DRUG NAME: Erdafitinib

SYNONYM(S): JNJ-42756493[1]

COMMON TRADE NAME(S): BALVERSA®

CLASSIFICATION: molecular targeted therapy

Special pediatric considerations are noted when applicable, otherwise adult provisions apply.

MECHANISM OF ACTION:

Erdafitinib is an orally administered pan-fibroblast growth factor receptor (FGFR) kinase inhibitor that targets all FGFR family members (FGFR 1, 2, 3 and 4). FGFRs are involved in cell proliferation and survival. By inhibiting the FGFR signaling pathway, erdafitinib decreases tumour cell viability. Erdafitinib has demonstrated antitumour activity in cancer cells harbouring activating FGFR genetic alterations.[2,3]

PHARMACOKINETICS:

Oral Absorption	T_{max} = 2.5 h (range 2-6 h); high-fat meal has no clinically meaningful effect on AUC and C_{max}, but delays absorption rate	
Distribution	highly bound to plasma protein, preferentially to α1-acid glycoprotein	
	cross blood brain barrier?	yes[4]
	volume of distribution	28.8 L
	plasma protein binding	99.7%
Metabolism	primary metabolized by CYP 2C9 (39%) and CYP 3A4 (20%)	
	active metabolite(s)	no information found
	inactive metabolite(s)	no information found
Excretion	primarily eliminated by fecal elimination	
	urine	19% (13% as unchanged erdafitinib)
	feces	69% (14-21% as unchanged erdafitinib)
	terminal half life	59 h
	clearance	0.36 L/h
Sex	no clinically significant differences	
Elderly	decreased clearance with increasing age (65-75 years old) does not lead to clinically meaningful effects on erdafitinib exposure	
Ethnicity	no clinically significant differences	

Adapted from standard reference[3] unless specified otherwise.

USES:

Primary uses:

*Bladder cancer

Other uses:

*Health Canada approved indication

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SPECIAL PRECAUTIONS:

Caution:
- ***central serous retinopathy*** has been reported with erdafitinib; ***ophthalmologic exams*** are recommended in all patients prior to starting treatment with erdafitinib[3]
- ability to ***drive*** and/or ***operate machinery*** may be impaired secondary to visual disturbances or eye disorders associated with erdafitinib[3]
- patients who are known to have ***CYP 2C9 *3/*3 genotype*** (poor metabolizers) may be at increased risk of erdafitinib toxicity due to the potential increase in exposure[3,5]

Carcinogenicity: No carcinogenicity studies have been conducted.[3]

Mutagenicity: Not mutagenic in Ames test. Erdafitinib was not clastogenic in mammalian *in vitro* and *in vivo* chromosome tests.[3]

Fertility: No dedicated fertility studies have been conducted. In animal toxicity studies, effects on female reproductive organs (such as necrosis of corpora lutea) were observed at exposures comparable to those expected in humans at the maximum recommended clinical doses. In male test subjects, effects on epididymides and testes were observed.[3,6]

Pregnancy: In animal studies where erdafitinib was administered during organogenesis, decreased fetal weight, fetal malformations, and embryo-fetal death were observed at exposures less than 0.1% of the expected human exposure with the maximum recommended clinical doses. Fetal malformations included major blood vessel malformations, vascular anomalies, limb malformations, and skeletal anomalies in multiple bones (e.g., vertebrae, sternebrae, ribs).[5] Pregnancy tests are recommended prior to starting treatment in female patients of reproductive potential. Contraception is recommended during treatment and for at least 1 month after the last dose for female patients of reproductive potential and male patients with female partners of reproductive potential. Male patients should not donate or store semen during treatment and for at least 1 month after the last dose of erdafitinib.[7]

Breastfeeding is not recommended due to the potential secretion into breast milk. Women should not breastfeed during treatment and for 1 month after the last dose of erdafitinib.[3]

SIDE EFFECTS:

The table includes adverse events that presented during drug treatment but may not necessarily have a causal relationship with the drug. Because clinical trials are conducted under very specific conditions, the adverse event rates observed may not reflect the rates observed in clinical practice. Adverse events are generally included if they were reported in more than 1% of patients in the product monograph or pivotal trials, and/or determined to be clinically important.[8,9]

ORGAN SITE	SIDE EFFECT
Clinically important side effects are in ***bold, italics***	
blood and lymphatic system/ febrile neutropenia	anemia (26%, severe 7%)
	leukopenia (26%)
	neutropenia (16%, severe <1%)
	thrombocytopenia (17%, severe 1%)
Eye (see paragraph following **Side Effects** table)	***blurred vision*** (17%)[5]
	cataract
	central serous retinopathy (18-22%, severe 2%); includes retinal pigment epithelial detachment (5%)

