Cavernous sinus thrombosis treatment pdf online application form online

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Treatment will be started as soon as possible, even before tests have confirmed if a bacterial infection is responsible.If tests later show that a bacterial infection did not cause the condition, antibiotic treatment may be stopped.Most people will require antibiotics for a few weeks to ensure the infection has been fully cleared from their body. The antibiotics will be given through an intravenous drip directly connected to one of your veins.Some people will experience side effects when taking antibiotics. These are generally mild and can include diarrhoea, nausea and a skin rash.AnticoagulantsYou may also be given a medicine called heparin to help dissolve the clot and prevent further clots. Heparin is an anticoagulant medicine, which means it makes the blood less sticky.Some people also need to take anticoagulant tablets for a few months or longer after leaving hospital.CorticosteroidsSome people are also given steroid medicine (corticosteroids). Corticosteroids can reduce inflammation and swelling in your body.Surgical drainageIf the symptoms of cavernous sinus thrombosis were caused by an infection spreading from a boil or sinusitis, it may be necessary to drain away pus from that site. This can be done either using a needle or during surgery.How long does treatment last?Several weeks of antibiotic treatment are usually necessary to ensure the infection has cleared. However, it can take a long time to recover fully, and it may be several months before you're well enough to leave hospital. Cavernous sinus thrombosis is a very serious condition. Even with prompt treatment, as many as 1 in 3 people with the condition may die.Some people who survive will develop long-term health problems due to damage to their brain, such as persistent headaches and fits, some degree of vision loss.Read more about the complications of cavernous sinus thrombosis. It's difficult to say exactly how many people are affected by cavernous sinus thrombosis, but it's thought to be very rare.The condition affects people of all ages and tends to be more common in women than men. This may be because pregnancy and taking the oral contraceptive pill can make women more vulnerable to blood clots. Page last reviewed: 05 August 2021 Next review due: 05 August 2024 personPersonal accountAccess through your institutionLog in here via OpenAthens or ShibbolethORSUBSCRIPTION OPTIONS Background: Cerebral sinus venous thrombosis (CSVT) is a relatively rare, potentially fatal neurological condition that can be frequently overlooked due to the vague nature of its clinical and radiological presentation. A literature search on PubMed using the keyword ¢ÃÂÂCerebral sinus venous thrombosis¢ÃÂÂ was performed. We searched for the epidemiology, risk factors, pathophysiology, clinical features, diagnosis, and treatment of CSVT. All full-text articles in the last 10 years, in adults (>18 years), and the English language were included. We aim to give a comprehensive review of CSVT, with a primary focus on the management of the disease. Summary: The literature search revealed 404 articles that met our criteria. CSVT is a relatively rare condition that accounts for approximately 1% of all forms of stroke. They can be subdivided into acute, subacute, and chronic forms based on the time of onset of clinical symptoms. It is a multifactorial disease, and the major forms of clinical presentation include isolated intracranial hypertension syndrome, focal neurological deficits, and cavernous sinus syndrome. MRI with magnetic resonance venogram (MRV) is considered the gold standard for diagnosis. Anticoagulation with heparin or low-molecular-weight heparin is the mainstay of treatment. 