Figure 2. Distribution of Adverse Events Associated with PARP Inhibitors by Organ Systems and Hematologic Subtypes from Case Reports between 2022 and 2025.

lower legs one month after starting Olaparib, even at a relatively low dose of 40 mg/day. Skin biopsy confirmed necrotizing cutaneous vasculitis with lymphocytic and neutrophilic infiltration, and symptoms improved following drug discontinuation (39). In another report, a 70-year-old woman with recurrent ovarian cancer developed Olaparib-induced grade 2 dermatosis during maintenance therapy (11). After 11 months of sole Olaparib treatment, the patient presented with multiple erythematous, pruritic skin eruptions localized to the trunk and extremities. Dermatologic assessment supported a drug-induced reaction. Olaparib was discontinued, and topical corticosteroids led to complete resolution of the skin lesions. Upon rechallenge, the dermatologic symptoms recurred, confirming the causal role of Olaparib. The treatment was permanently discontinued, and no recurrence was observed during follow-up.

Other PARP inhibitors, although less commonly, have also been associated with more severe dermatologic toxicities. In an 82YOF with high-grade serous ovarian adenocarcinoma treated with Niraparib 300 mg daily, cutaneous manifestations included petechial purpura on the lower limbs and oral bleeding, coupled with severe pancytopenia (18). A 57-year-old woman with recurrent endometrial carcinoma developed Sweet syndrome - an Acute febrile neutrophilic dermatosis condition - while receiving Niraparib. The patient presented with fever and painful erythematous plaques, consistent with acute febrile neutrophilic dermatosis. The condition improved after Niraparib was discontinued and systemic corticosteroids were initiated (40).

A more severe dermatologic toxicity was reported in a case related to Rucaparib. A 63-year-old woman receiving Rucaparib 300 mg twice daily developed DRESS syndrome, presenting with a diffuse, erythematous, pruritic, maculopapular rash that involved approximately 90% of the body surface area, including the trunk, extremities (with palm involvement), and facial swelling, together with other symptoms such as fever, eosinophilia, and lymphocytopenia (0.27 k/μL). Management included discontinuation of Rucaparib and initiation of systemic corticosteroids (methylprednisolone followed by oral prednisone taper) plus topical triamcinolone. Within 72 hours, there was rapid improvement in rash, organ function, and laboratory abnormalities (33).

Evidence from these cases show that dermatologic

toxicities, although relatively uncommon, can manifest in a wide range of severity, from benign rashes to severe immune mediated dermatoses, and may require temporary or permanent discontinuation of the PARP treatment.

Gastrointestinal Adverse Effects of PARP Inhibitors

Gastrointestinal (GI) AEs are usually mild, including nausea, vomiting, and diarrhea, while severe GI toxicities are uncommon. A report of a Caucasian women with early stage of breast cancer and a BRCA mutation treated initially with Olaparib 300mg twice a day experienced mild gastrointestinal symptoms, including grade 1 nausea and diarrhea (41). In a case using Niraparib therapy for brain metastasis, the patient developed nausea, fatigue, and asthenia during the first week, followed by a recurrence of epilepsy, which prompted temporary treatment suspension (19). Another patient, treated with Niraparib for metastatic tonsillar carcinoma, developed gastrointestinal bleeding due to colon metastasis (17). Rucaparib was also associated with mild gastrointestinal adverse effects, including grade 1-2 diarrhea, nausea, and fatigue. These AEs were reported in a 52-year-old man with mCRPC receiving 600 mg twice daily, later reducing to 500 mg twice daily which effectively managed symptoms and allowed continuation of therapy (42). Similar gastrointestinal side effects were also reported in a patient who developed Rucaparib-associated DRESS syndrome (33). In addition, Veliparib has been associated with delayed lower gastrointestinal hemorrhage, as documented in a FAERS case 46 days after treatment initiation (34).

These reports indicate that gastrointestinal symptoms are generally manageable, but serious bleeding events may occur, highlighting the need for close monitoring.

