·综述·

自发性低颅压影像学及治疗研究进展

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【摘要】 自发性低颅压以体位性头痛为主要临床表现，是中枢神经系统少数明确诊断后可治愈的疾病之一，但仍有部分患者头痛发作无典型体位性特点，给临床诊断带来较大困难，也常因误诊、漏诊而延误最佳治疗时机。近年来，影像学技术的日益进步，有助于临床早期诊断，并发展出多种新型治疗方法。本文拟对近年来自发性低颅压的影像学检查和治疗方法进行综述。

【关键词】 颅内低压； 体层摄影术，X线计算机； 磁共振成像； 脊髓造影术； 脑血管造影术； 综述

Research progress of imaging and therapy in spontaneous intracranial hypotension

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【**Abstract**】 Characterized by "postural headche", spontaneous intracranial hypotension (SIH) is one of the few diseases of central nervous system (CNS) that can be completely cured with definitive diagnosis. However, if the headache attacks are not typically postural, it brings doctors much difficulties to diagnose and make more misdiagnosis or diagnostic errors. In recent years, rapidly developed imaging technologies contribute to early diagnosis in clinic and a lot of new treatment methods were emerged. Here we summarize the literatures about imaging and treatment methods in the past few years to help doctors increase the understanding of SIH.

【**Key words**】 Intracranial hypotension; Tomography, X - ray computed; Magnetic resonance imaging; Myelography; Cerebral angiography; Review

低颅压综合征（IHS）系腰椎穿刺脑脊液压力＜60或70 mm H_2O（1 mm H_2O = 9.81 × 10^{-3} kPa，80～180 mm H_2O）时产生的一组综合征，包括体位性头痛，站立时出现或加重、卧位时减轻或消失；恶心、呕吐；视觉障碍或听觉障碍等。病因较为复杂，可继发于腰椎穿刺、脊髓血管造影、神经外科手术等有创性操作，亦可见于颅脑创伤（TBI）、恶病质、尿毒症、全身感染等内科重症疾病。Schaltenbrand于1938年首次报告1例无明显诱因导致头痛的低颅压患者，并定义为“低颅压综合征”，其中，病因不明者为自发性低颅压（SIH）[1]。本文拟重点阐述自发性低颅压的影像学检查和治疗方法。

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一、影像学检查

影像学检查仅能反映出自发性低颅压的某些间接临床征象，而不能作为明确诊断的必要条件[2]。

1. 头部CT检查 多数自发性低颅压患者头部CT正常，少数可见侧脑室、第三和第四脑室、基底池狭窄，可能是脑水肿所致，颅内压恢复正常后消失。有个案报道，自发性低颅压患者头部CT可见硬膜下血肿和硬膜下积液等（图1）[3]，增强扫描无特异性征象。目前尚无公认的临床准确定位脑脊液漏的方法。2015年，Seo等[4]首次报告1例经三维CT（3D-CT）增强扫描成功准确定位脑脊液漏的病例，表明3D-CT增强扫描定位脑脊液漏的准确率高于核素脑池或脊髓池造影术。