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Studies also show an increased risk of CSVT in overweight or obese OCP users and are carriers of hereditary thrombophilia [15, 16]. Local head and neck infections are the most common infectious cause of CSVT, and studies have shown that it is more common in less developed countries [17, 18]. Intracranial neoplasia may result in CSVT due to local compression or tumor invasion of the breast [19]. A retrospective study conducted in a tertiary center involving 90 patients with CSVT showed that 14% of their patients had a history of some form of head trauma before diagnosis, 7% of the affected individuals had head and neck infections and 8% had brain tumors [20] .Phofisiology The physiopathological mechanisms of CVT are not fully understood and are a complex mixture of changes in hemodynamics and cerebral parenchyma [21]. The current knowledge of CVT physiopathology is based on studies of available animal models. The clinical presentation of CVT can be explained by 2 pathological mechanisms - thrombosis of the brain veins and thrombosis of venous brain sinuses (Fig. 2). Physiopathology of the CVT [22]. LCF, cephalorraquidian liquid. Studies of the range model showed that sinus thrombosis leads to sinus occlusion and blood reflux in vernulas and capillaries, resulting in increased local pressure, eventually resulting in a massive cerebral edema [23]. Extensive anatomoses found in the venous brain system usually allow the development of collateral circulation if there is an occlusion in the venous flow. Occlusion of the cortical vein may result in increased pressure of small vessels, depending on the extent of thrombosis and the availability of guarantees. This can further lead to the interruption of the hematoencephalic barrier, leakage of blood components in the interstitial space, causing vasogenic edema and damage to the oies oies oa odived onaidiuqarrolafec odiuqÃl od meganerd an esaerced.]42[odnumiuqnerap It may result from the dysfunction of arachnoid granulation, causing an increase in intracranial pressure. A contained increase in pressure results in capillary hypertensive, cerebral edema and venous hemorrhage [25]. This also results in a decrease in the pressure of cerebral perfusion and metabnlica failure, followed by intracellular edema and cytothanxic edema [26]. Extension of thrombosis, patient age and underlying etiological factors. The most common presentation includes signs of high intracranial pressure, such as headache, reduced visual acuity and papiled, ficits ficts focal, seizures and diffuse encephalopathy. Others unusual presentations include subarachnã³ide hemorrhage, thunderclap head pain, recurrent transthers, tinnitus, isolated head pain and cranial nerve paralysis in the cave -sinus thrombosis scene [4] .. 80% .. 80% .. 80% .. 80% .. 80% of CSVT patients [27]. Patients with head pain often present in isolation or associated with other signs and symptoms, such as vain, papiledema, visual field defects, loss of vision, tinnitus and abducens nerve palsy. Loss of vision and papiledema are generally observed in patients with chronic anything [10]. Studies have shown that patients with TCV may have isolated head pain without any evidence of high intracranial hypertensive hypertenses and is often observed in patients seeking early moms [28]. The head pain due to increased intracranial pressure may occur as a result of great venous thrombosis, such as the sagittal sinus. These patients have sudden, severe, widespread and progressive head pain that worsens with Valsalva, striving and lying down [29]. Some individuals may develop a sudden, severe, Form called Thunderclap head pain that mimics subarachnoid hemorrhage [30]. With the involvement of the lateral breast, patients may have increased intracranial pressure and symptoms similar to an infection in the middle ear, such as fever, ear discharge, headache and pain over the mastoid region [31]. focal neurological deficits The most common form of deficit is the Fraqueza engine observed in the form of monoparesis, hemiparesis or bilateral involvement in patients with thrombosis of the deep venous system. Although motor involvement is