Hepatic Adverse Effects of PARP Inhibitors

Hepatic toxicity, though rare, has been reported. The first reported case in the United States in 2022 involved a 56-year-old women with advanced high-grade endometrioid and serous carcinoma and BRCA mutation treated with Olaparib The patient developed immune-mediated severe acute hepatocellular liver injury, presenting with markedly elevated Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST), and an impending fulminant liver failure. Discontinuation of Olaparib and treatment with corticosteroids led to rapid recovery (43). Another report presented a 70-year-old female with ovarian carcinoma experienced severe (grade 4) hepatocellular injury while receiving Olaparib, with ALT peaking at 871 U/L and AST at 773 U/L. Her liver enzymes normalized upon discontinuation of Olaparib but rebounded upon rechallenge, confirming the drug's role in the injury. No cross-reactivity occurred when switched to Niraparib (44). In addition, a 63-year-old woman receiving Rucaparib developed systemic manifestations including liver enzyme elevations as part of DRESS syndrome, which improved after discontinuation of therapy and initiation of systemic corticosteroids (33).

These findings emphasize that hepatic toxicity is uncommon but clinically significant, with cases beginning to emerge since the first report in 2022. Switching PARP inhibitors may be a viable strategy for managing affected patients.

Renal Adverse Effects of PARP Inhibitors

Renal adverse events (RAE) have been described primarily in combination regimens. An 83-year-old male with BRCA2-mutant lung squamous carcinoma developed an acute grade 2 immune-related renal toxicity, with creatinine elevation and proteinuria, while receiving Olaparib with the PD-1 inhibitor sintilimab. Renal function normalized after discontinuing the immune checkpoint inhibitor (45). A case presented at the American Society of Nephrology's Kidney Week 2023 reported a 61-year-old woman with type II diabetes and hypertension who developed creatinine elevation during Olaparib treatment for metastatic ovarian cancer with carcinomatosis. The patient's baseline creatinine rose from 0.8 mg/dL to 2.0 mg/dL, while Estimated Glomerular Filtration Rate declined from 67 mL/min to 28 mL/min over several months following treatment. Urinalysis was unremarkable, and imaging showed no structural abnormalities, suggesting Olaparib-related renal dysfunction in a patient with pre-existing comorbidities (46).

In addition, a 63-year-old woman receiving Rucaparib 300 mg twice daily for ovarian cancer developed DRESS syndrome, which included mild acute kidney injury with elevated creatinine (1.7 mg/dL) alongside rash, eosinophilia, and lymphocytopenia. Management involved discontinuation of Rucaparib and initiation of systemic corticosteroids, leading to rapid improvement in renal function (33). A comprehensive FAERS analysis revealed that RAE occurs predominantly in patients over 85 years old, with Veliparib exhibiting a higher risk compared to other PARP-inhibitors, reporting a reporting odds ratio (ROR) of 29.20, a proportional risk ratio of

19.80, and empirical Bayes geometric mean of 19.80 (47). Another FAERS-based study analyzing 2,726 cases of acute renal failure associated with PARP inhibitors over eight years found that Olaparib has the highest mortality rate (9.88%) with median time to onset of 57 days, compared to Niraparib (mortality: 2.52%, onset: 36 days) and Rucaparib (mortality: 2.94% mortality, onset: 85 days). These findings indicate that the risk, severity, and timing of RAEs vary among different PARP inhibitors (48).

Overall, renal adverse events from PARP inhibitors can be clinically significant. Patient age, comorbidities, and combination therapy regimens influence risk and severity, making close renal monitoring essential for early detection and management.

