DRUG NAME: Pemigatinib

SYNONYM(S): INCB054828[1]

COMMON TRADE NAME(S): PEMAZYRE®

CLASSIFICATION: molecular targeted therapy

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

MECHANISM OF ACTION:

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

PHARMACOKINETICS:

Oral Absorption	T_{max} = 1 h (range 0.5-6 h); time to steady state = 4 days; a high-fat and high-calorie meal had no clinically meaningful effect on pemigatinib pharmacokinetics	
Distribution	highly bound to plasma protein, predominantly to albumin	
	cross blood brain barrier?	yes[3]
	volume of distribution	235 L
	plasma protein binding	91%
Metabolism	primary metabolized by CYP3A4	
	active metabolite(s)	no information found
	inactive metabolite(s)	no information found
Excretion	primarily eliminated by fecal elimination	
	urine	13% (1% as unchanged pemigatinib)
	feces	82% (1% as unchanged pemigatinib)
	terminal half life	15.4 h
	clearance	10.6 L/h
Sex	no clinically significant differences in the pharmacokinetics of pemigatinib	
Elderly	no clinically significant differences in the pharmacokinetics of pemigatinib	
Ethnicity	no clinically significant differences in the pharmacokinetics of pemigatinib	

Adapted from standard reference[2] unless specified otherwise.

USES:

Primary uses:
*Biliary tract cancer

Other uses:
Myeloid/lymphoid neoplasms[4]

*Health Canada approved indication

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SPECIAL PRECAUTIONS:

Caution:
- patients with ***pre-existing severe renal or hepatic impairment*** require starting dose reduction[2]
- ***serous retinal detachment*** has been reported with pemigatinib; ***ophthalmologic exams*** are recommended in all patients prior to starting treatment with pemigatinib[2]
- ability to ***drive and/or operate machinery*** may be impaired secondary to visual disturbances or ocular toxicities[2]

Carcinogenicity: No carcinogenicity studies have been conducted.[2]

Mutagenicity: Not mutagenic in Ames test. Pemigatinib was not clastogenic in mammalian *in vitro* and *in vivo* chromosome tests.[2]

Fertility: no information found

Pregnancy: In animal studies where pemigatinib was administered during organogenesis, decreased fetal weight and fetal malformations were observed at exposures approximately 0.2 times the expected human exposure with clinically recommended doses. Fetal malformations included vertebral anomalies, major blood vessel variations, and reduced ossification. At exposures approximately 0.6 times the expected human exposure, pemigatinib caused 100% embryo-fetal mortality due to post-implantation loss. Pregnancy tests are recommended prior to starting treatment. In female patients of reproductive potential and male patients with female partners of reproductive potential, contraception is recommended during treatment and for at least 1 month after the last dose.[2]

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 pemigatinib.[2]

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.[5-7]

ORGAN SITE	SIDE EFFECT
Clinically important side effects are in ***bold, italics***	
blood and lymphatic system/ febrile neutropenia	anemia (43%, severe 6%)
	leukopenia (18%, severe 1%)
	leukocytosis (27%, severe 1%)
	lymphopenia (36%, severe 8%)
	thrombocytopenia (28%, severe 3%)
eye (see paragraph following **Side Effects** table)	***blurred vision*** (21%, severe 3%)[4]
	dry eye (35%, severe <1%); includes keratitis and increased lacrimation
	serous retinal detachment/retinal pigment epithelial detachment (7%, severe <1%)
	trichiasis (18%, severe 3%)[4]
gastrointestinal	*emetogenic potential:* low[8]
	abdominal pain (23%, severe 5%)
	constipation (35%, severe <1%)