common, these patients rarely have visual or sensory deficits [32]. The thrombosis of the cavernous sinus is usually associated with infection and affects extra eye movements due to the involvement of the oculomotor, abducer and/or trochlear nerves. They present headache, eye pain, constitutional symptoms, proptosis, quimosis, periorbital edema and paralysis of eye movements [33]. Assessments are a common presentation in patients with CSVT, especially in the acute phase of the disease [33]. It is more common in patients with hemorrhagic supratentorial lesions, upper sagittal sinus or cortical vein thrombosis and in patients with focal neurological deficits [10]. A prospective study of 194 patients with acute CT found early symptomatic crises in 44.3% of patients and epilepticus status in 12.8% of patients. They also found a high incidence of Todd's parsy associated with seizures [34]. The encephalopathy of diffuse is a more serious presentation of the CSV and individuals with deep affection of the venous system can develop encephalopathy that results in a change of consciousness, changes in mental status, delirium, apathy, or symptoms of deexecutors. These patients may also develop bilateral or multifocal deficits or present seizures. Encephalopathy is a common occurrence among the elderly [12]. The discvt summed upof clinical manifestations - progressively worsening headache (most common ocits³Ãngaid ed megami A adazirodatupmoc aifargomoT ed o£Ã§ÃazilatigiD .]11[ )ASA(noitaicossA ekortS naciremA e)AHA(noitaicossA traeH naciremA alep siairetra soir³Ãtirret soir¡Ãv me otrafni moc uo odnumi megiro ad lacitroc aigarromeh moc setneicap me adadnemocer ©Ã larberec osonev ametsis od megami A .TVC ed o£Ã§Ãazilacol e ocits³Ãngaid o arap oir¡Ãssecen ,esil¡Ãna amitlºÃ me ,©Ã e oruo o£Ãrdap o ©Ã gnigamiorueNgnigamiorueN .]63[sanainarcartni seµÃ§Ãcefni omoc sasuac sartuo riulcxe arap adazilaer res edop)PL(rabmol o£Ã§Ãnup amU .]63[TVC ad sodaicossa ocig³Ãloite ocsir ed serotaf racifitnedi a maduja setset setsE .o£Ã§Ãalugaoc ed lifrep mu e etnegnarba ocil³Ãbatem leniap ,atelpmoc eugnas ed megatnoc odniulcni ,sotief setset ed leniap mu ret meved oir³Ãtarobal od TVSCstseT moc setneicap sO .sodatefa soudÃvidni ed o£Ã§Ãceted a arap laicnesse ©Ã odairporpa ocinÃlc nemuca e atiepsus ed ecidnÃ otla mU .TVSC ed ocits³Ãngaid o arap licÃfid etnemralucitrap anrot sacinÃlc seµÃ§Ãatneserpa ed edadeirav ednarg A ocits³ÃngaiD .]33[sadazilareneg seµÃsluvnoc ed uo omal¡Ãt od ,larberec etsah ad otnemivlovne oa odived rerroco edop amoc O .siaretalinu saigarromeh uo sotrafni sednarg a odived larberec etsah ad o£Ãsserpmoc uo o£Ãlafecneid ed ratluser edop larberec o£Ã§Ãainreh A .]53[omsitum e oirÃled ,ais©Ãnma omoc siatnematropmoc seµÃ§Ãatsefinam matneserpa euq ,etnemlartnec sadazilacol siaretalib sacimal¡Ãt seµÃsel me atluser osonev ametsis od adnuforp esobmort A .]01[siacof socig³Ãloruen sticif©Ãd e seµÃsluvnoc ,a§Ãebac ed rod atneserpa adalosi lacitroc aiev ad esobmort A .]22[sadazilareneg uo siacof seµÃsluvnoc e siaretalib uo siaretalinu serotom sticif©Ãd me ratluser edop latigas oies od otnemivlovne e aisafa moc ratneserpa edop odreuqse osrevsnart oies od otnemivlovne O .]6[ )%2,31(lausiv opmac ed sotiefed e ,)%5,31(aipolpid ,)%9,31(amoc uo ropertse ,)%1,91(aisafa ,)%22(odaretla latnem odatse ,)%3,82(amedelipap ,)%2,73(aiserap ,)%3,93(seµÃsluvnoc ,)%8,88 â Recommended is a computed tomography head (CT) not contrasted for all patients with acute acute presence complaints such as headache, focal neurological deficits, altered mental status, and seizures [10]. In about 30% of cases, CT may be normal or show nonspecific abnormalities; thus, a normal scan does not rule out CSVT [37]. Non-contrast CT shows direct signs like dense clot sign (visualization of thrombus in the cerebral veins and sinuses) and string or cord sign (string or cord-like density indicating CSVT); as well as indirect signs like cerebral edema, mass effect, and intracranial hemorrhage that can range from small juxtacortical lesions to subarachnoid hemorrhage and large hemorrhagic infarcts. Contrast-enhanced CT shows direct signs like empty delta sign (a low-attenuated center surrounded by a triangular area of contrast enhancement) and indirect signs, similar to non-contrast CT [38, 39] (Fig.