Cardiovascular Adverse Effects of PARP Inhibitors

Although narrative case reports of cardiovascular toxicity from PARP inhibitors remain scarce, very few instances have been documented between 2022 and 2025. In one case, a 55YOF patient with advanced high-grade serous ovarian cancer developed pulmonary embolism (PE) approximately 2.5 months after starting Niraparib maintenance therapy (200 mg daily). The patient presented with dyspnea, chest pain, and lower abdominal pain, and laboratory tests revealed markedly elevated D-dimer (23.19 μg/ml) and fibrinogen (4.38 g/L). Computed Tomography (CT) imaging confirmed pulmonary vein thrombosis. The patient was treated with anticoagulation, Niraparib was discontinued, and she recovered without further PE symptoms (49). Another case involved a 55YOF patient with stage IVA ovarian adenocarcinoma who developed severely elevated blood pressure (170/110 mmHg) during Niraparib therapy, contributing to the later development of central variant posterior reversible encephalopathy syndrome (PRES) (50). In another report, a 49YOF case with a BRCA2 pathogenic variant and a history of ER-positive, PR-negative, HER2-negative breast cancer initially treated with Olaparib experienced disease progression. Fourth-line PARP inhibitor therapy with Talazoparib for metastatic triple-negative breast cancer (PD-L1 positive) was complicated by cardiovascular-related AEs, including DVT and DIC. DVT occurred during corticosteroid therapy and was managed with anticoagulation, but subsequent progression to DIC prevented continuation of systemic treatment (35). Real-world pharmacovigilance data have identified significant safety signals with more than twenty-time higher odd ratios of blood pressure fluctuation in patient treated with Niraparib, suggesting a strong association between the drug and hypertensive episodes (7). A systematic review and meta analysis (51), as well as FAERS analysis (52) have documented an increased risk of hypertension and thromboembolic events in patients treated with PARP inhibitors.

Neurologic Adverse Effects of PARP Inhibitors

While most neurologic symptoms like fatigue or dizziness are sometimes attributed to anemia (19, 30), true neurologic toxicities are infrequently documented. A 66YOF patient with high-grade muscle-invasive urothelial carcinoma of the bladder initially underwent cystectomy, and chemotherapy, followed by radiotherapy for recurrent pelvic disease. Genetic testing revealed a biallelic ATM mutation, prompting treatment with Talazoparib at 1mg per day, which was later dose-reduced (0.5 mg/ day) due to fatigue and dizziness not attributable to anemia. Dose reduction vanished the side effect and initial complete response was achieved after 16 months of treatment. However, due to medication unavailability, therapy was interrupted, leading to recurrence and the patient ultimately died from progressive metastatic disease (53).

In another case, a 48YOF patient with a history of long-standing epilepsy and stage IIIC high-grade serous ovarian cancer experienced a recurrence of seizures 11 weeks after initiating Niraparib maintenance therapy. This neurological event prompted temporary suspension of treatment for four weeks, during which she recovered. Niraparib was then resumed at a reduced dose with gradual titration, and the patient tolerated therapy without further neurologic complications, maintaining stable disease and a 29 months PFS. Thus, while Niraparib may not have directly caused new-onset seizures, it precipitated a recurrence of a pre-existing neurologic condition in the context of bone marrow suppression (19).

Additionally, a 55YOF patient with stage IVA ovarian adenocarcinoma, previously treated with chemotherapy and surgery, on maintenance with Niraparib was associated with PRES, presenting with generalized tonic-clonic seizures, neurological deterioration (Glasgow Coma Scale of 10), and basal ganglia/thalami hyperintensities on Magnetic Resonance Imaging. The patient was treated with anti-hypertensive therapy and immunoglobulins for 5 days, resulting in complete recovery. Niraparib was permanently discontinued. This case illustrates that PARP inhibitors like Niraparib can precipitate severe neurologic complications

such as central PRES, particularly in the context of immunotherapy and hypertension (49). Furthermore, a FAERS-based analysis reported significant associations between PARP inhibitor treatment and certain neurologic reactions, such as taste and smell disorders (ROR with Niraparib=9.17) or photosensitivity reaction (ROR with Niraparib and Rucaparib are 21.77 and 18.92, respectively) (7).

In general, neurologic symptoms, though less commonly discussed than other toxicities, are part of the broader AE spectrum of PARP inhibitors. The absence of detailed case narratives highlights the need for more individual case reporting and clinical documentation of neurologic complications in future studies.

