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Direct oral anticoagulants in management of antiphospholipid syndrome

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The antiphospholipid syndrome (APS) is an autoimmune disorder characterized by venous and arterial thrombosis and recurrent fetal losses, frequently accompanied by a moderate thrombocytopenia and the presence of persistent circulating anti-phospholipid antibodies (aPL). The medical management of patients with APS aims mainly at avoiding the thrombotic and/or obstetric recurrences. To reach that, the current mainstay of treatment are: Bridge therapy for at least 5 days with heparin (un-fractionated or low molecular weight heparin) followed by long-term anticoagulation with vitamin K antagonist (VKA) such as warfarin with a recommended target international normalized ratio (INR) of 2.5. The intensity of continuous anticoagulation is still debated. Treatment with VKA is complicated because it has several pitfalls including numerous food and drug interactions (i.e., immunosuppressive agents such as azathioprine), which require frequent INR monitoring. Furthermore, the effective evaluation of the anticoagulation effect may be difficult by the variable response of thromboplastin reagents to aPL (particularly LA), that would make the estimation of anticoagulation intensity with prothrombin time (PT)/INR uncertain. To overcome these and other limitations, a group of relatively new class of drugs that inhibit a single enzyme of the coagulation cascade called direct oral anticoagulants (DOACs) has been introduced. Major phase III prospective and randomized controlled trials (RCT) have shown the efficacy and safe profile of DOACs for venous thromboembolism (VTE) treatment. However, these results are not generalizable to patients with APS, despite these trials probably included patients with this syndrome. There are many available case reports and case series that support the use of DOAC therapy for secondary thrombo prophylaxis for APS patients with previous VTE who require an INR target of 2-3. The use of DOACs in patients with previous arterial thrombosis or in patients requiring a target INR >3 is still matter of discussion. It is unclear if DOACs can replace warfarin for the long-term secondary thrombosis prevention in APS patients with VTE. The necessity for controlled outcome trials of DOACs in APS patients will depend on the results of the ongoing trials.

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3DCRT versus RapidArc treatment for breast cancer: Dosimetric study

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The aim of this study is to compare 3DCRT to RapidArc planning systems using LNAC of 6 MV, 15 MV and 18 MV in terms of dosimetric outcomes of iso-dose distribution, dose volume histogram (DVH), PTV and at risk organs in 6 patients with breast cancer. Plans were created for 6 patients with breast cancer who had received radical RapidArc treatment from 2012 to 2014 at KAMC (King Abdullah Medical City). Dosimetric evaluation metrics were used to compare the two plans in terms of mean, maximum and minimum doses to PTV, Homogeneity Index (HI), Conformity Index (CI), Target Coverage Index (TCI) and mean and maximum doses to critical organs and normal tissue. Dose to 95% of the PTV (D95%) was used to quantify PTV coverage. Mean value of the PTV which was 51.38 ± 2.172 in RapidArc compared to 52.21 ± 1.963 in 3DCRT, which means that RapidArc plan achieved lower mean and maximum doses to the PTV. The maximum dose to the PTV in RapidArc was higher compared to 3DCRT with lower maximum dose to the PTV, ($p=0.011$). These results explain the statistical advantage in PTV coverage metrics in RapidArc modality. PTV dose coverage, as measured by the minimum dose and the dose to 95% of the volume was higher in the RapidArc plan. RapidArc plan also showed a more homogeneous dose distribution in PTV, achieving an HI of 1.262 ± 0.037 compared with 1.271 ± 0.024 in the 3DCRT plan however, RapidArc and 3DCRT achieved similar CI values and improvement in target coverage index (TCI) in which (TCI) in RapidArc was (0.006 ± 0.003) and (0.008 ± 0.006) in 3D-CRT, ($P=0.202$). Volumetric modulated arc therapy (VMAT) is better than 3DCRT in term of PTV, conformity and homogeneity for breast cancer.

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