Ã Ã3). Presentations of CVT on CT head. The figure was reproduced with permission from the Radiology Assistant website (. aDense clot sign (blue arrows, dense transverse sinus due to thrombosis). Hemorrhagic infarction of the temporal lobe (red arrow). bEmpty delta sign (right and left transverse sinus; and sigmoid sinus thrombosis).CT VenographyVenous enhancement following a CT scan help evaluate filling defects in the cortical sinuses and veins, increased collateral venous drainage, as well as enhancement of sinus walls in these patients [40]. CT venography (CTV) has an overall sensitivity of about 95% and may be useful for patients with a subacute or chronic presentation [5]. Studies support the use of CTV for the diagnosis of cerebral sinus thrombosis and show it has less diagnostic value for cortical vein thrombosis [41]. CTV is a rapid and reliable option, especially in those patients with contraindication to perform an MRI.MRI and Magnetic Resonance VenogramMRI with contrast and magnetic resonance venogram (MRV) is the most diagnostic modality for CSVT, and the appearance of thrombus on different MRI sequences are time dependent. Within 5 days, it appears isointense on T1 and hypointense on T2-weighted images. In 6¢ÃÂÂ15 days, thrombus appears hyperintense on both T1 and T2 sequences. After 15 days, they appear isointense on T1 and iso- or hyperintense on T2 sequences. Four months later, no abnormalities on T1 and subtle abnormalities on T2 sequences can be detected [42-44] (Fig.Ã Ã4). The MRI appearances in the acute form occur due to an increased amount of deoxyhemoglobin in the thrombus, and the changes seen with the subacute form is due to an increased concentration of methemoglobin [8]. The absence of changes in the T1 signal sequence and normal flow pattern on the T2 sequence makes diagnosis particularly difficult in the acute form of the disease [45]. Gradient-echo T2 and susceptibility-weighted imaging sequences help identify a thrombus as a hypointense area and can be useful in the detection of acute CSVT. Susceptibility-weighted sequences are also beneficial for the diagnosis of an isolated cortical vein involvement [46]. MRV is a noninvasive diagnostic modality used for the diagnosis of CSVT suggested by the absence of normal flow in a vein or sinus. A contrast-enhanced MRV is considered more sensitive to time-of-flight MRV for the detection of thrombus in smaller veins [47]. Time-dependent appearance of thrombus on different sequences of MRI brain. The figure was reproduced with permission from the Radiology Assistant website (. aT1-weighted images: red arrow, hyperintense signal due to thrombosis. b T2-weighted image: blue arrow, normal flow in right sigmoid sinus and jugular vein; red arrow, hyperintense signal due to thrombosis.Digital Subtraction AngiographyCerebral angiography was considered the primary diagnostic procedure used for the ed a§Ãneserp an omsem ,aduga CCT moc setneicap so sodot a anirapeh ed acitu ªÃparet megasod :]25[TVSC an o£Ã§Ãalugaocitna aiparet arap eviapnes o£Ã§Ãcefni a euq rative arap evlovnesed es oenÃugnas olug¡Ãoc O .sosonrevac soies so arap ahlapse es oin¢Ãrc uo otsor on o£Ã§Ãcefni amu odnauq revlovnesed es edop eugnas ed elortnoc ed soiasne ¡Ãh o£Ãn siop ,osrevortnoc ocip³Ãt mu ©Ã adnia ,soterid siaro setnalugaocitna ed osu o omoc meb ,o£Ã§Ãalugaocitna ad o£Ã§Ãarud A .]15[ )WML(ralucelom osep oxiab ed anirapeh euo adanoicarf o£Ãn anirapeh moc sodatart CCT moc setneicap me sniur sodatluser ed saxat san o£Ã§Ãuder amu uortsom sodutse sesse moc adazilaer esil¡Ãnatem amU .]05 ,94[TVSC moc setneicap me setnalugaocitna ed osu oa lev¡Ãrovaf etnemacinilc aicnªÃdnet amu marartsom obecalp o moc o£Ã§Ãalugaocitna ad aic¡Ãcife a e