

Digoxin

Come from digitalis a.k.a a Foxglove plant
 family Scrophulariaceae.
 a.k.a a figwort family
 or Plantaginaceae.

↑ use contractility Low TI Mostly use is digoxin
 use in HF Digiroxin: seldom use

MOA

Regula. of cytosolic Ca^{2+} conc.

↑ use contractility

Neurohormonal Inhibition

Inhibit Na^+/K^+ ATPase → ↑ use Na^+ inside the cell
 (act on M_2 receptor)

↑ use force of contrac.

Low doses

↑ use Na^+ exchange for Ca^{2+}
 by Na^+/Ca^{2+} exchanger, as Na^+ is in excess amount inside the cell
 so Ca^{2+} is exchanged for Na^+ (out)

↑ CO
 ↑ vagal stim. (release Ach)
 so HR & O_2 demand ↓

↓ symph stim
minimal effect

more Ca^{2+} avail for next contractility cycle.
 This raise potential from -90mV → -70mV
 (which ↑ risk of arrhythmia as cell become easily excitable)

↓ AV conduction thus useful for ATRIAL FIBRIL (1-2mg/ml).
 This is the reason, low serum Digoxin conc. is targeted in HFrEF 0.5-0.8mg/ml

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THERAPEUTIC USES

- Severe HFrEF after initiation of ACE, β -bloc & Diuretic (ABD) BAD
- 0.5-0.8mg/ml
- High conc. = ↑ mortality ↓ admissions

AFib/flutter + Diastolic or right sided HF
 (if both are present) otherwise NOT use in alone RSHF

For mild → moderate HF (ACE, β -bloc, Aldosterone anta, Vaso & renal dilators e.g. Nitrates (veno) Hydralazine (arterio) and diuretics are used so Digoxin NOT req.

KINETICS

- Oral + IV
- Substrate of P-glycoprotein so CAV = ↑ use digoxin level (clarithro, vera, Amioda).

large Vd (dose given acc. to lean body mass)

LD require in AFib

$t_{1/2}$ 30-40hrs

ADRs

Toxicity = N/v/Anorexia

- Blurred or yellow vision (xanthopsia)

Sever toxic = ventric tachycardia

Manag: - DigiFab & antiarrhyth

Caution = drug that ↓ AV cond verapamil, Dilti, β -blocker [vera has block heart (dil)]

Manag: Discontinue Digoxin

- Monitor K^+ level esp if co-adm Thiazid/loop so replenish K^+ level in case of ↓ use