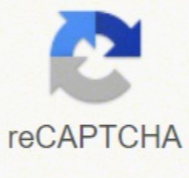
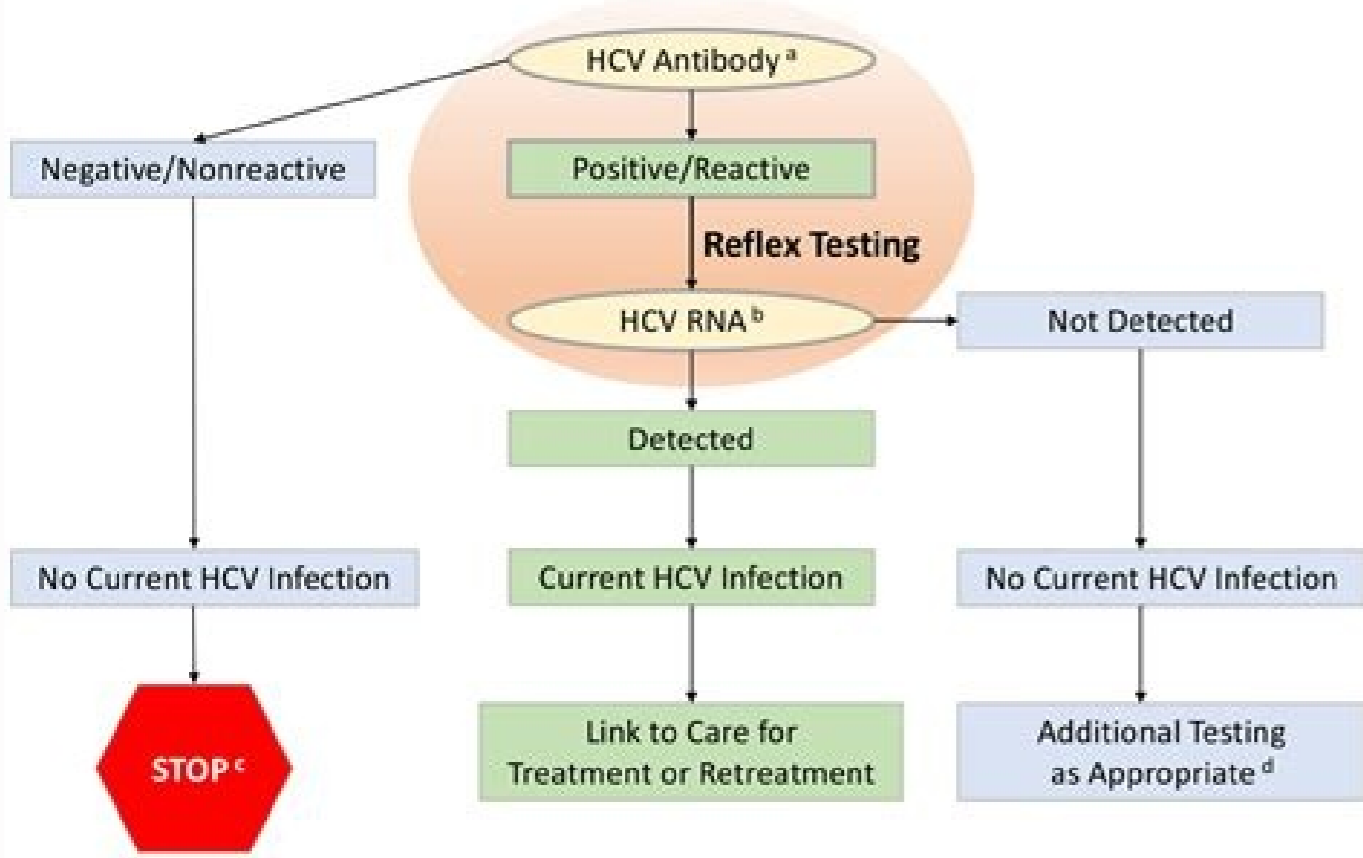