2. 头部MRI检查 （1）脑膜强化征象：Mokri[5]于2004年首次提出自发性低颅压患者存在脑膜强化征象至今，脑膜强化征象（图2[6]）业已成为自发

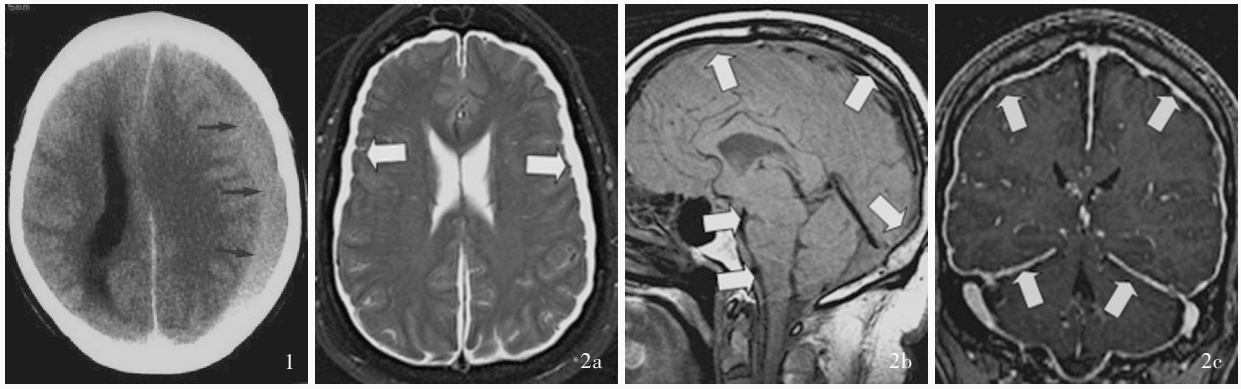

图1 横断面CT显示，左侧额顶叶硬膜下低或等密度影，考虑硬膜下血肿（箭头所示），并可见中线移位[3] **图2** 头部MRI检查所见[6] 2a 横断面T_2WI显示，双侧硬膜下积液（箭头所示） 2b 矢状位T_1WI可见中脑和视交叉下移等低颅压征象（箭头所示） 2c 冠状位T_1WI显示，硬脑膜弥漫性边缘规则的强化（箭头所示）

Figure 1 Axial CT showed left fronto-parietal subdural iso-equidensity shadow (arrows indicate), suggesting subdural hematoma, and midline shift could be seen[3]. **Figure 2** Head MRI findings[6] Axial T_2WI showed bilateral subdural effusion (arrows indicate, Panel 2a). Sagittal T_1WI showed shifting down of midbrain and optic chiasma, suggesting intracranial hypotension (arrows indicate, Panel 2b). Coronal T_1WI showed diffuse and regular enhancement of dura mater (arrows indicate, Panel 2c).

性低颅压的最典型MRI表现[7-10]，即大脑皮质凸面和小脑幕表面硬脑膜呈弥漫性、连续性强化征象，而脑干和侧裂较少强化，且随颅内压的恢复而消失。绝大多数自发性低颅压患者MRI可见脑膜强化征象，但仍有20%～30%的患者无该征象，而且该征象并非仅见于自发性低颅压患者[11-12]，因此，脑膜强化征象不能作为自发性低颅压的诊断或排除依据。脑膜强化征象的形成机制业已达成共识，即Monro-Kellie假说，脑和脊髓体积、脑脊液体积、脑血容量（CBV）三者呈动态平衡，当脑脊液减少时，脑血容量代偿性增加，脑血流量（CBF）增快，即静脉扩张，血管通透性增强，从而导致更多的对比剂在硬脑膜血管或间质周围沉积，而软脑膜存在血-脑屏障（BBB），故不如硬脑膜和静脉窦扩张明显。此外，亦有学者报告，软脑膜强化伴大脑镰前动脉扩张患者，排除动脉瘤和自发性低颅压后，经一般支持治疗和自身硬膜外静脉血补片（EBP）治疗，可以获得较好效果[13-14]。（2）脑组织移位：随着脑脊液的减少，其缓冲作用降低，脑组织下移接近颅底，从而导致部分脑组织移位，颅后窝拥挤[7-8,12]，随着脑脊液逐渐恢复，该现象消失。其中较为常见的是中脑导水管开口处下移、脑干腹侧接近斜坡甚至小脑扁桃体下疝[9]（图3[15]）。有学者提出“下垂脑”概念[4,9,16]，即鞍上池消失、桥前池狭窄、视交叉受压下移、垂体受压，均提示脑干下移，称为下垂脑。值得注意的是，与传统认为的脑脊液漏导致脑组织移位这一观点不同，Ohwaki等[2]对250例疑似自发性低颅压患者进行核素脑池造影术，发现脑脊液漏患者占74.40%（186/250），而脑组织移位患者仅13.21%（21/159），因此，对脑脊液漏与脑组织移位的相关性提出质疑。（3）硬膜下血肿和硬膜下积液：硬脑膜边缘细胞缺乏胶原纤维，结构不紧密，导致血管代偿性扩张时液体外渗形成积液，其发生率约为50%，好发于额顶部（图4[17]），无占位效应，严重时约25%患者发生血管破裂，从而导致硬膜下血肿[4,9,11]，甚至中线移位或占位效应[11,18]，临床症状急剧恶化出现意识障碍，需外科手术紧急干预[3]。研究显示，自发性低颅压患者发生硬膜下血肿和硬膜下积液的比例为10%～69%[3]，且易出现严重头痛、神经功能缺损症状恶化、占位效应甚至意识障碍等。2015年，Xia等[19]回顾分析25例出现硬膜下血肿的自发性低颅压患者的临床资料，发现存在硬膜下血肿患者较不存在硬膜下血肿患者病程更长[（43.10±29.70）天对（26.90±24.90）天；$P=0.043$]，存在硬脑膜强化征象或静脉扩张征象常意味硬膜下血肿的可能性较大。值得注意的是，Chotai等[20]报告1例以双侧硬膜外血肿为主要临床表现的自发性低颅压患者，行血肿穿刺置管引流术后病情恶化并脑疝形成，提示此类患者行外科手术时应慎重。除外科手术干预外，一般支持治疗和自身硬膜外静脉血补片治疗（首次治愈率达72%）也可取得较满意的效果[21-22]。（4）其他：包括脑组织肿胀[11]、垂体充血[4,9,11,23]、脑室狭窄[24]、脑桥等脑干结构受压[25]、静脉窦扩大[8]（图5[26]），以及脊髓蛛网膜外隙积液、脑脊液漏、软