a§Ãnaruges a odnarapmoc alacse aneuqep me sodutsE .siotoc sod o£Ãsnapxe a ridepmi e solug¡Ãoc ed o£Ã§Ãuloser a revomorp arap TVSC moc setneicap me otnematart ed acitu ªÃparet aiparet]84 ,24 ,04 ,8[CCT ad ocits³Ãngaid on sadazilitu megami ed sedadiladom sad sartnoc e s³Ãrp so odnarapmoC .2 alebaT an adimuser ¡Ãtse aifargoigna A latigid o£Ã§Ãartbus amu e VRM/MR ,VTC/TC odniulcni ,megami ed sedadiladom sair¡Ãv ed sacits³Ãngaid sedadiladom ed o£Ã§Ãarapmoc ad snegatnavsed e snegatnaV .]01[soivan" wercskroCÃ ¬â ¢Ã sodatalid e sosoutrot siaretaloc moc siacitroc saiev ed atpurba adarap amu ¡ÃH .adatalid laretaloc o£Ã§Ãalucric e odasarta otnemaizavse ,saiev uo soies son otelpmoc uo laicrap otnemihcne ed otiefed mu meulcni siausu satrebocsed sA .siasrevsnart soies sod aisalpa uo aisalpopih e roirepus latigas oies od roiretna etrap ad aisalpopih ,roirepus latigas lasunis o£Ã§Ãacilpud omoc ,sacim´Ãtana seµÃ§Ãairav ed a§Ãneserp an raterpretni arap arodaifased res edop aifargoigna A .sievÃnopsidni uo savisulcnocni o£Ãs megami ed sedadiladom sartuo odnauq odazilaer Ã .MR e VTC alep odÃutitsbus meb otium lof sam ,sanainarcartni sasonev sedadilamrona ed HWML HWML .larberecartni aigarromeh ed osu o e TVC moc setneicap so sodot ed %05 a atefa euq o odnatefa ,setneuqerf o£Ãs acsub ed selortnoc ed selortnoC .a§Ãebac ed rod e o£Ãsiv ed adrep me ratluser edop euq o ,anainarcartni o£Ãsserp ad otnemua o e seµÃslvnoc ed ojenam o etnemlapicnirp evlovne oci¡Ãmotnis oci¡Ãmopmisseparet otnematart O .]55[anainarcartni aigarromeh Ã adÃubirta ©Ã o£Ã§Ãaroireted ajuc setneicap me odacidni ©Ã o£Ãn ,otnatne oN .etnalugaocitna aiparet Ã mednopser o£Ãn uo lataf odatluser mu mªÃt euq setneicap me osrucer omitlºÃ omoc adasu ajes ralucsavodne esil³Ãbmort a euq adnemocer aigolotameH me samroN ed ocin¢ÃtirB ªÃtimoC O .TVSC moc setneicap me socitÃlobmort ed osu od o£Ãsneerpmoc rohlem amu recenrof edop odutse etse ,omix³Ãrp orutuf mu mE .]45[miur odatluser moc sevarg TVC moc setneicap me odatluser o ¡Ãrarohlem esil³Ãbmort a es ranimreted arap otnemadna me ocirtnªÃcitlum e odazimodnar elortnoc ed odutse mu ©Ã larberec asonev esobmort ed odutse o arap o£Ã§Ãalugaocitna uo esil³Ãbmort A .]35[ele a sadaicossa sadargnas seµÃ§Ãacilpmoc siapicnirp san ovitacifingis otnemua mu met esil³Ãbmort a euq uortsom sodutse 51 ed acit¡Ãmetsis o£Ãsiver amU .sadÃulco siarberec saiev uo oies o erbaer e olug¡Ãoc od o£Ã§Ãulossid a aduja esil³Ãbmort A .]5[soda§Ãnava sortnec me adazilaer ralucsavodne esil³Ãbmort ed lanoicida otnematart ed o£Ã§Ãpo amu ret medop setneicap sesse euq eregus ASA/AHA .otnematart od rasepa ,etnemacinilc rarioreted es uo rarohlem o£Ãn medop setneicap snugla ,otnatne oN ;etnalugaocitna aiparet moc lev¡Ãrovaf ocinÃlc odatluser mu mªÃt tvscsisilobmoRT ed setneicap sO .]5[3 ¬â 2 ed lanoicanretni sniarm oitar moc areves ailifobmort a odaicossa TVSC uo TVSC 2 s³Ãpa VET ,etnerrocer TVSC me adiv ad ognol oa etnemlaicnetop e adacovorp o£Ãn TVSC me sesem 21 a 6 ,adacovorp TVSC an sesem 6 - 3 rop o£Ã§Ãalugaocitna a madnemocer ASA/AHA ad sezirterid sA .odip¡Ãr etnalugaocitna otiefe ed o£Ãsrever amu air¡Ãssecen ©Ã es uo HPML Ã socigr©Ãla setneicap me otecxe ,adanoicarf o£Ãn anirapeh A of antiepileptic drugs, although favored by some studies, remain controversial [56]. A study identified intracranial hemorrhage, cortical vein thrombosis, and focal neurological deficits as independent predictors of early seizures and suggested that prophylactic antiepileptic therapy may benefit these patients [34]. Another study found focal sensory deficits and admission imaging showing focal edema or infarcts (ischemic/hemorrhagic) as significant predictors of seizures in CVT patients [57]. Several studies have also demonstrated the presence of supratentorial lesion to be a predictor for seizures [58, 59]. Though the duration of treatment is uncertain, it is recommended to give antiepileptic therapy to acute CSVT patients with seizures and supratentorial lesions to prevent recurrence [52].Treatment of Raised Intracranial