Grupo CTO
Medicina

Impugnaciones MIR

Pregunta 139

Bibliografía:

Author:Naomi DL Fisher, MDSection Editor:George L Bakris, MDDeputy Editor:John P Forman, MD, MSc

Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: Jan 2019. | This topic last updated: Dec 05, 2017.

- Up to date (acción nefrotóxica de los aminoglicósidos)

Acute kidney injury (AKI) due to acute tubular necrosis is a relatively common complication of aminoglycoside therapy, with a rise in the serum creatinine concentration of more than 0.5 to 1 mg/dL (44 to 88 micromol/L) or a 50 percent increase in serum creatinine concentration from baseline occurring in 10 to 20 percent of patients [1,2]. Aminoglycosides are freely filtered across the glomerulus; almost all of the drug is then excreted, with 5 to 10 percent of a parenteral dose being taken up and sequestered by the proximal tubule cells (PTCs), where the aminoglycoside can achieve concentrations vastly exceeding the concurrent serum concentration [3].

The intracellular accumulation of aminoglycosides is confined primarily to the S1 and S2 segments of the proximal tubule. However, following renal ischemia, the S3 portion is also a site of intracellular aminoglycoside concentrations. AKI can occur even if drug levels are closely monitored [4].

- Up to date (efecto de ATII sobre la eferente)

ANGIOTENSIN II AND AUTOREGULATION OF GFRAs the renal perfusion pressure is diminished (due for example to antihypertensive therapy), the kidney is initially able to maintain both blood flow and glomerular filtration via the phenomenon of autoregulation (figure 1) [7]. The first part of the autoregulatory response is decreased afferent (precapillary) arteriolar tone, thereby allowing more of the systemic pressure to be transmitted to the glomerulus. Afferent dilatation is mediated both by tubuloglomerular feedback and by a direct myogenic response. With more marked reductions in renal perfusion pressure, renin release is stimulated; the ensuing increase in angiotensin II production maintains both intraglomerular pressure and the glomerular filtration rate (GFR) via a preferential increase in resistance at the efferent arteriole [7]. The net effect is that the GFR and renal blood flow do not begin to fall until these autoregulatory changes in arteriolar resistance are maximized.