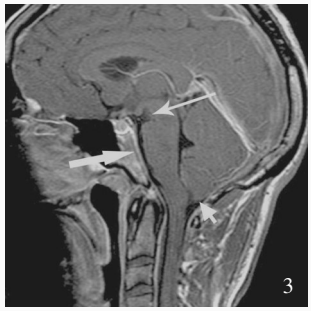
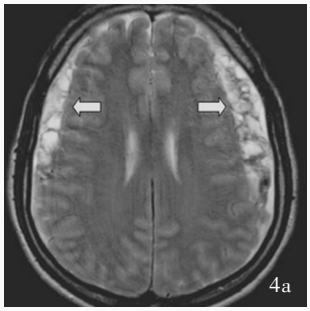
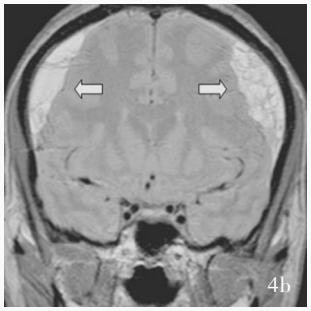
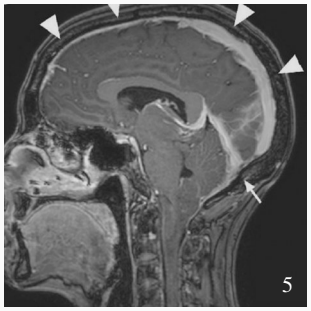

图3 矢状位增强T_1WI显示，小脑扁桃体下疝（短箭头所示），桥前池扁平（粗箭头所示），脑干腹侧接近斜坡（细箭头所示）[15] **图4** 头部MRI检查显示，双侧硬膜下血肿（箭头所示）[17] 4a 横断面T_2WI 4b 冠状位T_2WI **图5** 矢状位增强T_1WI显示，上矢状窦（粗箭头所示）和横窦（细箭头所示）扩张[26]

Figure 3 Sagittal enhanced T_1WI showed caudal displacement of the cerebellar tonsils (short arrow indicates), slight flattening of the anterior pons (thick arrow indicates) and the ventral brain stem was close to clivus (thin arrow indicates)[15]. **Figure 4** Head MRI showed bilateral subdural hematoma (arrows indicate)[17]. Axial T_2WI (Panel 4a). Coronal T_2WI (Panel 4b). **Figure 5** Sagittal enhanced T_1WI showed expanding of superior sagittal sinus (thick arrows indicate) and transverse sinus (thin arrow indicates)[26].