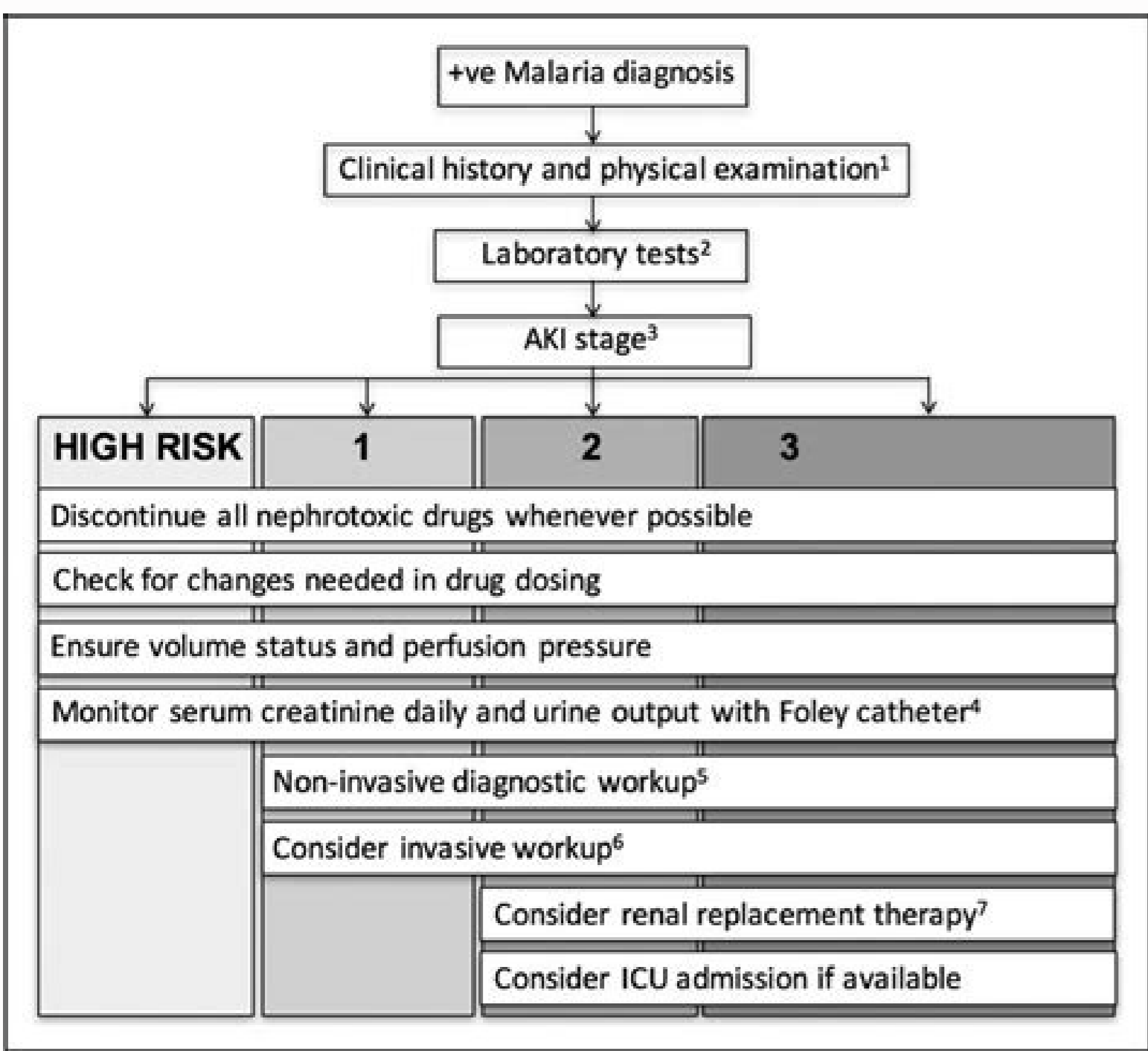