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ORGAN SITE	SIDE EFFECT
Clinically important side effects are in ***bold, italics***	
	diarrhea (47%, severe 3%)
	dry mouth (34%)
	dyspepsia (24%)[4]
	gastroesophageal reflux disease (<15%)
	intestinal obstruction (2%)
	nausea (40%, severe 2%)
	stomatitis (35%, severe 5%)
	vomiting (27%, severe 1%)
general disorders and administration site conditions	fatigue (42%, severe 5%)
	peripheral edema (18%, severe 1%)
	pyrexia (18%)[4]
infections and infestations	cholangitis, infective (3%)
	urinary tract infection (16%, severe 3%)
investigations	albumin decrease (34%)
	alkaline phosphatase increase (41%, severe 11%)
	ALT increase (43%, severe 4%)
	AST increase (43%, severe 6%)
	creatinine increase (41%, severe 1%); see paragraph following **Side Effects** table
	blood bilirubin increase (26%, severe 5%)
	weight loss (16%, severe 2%)
metabolism and nutrition	decreased appetite (33%, severe 1%)
	dehydration (15%, severe 2%)
	hypercalcemia (15%, severe 2%)
	hyperglycemia (36%, severe <1%)
	hyperkalemia (12%, severe 2%)
	hyperuricemia (30%, severe 10%)
	hyperphosphatemia (60%); see paragraph following **Side Effects** table
	hypocalcemia (17%, severe 3%)
	hypoglycemia (11%, severe 1%)
	hypokalemia (26%, severe 5%)
	hyponatremia (39%, severe 12%)
	hypophosphatemia (23%, severe 12%); see paragraph following **Side Effects** table
musculoskeletal and connective tissue	arthralgia (25%, severe 6%)
	back pain (20%, severe 3%)

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ORGAN SITE	SIDE EFFECT
Clinically important side effects are in ***bold, italics***	
	extremity pain (19%, severe 2%)
nervous system	dizziness (21%)[4]
	dysgeusia (40%)
	headache (16%)
renal and urinary	acute kidney injury (2%)
respiratory, thoracic, and mediastinal	epistaxis (29%)[4]
	pleural effusion (3%)
skin and subcutaneous tissue	abnormal hair growth (<15%)
	alopecia (49%)
	dry skin (20%, severe <1%)
	hand-foot skin reaction (15%, severe 4%)
	nail toxicity (43%, severe 2%); see paragraph following **Side Effects** table
	rash (35%, severe 6%)[4]

Adapted from standard reference[2] unless specified otherwise.

Hyperphosphatemia is an on-target pharmacological effect of FGFR inhibition[9]. Prolonged hyperphosphatemia can cause calcium precipitation and lead to soft tissue mineralization. Cutaneous calcification, non-uremic calciphylaxis, and calcinosis have been reported in patients treated with pemigatinib. Median time to onset of hyperphosphatemia is 14 days and usually occurs within the first 6 months of treatment. Assess serum phosphate at baseline and periodically throughout treatment. Advise patients to avoid concurrent therapy with drugs that may alter serum phosphate levels (e.g., antacid and phosphate-containing supplements). Management of hyperphosphatemia may include low phosphate diet, phosphate lowering therapy, and/or pemigatinib dose interruption, dose reduction, or permanent discontinuation.[2]

Hypophosphatemia is also reported during pemigatinib treatment and may be a result of negative feedback effects on phosphate regulation.[9] Although grade 3-4 reductions in serum phosphate levels have been observed, no serious events have been reported in patients receiving pemigatinib. If pemigatinib treatment is interrupted or serum phosphate levels fall below the normal range, consider discontinuing phosphate lowering therapy and/or low phosphate diet.[2]

Ocular toxicity (including serous retinal detachment, blurred vision, and dry eye) has been reported with pemigatinib.[2] FGFR inhibition and its downstream effects on signaling pathways may lead to dysregulation of the outer retinal barrier causing subretinal fluid buildup.[1,9] Most cases of serous retinal detachment have been reported within the first 6 months of treatment. Ophthalmologic exams are recommended prior to initiating pemigatinib and throughout treatment. Artificial tears or other similar lubricating agents are recommended for prevention and management of dry eye.[2] Preservative-free eye lubricants are preferred because repeated exposure of the ocular surface to preservatives may cause increased inflammation and worsen dry eye over time.[10] Patients reporting visual symptoms such as blurred vision, floaters, flashes of light, or eye pain while taking pemigatinib should be urgently referred for ophthalmologic evaluation. Depending on the severity and persistence of the reaction, pemigatinib 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.[2,4]

Nail toxicity is reported in about 40% of patients treated with pemigatinib. Symptoms may include nail discolouration, nail ridging, nail infection, loosening or loss of nails (onycholysis, onychomadesis), or nail bed