Pulmonary Adverse Effects of PARP Inhibitors

Pulmonary adverse effects of PARP inhibitors have been reported sporadically, with interstitial lung disease (ILD) being the main manifestation. An 80-year-old woman with stage IV ovarian serous adenocarcinoma, previously treated with surgery and chemotherapy, developed ILD with diffuse ground-glass opacities (GGO) on CT scans after 15 weeks of Olaparib (300 mg twice daily), which resolved with drug discontinuation and corticosteroid plus antibiotic therapy. She later developed ILD again 15 weeks after starting Niraparib (200 mg daily), presenting with dyspnea and CT findings resembling hypersensitivity pneumonitis. Bronchoalveolar lavage confirmed a drug-induced process with elevated lymphocytes and eosinophils. The patient's condition improved after stopping Niraparib and giving corticosteroids. This case suggests cross-reactivity between Olaparib and Niraparib, cautioning against switching PARP inhibitors after ILD (54). An additional case described a 74-year-old woman with recurrent ovarian cancer who developed prolonged fever after more than a year of Olaparib therapy at 300 mg twice a day. She had no other symptoms, and initial chest X-ray was normal, but CT showed GGO and small centrilobular nodules, and ILD biomarkers were elevated (Surface Protein D (SP-D) 169 ng/mL) consistent with early ILD. Fever partially improved after discontinuing Olaparib. Short-term corticosteroid therapy led to complete resolution of CT findings and fever (55). In another case, a 49YOF patient with a BRCA2 pathogenic variant and metastatic triple-negative breast cancer (PD-L1 positive) developed drug-induced ILD while receiving fourth-line Talazoparib therapy, presented with respiratory symptoms attributed to PARP inhibitor–related pulmonary toxicity. High-dose corticosteroids were administered, resulting in partial improvement of pulmonary function and symptomatic relief. However, the severity of the lung injury required temporary discontinuation of systemic therapy, and the patient remained at risk for additional complications during ongoing treatment (35).

Two case series reported Olaparib-induced ILD in women with advanced ovarian cancer receiving 600 mg/day. In the first series, three patients aged 51, 72, and 78 presented with a spectrum of symptoms including fever, fatigue, cough, dyspnea, and hypoxemia. CT scans revealed diffuse ground-glass opacities, with one patient showing nonspecific interstitial pneumonia and organizing pneumonia patterns. Laboratory findings included elevated Krebs von den Lungen-6 (KL-6) (715 U/mL, 754U/mL, and 356U/mL, respectively), SP-D (182-231 ng/mL) and bronchoalveolar lavage fluid (BALF) lymphocytosis (up to 81%). Management strategies varied: one patient improved with Olaparib discontinuation alone, while two required corticosteroid therapy, including prednisolone taper or steroid pulse therapy. All patients ultimately had favorable outcomes, with complete resolution of symptoms and imaging abnormalities (56). The second series reported similar findings in three women aged 52, 73, and 76, who developed ILD 55–125 days after starting Olaparib. Presenting symptoms were primarily fever, with one patient also experiencing fatigue and requiring supplemental oxygen. CT scans demonstrated bilateral diffuse ground-glass opacities and fine reticulonodular opacities, predominantly in the upper lobes. BALF analysis showed marked lymphocytosis (>75%), and bacterial cultures were negative. Olaparib was discontinued in all patients, and oral prednisolone (0.5–1 mg/kg/day) was administered, leading to rapid improvement in pulmonary symptoms and imaging in two patients. The third patient recovered from ILD but later died from progressive ovarian cancer (57).

Hence, pulmonary adverse effects of PARP inhibitors, particularly Olaparib-induced ILD, are uncommon but potentially serious, often presenting with fever and ground-glass opacities, and generally respond to drug discontinuation and corticosteroid therapy. However, cross-reactivity between PARP inhibitors warrants caution when switching agents.

In summary, PARP inhibitors demonstrate diverse organ adverse effects, with 25% (11/44) reported cases involving multiple systems simultaneously, underscoring their broad and variable severity. The number of adverse effects reported by cases or case

series, categorized by affected organ systems, is illustrated in Figure 1. Severe adverse effects, defined as grade 3 or higher according to the Common Terminology Criteria for Adverse Events (58) (CTCAE), accounted for 58% (51/88) of reported events and are further detailed by specific PARP agents and organ systems in Figure 3. Hematologic toxicities were the most frequently observed severe events, followed by dermatologic complication, highlighting the need for careful monitoring across multiple organ systems during treatment. A synopsis of PARP inhibitor–associated adverse effects reported between 2022 and 2025, organized by affected organ systems, common adverse effects, severity range, and management strategies, is provided in Table 1, aiming to provide a comprehensive overview of PARP inhibitor toxicity across organ systems.

