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Interface to exchange data with Autodesk Revit now included at no ...2021. 11. 22. - SKM PowerTools 7.0.4.6 could be downloaded from the developer's website when we last checked. We cannot confirm if there is a free download of this software available. The software lies within Photo & Graphics Tools, more precisely 3D Design. Some of the software aliases include "PowerTools For Windows2", "SKM PowerTools" Welcome to SKM Software, the India's leading low cost web design company. Pandua, Hooghly, Kolkata, West Bengal, India 712149Query for Failed Equipment in any Scenario. Currently, a query can only be performed that returns components that pass or fail in the current scenario. It would be extremely helpful if there was an attribute that allowed one to make a query that returns a component that fails in ANY scenario. For example, if I am currently in data visualizer in ... Familial hypercholesterolemia (FH) is a common yet underdiagnosed autosomal dominant disorder that affects ≈1 in 220 individuals globally.1 FH is characterized by lifelong elevation of low-density lipoprotein cholesterol (LDL-C) and if untreated leads to early-onset atherosclerosis and increased risk of cardiovascular events. Affected men and women who are untreated have a 30% to 50% risk of a fatal or nonfatal cardiac event by ages 50 and 60 years, respectively.1The most common causes of FH are pathogenic variants of the LDL receptor (LDL-R) gene, which are responsible for 85% to 90% of genetically confirmed FH. Pathogenic variants of the apolipoprotein (ApoB) gene, resulting in decreased binding of LDL to the LDL-R, or gain-of-function mutations in the gene for proprotein convertase subtilisin/kexin 9 (PCSK9), resulting in increased destruction of LDL-R, are responsible for 5% to 15% and 1% of cases of FH, respectively.2 Autosomal recessive FH, caused by homozygous mutations in the LDL-R adaptor protein-1, is associated with a mild homozygous FH (HoFH) phenotype and is beyond the scope of this review.3With the exception of HoFH, FH is generally a silent disease. HoFH typically presents with pathognomonic physical findings in childhood, including xanthelasma, tendon xanthomas, and corneal arcus. By contrast, in the Spanish Familial Hypercholesterolemia Cohort study, xanthomas and corneal arcus were present in 26 000 individuals suggest that at any given LDL-C level, having an identified FH mutation is associated with significantly higher cardiac risk than an individual with the same LDL-C but no apparent pathogenic FH mutation.21 In this study, individuals with an LDL-C level ≥190 mg/dL (4.9 mmol/L) and no pathogenic FH mutation were at a 6-fold higher risk of CAD than the reference group with an LDL-C ≤130 mg/dL. However, individuals with an LDL-C level ≥190 mg/dL (4.9 mmol/L) and a pathogenic FH mutation were at a 22-fold higher risk than the reference group.21 possibly reflecting greater atherogenicity of lifelong LDL-C elevation in FH compared with LDL-C elevation acquired later in life.Although there are no internationally agreed-upon criteria for the diagnosis of FH, useful diagnostic criteria have been developed. The main diagnostic tools used for FH include the US Make Early Diagnosis to Prevent Early Death (MEDPED) criteria (Table 1),22 the UK Simon Broome system (UK FH Register criteria; Table 2),23 the Dutch Lipid Clinic Network criteria (Table 3),24 and the National Lipid Association expert panel recommendations (Table 4).25, 26 The Dutch Lipid Clinic Network, which uses a scoring system to predict the likelihood of an index patient having FH, and the Simon Broome criteria are the only tools that incorporate genetic test results into their algorithm. In the Simon Broome criteria, a positive genetic test is sufficient for a definitive diagnosis of FH, while in the Dutch Lipid Clinic Network criteria, a positive genetic test should be accompanied by an additional measure (eg, elevated LDL-C levels) to fulfill the definite diagnosis criteria. Although the existing diagnostic tools differ from each other, both in structure and the cut-off values of the LDL-C level necessary for diagnosis, their predictive values are comparable.25 Among these diagnostic strategies, the Dutch Lipid Clinic Network criteria provide the most detailed assessment of the likelihood of a diagnosis of HoFH and are sometimes favored by insurance providers for the purpose of denial or approval of treatment with PCSK9 inhibitors among patients with FH. More recently, the American Heart Association proposed a simple set of criteria; however, these criteria have not yet been widely accepted or implemented (Figure 2).27Table 5 highlights some of the differences between