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infection (paronychia). Median time to onset is 6 months.[2,9] To prevent nail toxicity, patients are advised to limit the use of nail polish and avoid nail biting or cutting nails too short. Recommended management of paronychia includes 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.[11] Depending on the severity and persistence of the nail toxicity, pemigatinib may be withheld, dose reduced, or permanently discontinued.[2]

Increased serum creatinine may occur secondary to inhibition of renal transporters of MATE1 and OCT2 by pemigatinib. Because creatinine is a substrate of MATE1 and OCT2, pemigatinib can decrease its renal tubular secretion. Glomerular function is not affected. Increased creatinine has been reported during the first cycle of pemigatinib treatment, with levels decreasing during the 7 day off-treatment period. In patients with persistent increased serum creatinine, consider alternative markers to evaluate renal function, such as calculated GFR (if not based on creatinine), cystatin C, or BUN.[2,4]

INTERACTIONS:

AGENT	EFFECT	MECHANISM	MANAGEMENT
esomeprazole[2]	no clinically meaningful changes in pemigatinib exposure	pH-dependent solubility of pemigatinib	no action is required when pemigatinib is taken concurrently with a proton pump inhibitor
grapefruit[2]	may increase plasma level of pemigatinib	may inhibit CYP 3A4 metabolism of pemigatinib in the intestinal wall	avoid grapefruit and grapefruit juice for the duration of treatment with pemigatinib
itraconazole[2,4]	88% increase in pemigatinib AUC and 17% increase in C_{max}	strong inhibition of CYP 3A4 by itraconazole	if concurrent use cannot be avoided, reduce pemigatinib dose to next dose level
ranitidine[2]	no clinically meaningful changes in pemigatinib exposure	pH-dependent solubility of pemigatinib	no action is required when pemigatinib is taken concurrently with H_2 antagonists
rifampin[2]	85% decrease in pemigatinib AUC and 17% increase in C_{max}	strong induction of CYP 3A4 by rifampin	avoid concurrent use

Pemigatinib is a *substrate* of ***CYP 3A4***. CYP 3A4 ***inhibitors*** may increase the plasma concentration of pemigatinib. Avoid concurrent use with moderate or strong CYP 3A4 inhibitors. If concurrent use cannot be avoided, reduce pemigatinib dose to next dose level. CYP 3A4 ***inducers*** may decrease the plasma concentration of pemigatinib. Avoid concurrent use with moderate or strong CYP 3A4 inducers.[2]

In vitro, pemigatinib is a *substrate* of P-gp and BCRP. However, P-gp or BCRP inhibitors are not expected to affect pemigatinib exposure. Pemigatinib is an inhibitor of P-gp, OCT2, and MATE1. Inhibition of OCT2 and MATE1 may increase serum creatinine.[2]

SUPPLY AND STORAGE:

Oral: Innomar Strategies (for Incyte Corporation) supplies pemigatinib as 4.5 mg, 9 mg, and 13.5 mg uncoated immediate-release tablets. Store at room temperature.[2,12]

Additional information: Tablets are packaged in blister cards in cartons containing a 14-day supply.[2]

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DOSAGE GUIDELINES:

Refer to protocol by which patient is being treated.

Adults:

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

Oral:	Cycle Length	
	3 weeks[2,13,14]:	***13.5 mg*** (range 4.5-13.5 mg) ***PO once daily for 14 consecutive days*** starting on day 1 (total dose per cycle 189 mg [range 63-189 mg]) Administer with food or on an empty stomach, at about the same time every day.
	n/a[4]:	13.5 mg PO once daily
Concurrent radiation:	no information found	
Dosage in renal failure:	eGFR ≥30 mL/min: no adjustment required[2] eGFR <30 mL/min: reduce starting dose to 9 mg once daily[2]	
Dosage in hepatic failure:	total bilirubin ≤3 x ULN: no adjustment required[2] total bilirubin >3 x ULN: reduce starting dose to 9 mg once daily[2]	
Dosage in dialysis:	intermittent hemodialysis (in end-stage renal disease): no adjustment required[2]	
Children:	safety and efficacy have not been established	

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