CONCLUSION

This review highlights the complex and multifaceted adverse effect profile of PARP inhibitors, with Olaparib and Niraparib being the most extensively documented in recent case reports from 2022 to 2025, while Veliparib being the least reported due to its investigational status. While PARP inhibitors are considered as targeted and generally well-tolerated alternatives to conventional chemotherapy, especially for patients with BRCA mutations or homologous recombination-deficient tumors, they are not without risk.

PARP inhibitors are associated with a range of AEs affecting multiple organ systems, reflecting the interrelated consequences of DNA repair disruption. Hematologic toxicities remain the most frequently observed and clinically significant adverse effects. Anemia is the most common, ranging from mild, self-limiting cases to severe pancytopenia and progression to MDS, particularly with Olaparib. Other cytopenias, including thrombocytopenia, leukopenia, pancytopenia, and lymphocytopenia have been reported with Niraparib and Olaparib, occasionally necessitating dose adjustments, supportive care, or treatment discontinuation. Talazoparib appears to exhibit a relatively favorable long-term tolerability profile, with some patients experiencing only mild anemia over extended treatment durations.

Dermatologic reactions, although rare, can be severe, including erythema nodosum, cutaneous vasculitis, and Sweet syndrome. Timely diagnosis and intervention, often with corticosteroids or treatment interruption, generally result in full recovery. Gastrointestinal, renal and hepatic AEs, as well as rare cardiovascular, neurologic, and pulmonary side effects, have also been reported, particularly in combination regimens or elderly patients, highlighting the need for vigilant monitoring.

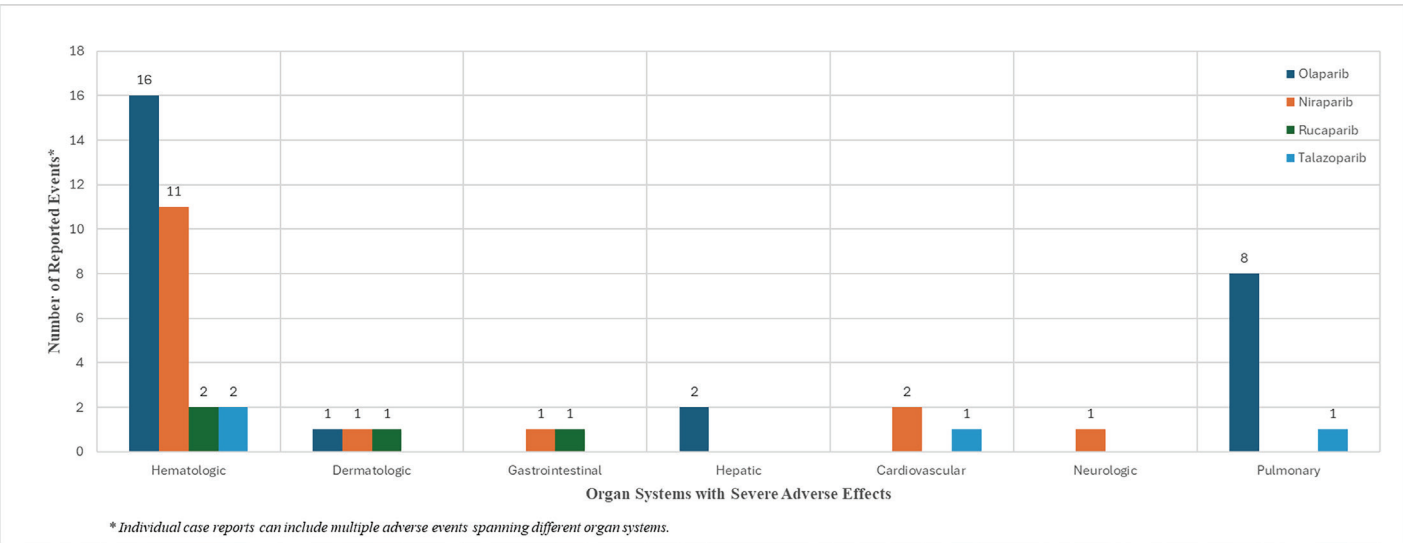

Figure 3. Number of Severe (Grade 3 and Higher) Adverse Effects Associated with PARP Inhibitors from Case Reports between 2022 and 2025.