these diagnostic criteria. The Familial Hypercholesterolemia Foundation, an organization dedicated to improving the awareness, diagnosis, and management of FH, has developed a mobile application28 that can assist both patients and providers with diagnosing FH. A special flow chart to detect FH in children has been developed and tested by the European Atherosclerosis Society consensus panel.29FH is Diagnosed if Total Cholesterol Exceeds These Cut-Off Points in mg/dL (mmol/L)Age, yFirst-Degree Relative With FHSecond-Degree Relative With FHThird-Degree Relative With FHGeneral Population7.5 mmol/L (290.0 mg/dL) (or when available, LDL-C >4.9 mmol/L [189.5 mg/dL])In pediatric patients: TC >6.7 mmol/L (259.1 mg/dL), or LDL-C >4 mmol/L (154.7 mg/dL), ANDDefiniteTendon xanthoma in the patient or first/second-degree relative, OR alternatively:Presence of LDL-R, ApoB, or PCSK9 mutationIn adults: TC >7.5 mmol/L (290.0 mg/dL) (or when available, LDL-C >4.9 mmol/L [189.5 mg/dL])In pediatric patients: TC >6.7 mmol/L (259.1 mg/dL), or LDL-C >4 mmol/L (154.7 mg/dL), ANDPossibleFamily history of MI 400 mg/dL (10.3 mmol/L), or LDL-C >310 mg/dL (8.0 mmol/L) with 1 high-risk feature, or LDL-C >190 mg/dL (4.9 mmol/L) with 2 or more high-risk features.FH patients with clinical ASCVD or evidence of advanced subclinical ASCVD are also considered to have severe FH. Markers of subclinical ASCVD are defined as a coronary artery calcium score >100 Agatston units and/or >75th percentile, or computed tomography angiography with at least 1 obstructive lesion (>50%) or multivessel lesions (1000 children with FH whose Lp(a) levels were >30 mg/dL (≈45 nmol/L) demonstrated a 1.45-times higher incidence (95% CI 0.99-2.13; P=0.05) of having a parent with FH and premature cardiovascular disease (CVD).44 Therefore, Lp(a) levels should be measured in the initial evaluation of patients with FH.4544TreatmentDietary and lifestyle modifications are the starting points for LDL-C lowering in patients with FH, but multidrug treatment is often required to achieve adequate LDL-C levels (Figure 4). In addition, all patients with FH should be counseled on the importance of not smoking or vaping, regular exercise, and maintaining a healthy body weight. ASCVD risk factors and comorbidities, such as hypertension and diabetes mellitus, should be treated.Pediatric PatientsThere are currently no long-term studies in children with FH that establish a reduction in vascular events in association with early initiation of LDL-C-lowering treatment. Results from 1 study demonstrated that children with FH have significantly greater carotid intima-media thickness than their unaffected siblings, which may be detectable as early as 8 to 10 years of age.46 During longitudinal follow-up, the difference in carotid intima-media thickness between FH and non-FH siblings progressively increased. Results from another study demonstrated small amounts of coronary artery calcification (indicative of advanced plaque) in some adolescents with FH.27 In addition, the results from another study showed that treatment with pravastatin for 2 years in children with FH aged 8 to 18 years resulted in stabilization or possible regression of carotid intima-media thickness.47 On the basis of these findings, as well as the documented long-term safety of statins in children over 10 years of follow-up48 and the Food and Drug Administration (FDA) approval of all 7 statins for the treatment of children with FH, treatment with statins (usually at reduced doses) is recommended beginning between the ages of 8 and 10 years in children with FH. Furthermore, the proven benefits of statins for ASCVD prevention and treatment of FH in adults support their use in this population.49Until recently there were no long-term studies in children with FH establishing a reduction in vascular events in association with early initiation of LDL-lowering therapy. We now have 20-year follow-up data on 214 children with genetically confirmed FH documenting that statin treatment during childhood slows the progression of carotid intima-media thickness. Additionally, childhood statin therapy has now been shown to reduce cardiovascular events and cardiac death assessed through age 39. As compared with their FH affected parents (n = 156), who did not receive statins as children, the incidence of cardiovascular events and death from cardiovascular causes, was much lower in FH patients treated since childhood (1% versus 26% and 0% versus 7%, respectively). These new data affirm the importance of initiating statins in childhood in the setting of FH.50Therapies for Pediatric Patients with FHA recently proposed LDL-C goal in pediatric patients is

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