Managing Hepatitis C in Non-Dialysis Chronic Kidney Disease

Hepatitis C and Chronic Kidney Disease (CKD)
- More prevalent among patients with CKD than in the general population.\(^1,2\)
- A recognized cause of progression to kidney failure\(^3,4\)
- Associated with reduced survival in the CKD population\(^1,3\)

Associations with Hepatitis C Virus (HCV) Infection, Mortality, and Decreased Kidney Function\(^2\)

Risk Factors for HCV Testing\(^4,5\)
- Born from 1945 through 1965
- Injected illicit drugs
- Clotting factor concentrates made prior to 1987
- Blood transfusions or solid organ transplants prior to July 1992
- Long-term hemodialysis treatment (including history)
- Healthcare-related exposure to HCV-infected blood
- Recipients of blood or organs from a donor later testing HCV positive
- HIV infection
- Signs or symptoms of liver disease (e.g., abnormal liver enzyme tests)
- Children born to HCV-positive mothers (test after age 18 months of birth)
- Receiving a tattoo in an unregulated setting
- Incarceration

Recommended Testing Sequence for Identifying Current HCV Infection\(^6\)

* For persons who might have been exposed to HCV within the past 6 months, testing for HCV RNA or follow-up testing for HCV antibody is recommended. For persons who are immunocompromised, testing for HCV RNA can be considered.

† To differentiate past, resolved HCV infection from biologic false positivity for HCV antibody, testing with another HCV antibody assay can be considered. Repeat HCV RNA testing if the person tested is suspected to have had HCV exposure within the past 6 months or has clinical evidence of HCV disease or if there is concern regarding the handling or storage of the test specimen.
**HCV Treatment**

Goal is sustained virologic response (SVR), defined as undetectable HCV RNA 12 weeks after treatment is complete.

Consider HCV genotype, extent of liver damage, CKD stage, transplant candidacy, prior HCV treatment, patient comorbidities, and the benefits and risks of antiviral treatment itself.

**Direct Acting Antivirals (DAAs)**

DAAs have been the focus of clinical research in recent years for their improved efficacy (90-100% SVR in most cases) and better tolerability compared to interferon-based therapy.1,3

Typically, multiple DAA classes are used in combination (NS5a inhibitor and/or protease inhibitor and/or polymerase inhibitor) to target different aspects of the HCV genome.

In patients with an eGFR >30 mL/min/1.73m², all licensed DAA regimens can be used.3,5

<table>
<thead>
<tr>
<th>Name</th>
<th>Class/Description</th>
<th>Genotype†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbasvir-Grazoprevir</td>
<td>-Elbasvir: NS5A inhibitor -Grazoprevir: NS3/4A protease inhibitor</td>
<td>HCV GT 1 or 4</td>
</tr>
<tr>
<td>Glecaprevir-Pibrentasvir</td>
<td>-Glecaprevir: NS3/4A protease inhibitor -Pibrentasvir: NS5A inhibitor</td>
<td>HCV GT 1-6</td>
</tr>
<tr>
<td>Ombitasvir-Paritaprevir-Ritonavir and Dasabuvir</td>
<td>-Ombitasvir: NS5A inhibitor -Paritaprevir: NS3/4A protease inhibitor -Ritonavir: CYP3A inhibitor -Dasabuvir: NS5B polymerase inhibitor</td>
<td>HCV GT 1</td>
</tr>
<tr>
<td>Ombitasvir-Paritaprevir-Ritonavir</td>
<td>-Ombitasvir: NS5A inhibitor -Paritaprevir: NS3/4A protease inhibitor -Ritonavir: CYP3A inhibitor</td>
<td>HCV GT 4</td>
</tr>
<tr>
<td>Ledipasvir-Sofosbuvir*</td>
<td>-Ledipasvir: NS5A inhibitor -Sofosbuvir: NS5B polymerase inhibitor</td>
<td>HCV GT 1, 4, 5, or 6</td>
</tr>
<tr>
<td>Sofosbuvir-Velpatasvir*</td>
<td>-Sofosbuvir: NS5B polymerase inhibitor -Velpatasvir: NS5A inhibitor</td>
<td>HCV GT 1-6</td>
</tr>
<tr>
<td>Sofosbuvir-Velpatasvir-Voxilaprevir*</td>
<td>-Sofosbuvir: NS5B polymerase inhibitor -Velpatasvir: NS5A inhibitor -Voxilaprevir: HCV NS3/4A protease inhibitor</td>
<td>HCV GT 1-6</td>
</tr>
</tbody>
</table>

* Sofosbuvir has significant renal elimination. Sofosbuvir-containing regimens not licensed for use in patients with a GFR <30 mL/min/1.73m². Other currently approved DAAs are not eliminated by the kidneys.8

† Consult Package Insert for specific indications

Disclaimer: Information contained in this National Kidney Foundation educational resource is based upon current data available at the time of publication. Information is intended to help clinicians become aware of new scientific findings and developments. This educational resource is not intended to set out a preferred standard of care and should not be construed as one. Neither should the information be interpreted as prescribing an exclusive course of management.

References