Table 1. Summary of Adverse Effects of PARP Inhibitors from Case Reports between 2022 and 2025

Organ System	PARP Inhibitors	Reported Adverse Effects	Severity Range	Management Strategies
Hematologic	Olaparib, Niraparib, Talazoparib, Rucaparib, Veliparib.	Anemia, leukopenia/ neutropenia, thrombocytopenia, pancytopenia, lymphocytopenia, MDS, AML	Mild (grade 1) to Severe (grade 4)	Dose adjustment, treatment interruption/suspension, transfusions, corticosteroids, supportive care, discontinuation.
Dermatologic	Olaparib, Niraparib, Rucaparib.	Erythema nodosum, cutaneous vasculitis, Sweet syndrome, DRESS	Mild (rash) to Severe (immune-mediated reactions, DRESS)	Topical or systemic corticosteroids, discontinuation
Gastrointestinal	Olaparib, Niraparib, Rucaparib Veliparib.	Nausea, vomiting, diarrhea, GI bleeding	Mild to Severe (hemorrhage)	Supportive care, dose adjustment, treatment suspension, monitoring
Hepatic	Olaparib, Rucaparib.	Hepatocellular injury, immune-mediated liver injury.	Mild (enzyme elevation) to Severe (grade 4 hepatotoxicity)	Treatment interruption/ discontinuation, switch agents
Renal	Olaparib, Rucaparib.	Creatinine elevation, proteinuria, acute renal injury	Mild (non-pathogenic) to Severe (renal failure)	Monitoring, dose adjustment, discontinuation, supportive care
Cardiovascular	Niraparib, Talazoparib.	Hypertension, blood pressure fluctuation, DVT	Mild to Severe (DVT)	Monitoring, blood pressure management, discontinuation.
Pulmonary	Olaparib, Talazoparib.	ILD	Mild to Severe (ILD)	Corticosteroids, drug switch, discontinuation.
Neurologic	Niraparib, Talazoparib, Rucaparib.	Fatigue, dizziness, taste/smell disorders, photosensitivity, hypersensitivity, PRES.	Mild to Severe (PRES)	Monitoring, temporary interruption, antihypertensive therapy, discontinuation.

Notably, the mechanisms of PARP inhibitors can simultaneously impact multiple organ systems, leading to overlapping and interrelated adverse effects. This interconnected nature of AEs underscores the importance of holistic patient monitoring. Personalized treatment strategies, including pre-treatment genetic screening, routine laboratory monitoring, careful assessment of organ functions, and proactive symptom management such as dose adjustments, agent switching and supportive care, are critical to maximizing therapeutic efficacy while minimizing risk. Increased awareness of both common and rare toxicities allows clinicians to make informed decisions, individualize therapy to each patient, and ensure safe long-term use of these targeted agents.

Overall, these findings reenforce that while PARP inhibitors offer substantial clinical benefit with a more favorable safety profile than traditional cytotoxic chemotherapy, individualized management and close monitoring are essential.

In conclusion, PARP inhibitors remain a cornerstone of precision oncology. Their efficacy in HRR-deficient and BRCA-mutated cancers is supported by robust evidence, and their adverse effects, though diverse, can be effectively managed with vigilant monitoring, individualized dosing, and timely intervention, ultimately enhancing patient outcomes and quality of life. Continued research and clinical trials are critical to capture rare or newly emerging adverse effects, such as recently reported hepatic toxicities, and to refine management strategies accordingly.

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CONFLICT OF INTERESTS

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