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ORGAN SITE	SIDE EFFECT
Clinically important side effects are in ***bold, italics***	
	blepharitis (3%)
	conjunctivitis (10%)
	dry eye (25%, severe 1%); increased lacrimation reported
	keratitis (5%)
endocrine	hyperparathyroidism (3%)
gastrointestinal	*emetogenic potential:* low[10]
	abdominal pain (<10%)
	constipation (27%)
	diarrhea (63%, severe 3%)
	dry mouth (39%)
	dyspepsia (7%)
	intestinal obstruction (2%)
	nausea (15%, severe 1%)
	stomatitis (56%, severe 10%)
	vomiting (10-13%)[5]
general disorders and administration site conditions	fatigue (29%, severe 1%)
	peripheral edema (10%)[5]
	pyrexia (15%, severe <1%)
hepatobiliary	hepatic cytolysis (1%)
infections and infestations	urinary tract infection (11%, severe 4%)
investigations	albumin decrease (29%)
	alkaline phosphatase increase (10-54%, severe 2%)
	ALT increase (27-46%, severe 3%)
	AST increase (22-44%, severe 2%)
	blood bilirubin increase (3%)
	creatinine increase (14-43%, severe 1%)
	weight loss (22%, severe 2%)
metabolism and nutrition	decreased appetite (27%, severe 3%)
	hypercalcemia (6-27%, severe 8%)
	hyperkalemia (24%)
	hyperphosphatemia (80%, severe 5%); see paragraph following **Side Effects** table
	hypomagnesemia (24%)
	hyponatremia (12-44%, severe 7-16%)
	hypophosphatemia (34%, severe 8%)

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ORGAN SITE	SIDE EFFECT
Clinically important side effects are in ***bold, italics***	
musculoskeletal and connective tissue	arthralgia (10%, severe 1%)
	extremity pain (13%)[5]
nervous system	dysgeusia (30%, severe <1%)
renal and urinary	acute kidney injury (5%)
	hematuria (12%, severe 2%)
	renal impairment, renal failure (3-5%)
respiratory, thoracic, and mediastinal	epistaxis (13%)
	nasal dryness (<10%)
skin and subcutaneous tissue	alopecia (25%, severe 1%)
	dry skin (27%, severe 1%)
	hand-foot skin reaction (30%, severe 10%)
	nail toxicity (70%, severe 12%); see paragraph following **Side Effects** table
	skin toxicity (<10%); includes rash, skin fissures, hyperkeratosis, exfoliation, and eczema
	pruritus (4%)
vascular disorder	vascular calcification (<1%)

Adapted from standard reference[3,7] unless specified otherwise.

Hyperphosphatemia is an on-target pharmacological effect of FGFR inhibition. Prolonged hyperphosphatemia can cause calcium precipitation and lead to soft tissue mineralization, non-uremic calciphylaxis, cutaneous calcinosis, and vascular calcification. Other complications of hyperphosphatemia may include secondary hyperparathyroidism, muscle cramps, and arrhythmia. Median onset of hyperphosphatemia is 16 days (range 6-449 days) and usually occurs within the first 4 months of treatment with erdafitinib. Assess serum phosphate at baseline and throughout treatment. Advise patients to avoid concurrent therapy with drugs that may alter serum phosphate levels (e.g., antacids and phosphate-containing supplements), especially before the initial dose modification period. Management of hyperphosphatemia may include low phosphate diet, phosphate lowering therapy, and erdafitinib dose interruption, dose reduction, and/or permanent discontinuation.[7]

Ocular toxicity, including central serous retinopathy (CSR), blurred vision, and dry eye has been reported. CSR has been reported in approximately 20% of patients treated with erdafitinib, with a median time to onset of 45 days. Reported CSR events include chorioretinopathy, retinal pigment epithelium detachment, retinopathy, and subretinal fluid. CSR may lead to visual field defect. Ophthalmologic exams are recommended prior to initiating erdafitinib and throughout treatment. Patients reporting visual symptoms during treatment such as blurred vision, floaters, flashes of light, or eye pain should be urgently referred for ophthalmologic evaluation. Dry eye prophylaxis with artificial tears or lubricating eye gels or ointments is recommended for all patients. Preservative-free eye lubricants are preferred because repeated exposure to preservatives may cause adverse effects on the ocular surface (e.g., increased inflammation) and worsen dry eye over time.[11] Depending on the severity and persistence of the adverse reaction, erdafitinib may be withheld, dose reduced, or permanently discontinued to manage symptoms. Patients experiencing visual disturbances should be instructed not to drive or operate machinery until symptoms have resolved.[3]

Nail toxicity is reported in 70% of patients treated with erdafitinib. Symptoms may include nail discolouration, nail ridging, nail infection, loosening or loss of nails (onycholysis, onychomadesis), and nail bed infection (paronychia). Median time to onset is 63 days.[12] To prevent nail toxicity, patients are advised to avoid nail-biting or cutting nails too short and to limit the use of nail polish and nail polish remover. Recommended management of paronychia includes

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diluted vinegar soaks, topical povidone-iodine, and/or topical/oral antibiotics. Management of onycholysis may require oral antibiotics if infection is present. If a hematoma or abscess develops, partial or complete nail removal may be necessary.[13] Depending on the severity and persistence of the nail toxicity, erdafitinib may be withheld, dose reduced, or permanently discontinued.[3]