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**Next**



- Source: Adapted from the following references:
  - 1. WHO, Recommendations on treatment of hepatitis C (2014).
  - 2. WHO, Recommendations on treatment of hepatitis C (2014).
  - 3. WHO, Recommendations on treatment of hepatitis C (2014).
  - 4. WHO, Recommendations on treatment of hepatitis C (2014).
  - 5. WHO, Recommendations on treatment of hepatitis C (2014).
  - 6. WHO, Recommendations on treatment of hepatitis C (2014).
  - 7. WHO, Recommendations on treatment of hepatitis C (2014).

Product	Presentation	Posology
<b>Paragard® (paritaprevir)</b>	Tablets containing 400 mg of paritaprevir	One tablet once daily
<b>Sofosbuvir</b>	Tablets containing 400 mg of sofosbuvir	One tablet once daily
<b>Sofosbuvir/velpatasvir</b>	Tablets containing 400 mg of sofosbuvir and 100 mg of velpatasvir	One tablet once daily
<b>Sofosbuvir/velpatasvir/Voxilaprevir</b>	Tablets containing 400 mg of sofosbuvir, 100 mg of velpatasvir and 100 mg of voxilaprevir	One tablet once daily
<b>Glecaprevir/pibrentasvir</b>	Tablets containing 300 mg of glecaprevir and 100 mg of pibrentasvir	Three tablets once daily
<b>Directly-acting drugs or drug combinations</b>		
<b>Sofosbuvir/sofosbuvir</b>	Tablets containing 400 mg of sofosbuvir and 90 mg of sofosbuvir	One tablet once daily
<b>Paragard®/sofosbuvir/sofosbuvir</b>	Tablets containing 400 mg of paritaprevir, 100 mg of sofosbuvir and 100 mg of sofosbuvir	Two tablets once daily
<b>Sofosbuvir/sofosbuvir</b>	Tablets containing 200 mg of sofosbuvir and 100 mg of sofosbuvir	One tablet twice daily (morning and evening)
<b>Glecaprevir/pibrentasvir</b>	Tablets containing 100 mg of glecaprevir and 33 mg of pibrentasvir	One tablet once daily

Kdigo hcv guidelines 2018.

Last Update: 3, 2021 Prerenal Etiology causes include any conditions that leads to decreased renal perfusion. Prolonged Renal Injury leads to intrinsic injury, since the decrease in renal perfusion cause tubular necrosis. Intrinsic acute kidney damage Intrinsic causes Intrinsic causes include any condition leading to severe kidney damage to the direct kidneys. Severe Renal Causes Acute renal causes include any condition that results in bilateral obstruction of urinary flow from the renal pelvis to the urethra. While the contralateral kidney remains intact, Patients with unilateral obstruction of the urethra typically maintain normal levels of creatinine. Symptoms of the pathology General vision of the four phases of the features Characteristic of aki phase (some patients can not go through all phases) Event of initiation of duration (renal injury) Symptoms of the underlying disease causing AKI may be present. Oliguric/diuretic phase Recovery from renal function and normalizing urine production. Substratives and variants Epidemiology of the classical features: cause the property Ta 85% of the intrinsic location of the AKIs The rectum segment of the proximal tube and the rectum segment of the distal tube (i.e., the thick ascending limb) are particularly susceptible to ischemic damage. The coiled segment of the jejunum is particularly susceptible to ischemic lesions. Damage caused by toxins. Ischemic etiology: The pre-renal occurs second to decrease of renal blood flow. TACO: The pre-renal occurs directly due to nephrotic substances. Classic features: the same as aki (see definitions & benign and four phases of AKI above) Diagn (see definitions & benign) induced by the contractor & Diagnostic management & Avoid the co-administration of RAAS inhibitors & NSAIDs in patients with reduced renal perfusion (e.g., congestive heart failure, of the artisan) Because doing so may significantly decrease your GFR. Treatment of underlying causes [7][8][9] The longer the underlying cause is present, the more the chance that AKI will progress to renal failure and/or CKD. Treat potential AKI causes early. Support and follow-up care The goal of support care is to avoid more kidney and potentially aggravating factors, support adequate renal perfusion and ensure early identification and treatment of complications. Medicines and nephrotoxic substances [9] The calculation of eGFR using conventional equations did not foresee the true GFR in patients with AKI. Reevaluate the Daily GFR based on patient urine production and creatinine path only. Imaging studies are not preferred by contractors, if possible. When using iodinated contrast, needed for a study or chronic diagnosis procedure (e.g. for stemi treatment), the lowest dose of classical diagnosis should be used. Volume status and blood pressure [8] [9] AKI patients are at high risk of developing fluid overload, which may compromise renal function and may increase mortality. Avoid the resurgence of aggressive fluid in patients who are not volume responsive. Consider Day 0 loop ties only in patients with signs of fluid overload. Diuretic Actions should not be routinely used to improve urine production in patients with AKI due to lack of benefit and potential for damage. [7] Parental fluid selection [8] [9] [30] The use of balanced fluid solutions IV has been associated with lower mortality and better kidney results in comparison to the use of normal saline solutions in patients with AKI. The distances of the electrolyte and the base get frequent laboratory studies (at least di) to monitor the presence of metabolic complications and treatment response (e.g. improvement in creatinine levels). Consider urgent renal replacement therapy for patients with refractory ranges of electrolytes or acid base. Additional considerations The risk of GI can be increased in aki due to urinary platelet dysfunction. [37] Consider a nutritional query for all patients with aki. [35] follow-up follow-up Inform patients about the management of the medicine and the prevention of the AKI. Monitoring creatinine is rich, eGFR, blood pressure and weight after discharge. [38][40] Ensure that patients in need of continuous renal replacement therapy have access to outpatient analysis services. Consider and refer to the monitoring of outpatient nephrology in patients with significant residual renal impairment (i.e. GFR

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