PressureCerebral edema is common in CSVT patients. However, mild swelling improves with anticoagulant therapy, which dissolves thrombus, improving flow, and thereby reducing intracranial pressure. Patients who develop a headache can be given analgesics for symptomatic relief. Transtentorial herniation of the brain is the most common cause of death in CSVT patients [60]. Carbonic anhydrase inhibitors like acetazolamide help in reducing the intracranial pressure (ICP) and alleviating headache as well as preventing visual damage [61]. The European Federation of Neurological Societies recommends the following [62]: LP can be considered before initiation of anticoagulation, in the absence of parenchymal abnormalities (large infarcts or hemorrhages) in patients with intracranial hypertension. Acetazolamide may be considered in patients with persistent papilledema. In patients with continuing visual deterioration despite repeated LP and Acetazolamide administration, shunting procedures like lumboperitoneal, ventriculoperitoneal shunts, and optic nerve fenestration can be considered. Steroid therapy is ineffective due to lack of scientific evidence. Anti-edema treatment should be carried dnuof osla yduts ehT .pu-wollof fo dne eht ta %4.9 dna esahp etuca eht ni %6.5 fo etar ytilatrom a dnuof TVSC fo esruoc larutan eht deiduts taht weiver citmetsys A.]06[snoisel assof roiretsop dna ,egahrromeh erehpsimeh thgir ,metsys suonev peed eht fo sisobmorht ,sutats latnem deretla ,ssensuoicsnoc deserped erew syad 03 ta ytilatrom fo srotcider gniylrednu dna ,sucitpelipe sutats ,PCI desiar morf gnitluser noitainreh lairotnetsnart erew ytilatrom etuca fo srotciderp ehTsemoctuO ? 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Large randomized control tests are required to provide stronger evidence for CVT therapeutic intervention. These are necessary additional studies that compare different therapies to help improve treatment for TCV patients because of increased mortality rates. Acknowledgments The authors would like to thank the Deanship of Scientific Research at Majmaah University for supporting this work under the number of project R-1441-96. Declaration of the authors not only conflicts of interest to declare. DECLARATION OF CONFLICT OF INTEREST The authors declare that they are not in conflict of interest. Foundation FontesDenship of Scientific Research at Majmaah University, under the number of Project R-1441-96.AUTOR CONTRIBUTIONS All authors were involved in the preparation of this manuscript. All the authors read and approved the manuscript. The corresponding author has full access to the data and has the right to publish this data. 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(iscvt.). J .aisÃnuT ad etrooc amu me ozarp ognol a odatluser e ocsir ed serotaf ,sacinÃlc sacitsÃretcarac :larberec asonev esobmorT .la te ,J ilauoaZ ,N assiM-hikeF ,H ilabreD ,M inamlesseM ,M ruosnaM ,Y mohdiS .29 - 985 :)1317(613 ;8991 .JMB .osonev oies od larberec adiges esobmort ad odutse ed opurg O .sair¡Ãtidereh sacit³Ãbmortorp seµÃ§Ãidnoc ed serodatrop]o£Ãs meuq ed o£Ã§Ãerroc[an e siaro sovitpecartnoc soir¡Ãusu me larberec lasunis esobmort ed ocsir od elortnoc-osac ed odutsE .PJ ekcuorbednaV ,MM nampooK ,J matS ,FS njiurB eD .48 - 975 :)5(37 ;6102 .lorueN amaJ .sasebo serehlum me larberec asonev esobmort ed ocsiR .la te ,RM rendleH ,MS sivliS ,A yavroM-georB ,S prodleddiM ,M dlonrA ,MS reibruuZ .37 - 6672 :)7(701 ;6002 .eugnaS .esil¡Ãna-atem amu :larberec aiev ad esobmort ed ocsir e siaro sovitpecartnoc ,sacilÃfobmort sedadilamronA .W onegA ,M rehtworC ,F ilatneD .32 - 714 :)6(543 ;1002 .deM J lgnE N .sa§Ãnairc me larberec eunevonis esobmorT .la te ,JD yelkcuB ,F htooB ,B nosnrojB ,C smadA ,M werdnA ,G rebeveD .23 - 7291 :)9(63 ;5002 .emarreD .sosodi setneicap me larud oies od esobmort e larberec aieV .I serodagitsevnI ,F airenigirraganiraB ,J matS ,GM resuoB ,P O £ÃhnaC ,MJ orreF .16 - 6532 :)7(04 ;9002 .emarreD .serehlum me larberec lasunis e asonev esobmorT .la te ,GM