INTERACTIONS:

AGENT	EFFECT	MECHANISM	MANAGEMENT
carbamazepine[3]	55% decrease in erdafitinib AUC with 22% decrease in C_{max}	strong induction of CYP 3A4 and weak induction of CYP 2C9 by carbamazepine	avoid concurrent use
fluconazole[3]	48% increase in erdafitinib AUC with 21% increase in C_{max}	moderate inhibition of CYP 2C9 and CYP3A4 by fluconazole	- if concurrent use cannot be avoided, consider erdafitinib dose reduction to the next lower dose based on tolerability[12] - monitor serum phosphate levels and watch for erdafitinib toxicity - if fluconazole is discontinued, erdafitinib may be resumed at the prior dose as tolerated[5]
itraconazole[3]	34% increase in erdafitinib AUC with 5% increase in C_{max}	strong inhibition of CYP 3A4 and P-gp by itraconazole	- if concurrent use cannot be avoided, consider erdafitinib dose reduction to the next lower dose based on tolerability[12] - monitor serum phosphate levels and watch for erdafitinib toxicity - if itraconazole is discontinued, erdafitinib may be resumed at the prior dose as tolerated[5]
metformin[3]	no clinically meaningful changes in metformin AUC and C_{max}	inhibition of OCT2 by erdafitinib	no metformin dose adjustment is required
midazolam[3]	no clinically meaningful changes in midazolam AUC and C_{max}	time dependent inhibition or induction of CYP 3A4 by erdafitinib	no midazolam dose adjustment is required

Erdafitinib is a *substrate* of ***CYP 3A4***. Strong CYP 3A4 ***inhibitors*** may increase the plasma concentration of erdafitinib. Avoid concurrent use with *strong* CYP 3A4 inhibitors if possible. If concurrent use cannot be avoided, erdafitinib dose reduction may be required. Monitor serum phosphate levels and watch for erdafitinib toxicity. If the CYP 3A4 inhibitor is discontinued, erdafitinib may be resumed at the prior dose as tolerated.[3,5]

CYP 3A4 inducers may decrease the plasma concentration of erdafitinib. Avoid concurrent use with *strong* CYP 3A4 inducers and drugs with a dual mechanism (e.g., strong CYP 3A4 induction with CYP 2C9 induction). For *moderate* CYP 3A4 inducers, if concurrent use cannot be avoided, consider gradually increasing erdafitinib dose to a maximum of 9 mg once daily. Monitor serum phosphate levels and watch for erdafitinib toxicity. If the *moderate* CYP 3A4 inducer is discontinued, erdafitinib dose may require adjustment for tolerance.[3,5,12]

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Erdafitinib is a *substrate* of ***CYP 2C9.*** *Moderate* or *strong* CYP 2C9 ***inhibitors*** may increase the plasma concentration of erdafitinib. If concurrent use cannot be avoided, erdafitinib dose reduction may be required. Monitor serum phosphate levels and watch for erdafitinib toxicity. If the CYP 2C9 inhibitor is discontinued, erdafitinib may be resumed at the prior dose as tolerated.[3]

In vitro, erdafitinib is an *inhibitor* of ***P-gp***. If erdafitinib is used concurrently with a ***P-gp substrate*** with a narrow therapeutic index, administer erdafitinib at least 6 hours before or after the substrate to mitigate the interaction.[3]

In vitro, erdafitinib is a time dependent inhibitor and inducer of CYP 3A4, an inhibitor of OCT2, and a substrate of P-gp. However, no clinically meaningful interaction is reported via these mechanisms.

SUPPLY AND STORAGE:

Oral: Janssen Inc. supplies erdafitinib as 3 mg, 4 mg, and 5 mg film-coated tablets. Store at room temperature.[3]

Additional information: Erdafitinib is supplied in bottles as follows:
- 3 mg tablets: bottles of 84 tablets (28 day supply for 9 mg daily dose);
- 4 mg tablets: bottles of 56 tablets (28 day supply for 8 mg daily dose); and
- 5 mg tablets: bottles of 28 tablets.

DOSAGE GUIDELINES:

Refer to protocol by which patient is being treated.

Adults:

BC Cancer usual dose noted in ***bold, italics***

Oral[3,14,15]*:*	***8 mg*** (range: 4-9 mg) ***PO once daily*** Recommended starting dose is 8 mg (as two x 4 mg tablets) once daily. Based on tolerability and serum phosphate levels, erdafitinib dose may be increased to 9 mg (as three x 3 mg tablets) PO once daily. Refer to protocol by which patient is being treated. Administer with food or on an empty stomach, approximately the same time every day.
Concurrent radiation:	no information found
Dosage in renal failure:	eGFR ≥30 mL/min/1.73 m^2: no adjustment required[3] eGFR <30 mL/min/1.73 m^2: no information found
Dosage in hepatic failure:	mild to moderate impairment (Child-Pugh A or B): no adjustment required[3] severe impairment (Child-Pugh C): no information found
Dosage in dialysis:	no information found

Children: safety and efficacy have not been established

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