Tab. 2. *Overview of the aetiology of magnesium deficiency in pregnancy or in the preconception period*

Aetiology	General population and pregnant women	Detailed information
genetic aetiology	G	hypercalciuric hypomagnesaemias[40]
	G	Gitelman-like hypomagnesaemias[40]
	G	mitochondrial hypomagneasemias[40]
	G	other genetic hypomagnesaemias[40]
aetiology of use of addictive substances	G	addictive substance use (heroin, morphine, cocaine, nicotine, alcohol, caffeine, or other addictive substances)[41]
nutritional deficiency or overnutrition	G	unmet nutritional or dietary requirements[14]
	G	parenteral nutrition[14, 42]
	P	malnutrition[3]
	G	starvation[42]
	G	diet high in fat or sugar[43]
gastrointestinal aetiology	G	fistulas[14], vomiting[14]
	G, P	diarrhoea[3, 14, 42]
	G, P	malabsorption[3, 14]
	G	acute pancreatitis[14, 42] (Note: can also be classified into section- transfer from the extracellular to the intracellular compartment)
	G	
	G	gastric bypass surgery[42]
	G	liver disorders[43]
renal losses	G	■ familial: genetic aetiology[14]
	G	■ acquired: from therapeutic agents[14]
	G	■ other acquired tubular dysfunctions and diseases[42, 44]
endocrine aetiology	G, P	hypercalcaemia[3, 14, 44] (reduced NaCl reabsorption)
	G	■ primary hyperparathyroidism[44]
	G	■ malignant hypercalcaemia[44]
	G	hyperthyroidism[44]
	G, P	hyperaldosteronism (reduced NaCl reabsorption)[3, 44]
	G, P	osmotic polyuria – poorly controlled diabetes mellitus[3, 44]
transfer from the extracellular to the intracellular compartment	G	treatment of diabetic ketoacidosis with insulin[42]
	G	refeeding syndrome[14, 42, 44]
	G	correction of metabolic acidosis[42, 44]
	G	ethanol withdrawal syndrome[42]
	G	hungry bone syndrome[42] – a state of profound hypocalcaemia[14]
	G	catecholamine excess[44]
	G	significant blood transfusion[44]
use of therapeutic agents	G	proton pump inhibitors[14, 42]
	G	aminoglycoside antibiotics[14, 42, 44]
	G, P	loop and thiazide diuretics[3, 14, 42 ,44]
	G	monoclonal antibodies – cetuximab and others[42]
	G	amphotericin B[14, 42]
	G, P	cytotoxic drugs: cisplatin[3, 14, 42, 44]
	G	certain antitubercular drugs[44]
	G	beta-adrenergic agonists: theophylline, salbutamol[44]
	G	digitalis[42]
	G	antacids[43]
	G	laxatives[43]
pregnancy and puerperal aetiology	P	excessive lactation[3]
other factors	G	stress[45]
	G	sweating[43], exercise[43]
	G	oestrogen therapy[43]
	G	low salt intake[43]
	G	low selenium intake[43]
	G	vitamin B6 deficiency[43]
	G	vitamin D hypervitaminosis or hypovitaminosis[43]

Note: endocrine aetiology can overlap with renal aetiology.
Sign: G indicates that information was mainly retrieved from available information from the general population.
Sign: P indicates causes of hypomagnesaemia relevant to pregnancy data.

Assessment of magnesium status in pregnancy

To evaluate magnesium status, serum magnesium concentration (5) can be used, or other methods such as measuring magnesium concentration in erythrocytes, saliva, and urine. Laboratory tests and clinical assessment are recommended for overall evaluation (22). According to Morton, A., (2018), serum magnesium concentrations in the preconception time range from 0.62 to 0.95 (mmol/l), and in the first, second, and third trimesters, their values are 0.65–0.9 (mmol/l), 0.62–0.9 (mmol/l), and 0.45–0.9 (mmol/l), respectively (3).

Maternal hypomagnesaemia and clinical manifestations

The deficiency, depending on the rate and factors contributing to magnesium loss (11), can manifest as nonspecific (11) and can be similar to other electrolyte imbalances (11), but can also manifest specifically.