组织积液、脊膜憩室[27]、硬脊膜强化征象、静脉丛扩张等。近年研究显示，T_1WI显示横窦扩张对自发性低颅压的诊断具有较好的提示意义（灵敏度93%、特异度94%）[10]。与多数研究不同，2015年Higgins等[28]和2014年Ajlan等[29]报告3例自发性低颅压致颅内静脉窦血栓形成（CVST）患者，提示自发性低颅压患者体位性头痛改变应警惕颅内静脉窦血栓形成的可能，前者可能是后者的危险因素。2015年，Higgins等[28]报告1例自发性低颅压致颈静脉狭窄和脑组织移位患者，打破自发性低颅压仅引起静脉扩张的传统认知。2014年，Ajlan等[29]报告1例深静脉损伤致意识障碍的自发性低颅压患者。值得注意的是，尽管MRI是筛查自发性低颅压的有效方法，但有文献报道，有1/4的自发性低颅压患者MRI正常，故不应因MRI正常而排除自发性低颅压的可能[30]。此外，近年来脊柱MRI开始应用于临床，某些征象如硬膜外隙和硬膜下静脉扩张、硬脑膜强化和脊膜憩室、蛛网膜外隙或硬膜外积液等均对脑脊液漏的诊断具有提示意义[10-11]。

3. 脑池和脊髓池造影术　脑池和脊髓池造影术可以获得不同时间点对比剂的分布情况，从而动态观察脑脊液循环[7]。有研究显示，自发性低颅压患者出现对比剂外渗和泌尿系统过早显影现象（图6）[4]，分别证明存在脑脊液漏和脑脊液吸收过快，从而证实脑脊液生成减少、吸收增加和脑脊液漏的发病机制。通常对比剂在脑池或脊髓池积聚的时间为24～48小时[10]，脑池或脊髓池积聚时间过短或过早在其他区域显影均提示脑脊液漏[12]。Spelle等[31]报告1例行脑池造影术的自发性低颅压患者注射对比剂6小时后即可见蛛网膜外隙显影，24小时后脑沟和脑回便不再显影。此外，接受检查时患者所处体位对阳性检出率亦有影响：坐位或站立位时由于重力作用导致对比剂较卧位时上行速度减慢，病灶显影时间延迟，阳性检出率降低[32]。此外，个体异质性导致膀胱充盈时间和脑脊液腔衰减时间不同，行腰椎穿刺等有创性操作时，检查者操作方式和对比剂性质均可能影响检测的敏感性，故诊断价值有限[4,32]。此外，注射对比剂至完成CT扫描的时间越长、脑脊液漏定位准确率越低，而动态CT扫描因其扫描迅速，可以较好地克服这一缺点[10]。研究显示，CT、MRI和核素脑池造影术检测脑脊液漏的灵敏度分别为67%、50%和55%[4]。但是由于某些有创性操作可能加速脑脊液漏，进而加重病情，故临床无需人为加入对比剂的MRI脑池造影术应用较多，其他造影术应用有限，但是MRI脑池造影术检测脑脊液漏的阳性检出率较低，尚待更合理的影像学方法[33]。2012年，Carstensen等[15]采用脊髓池造影术成功准确定位1例自发性低颅压患者的脑脊液漏，并在后续治疗中获得较好临床结局。2016年，Choi等[34]报告18例行多探头CT脊髓池造影术的自发性低颅压患者，脑脊液漏阳性检出率为17/18，其中10例（10/18）脑脊液漏定位准确，并认为“假憩室征（pseudodiverticular sign）”和“灰轮征（gray-rim sign）”可以作为自发性低颅压的影像学特征。

4. 脑血管造影术　研究显示，自发性低颅压患者存在大脑镰前动脉和眶上静脉等动脉和静脉（窦）扩张[4,13,35]，证实血管代偿机制并进一步说明硬脑膜强化征象与脑血容量增加有关。静脉铰链角

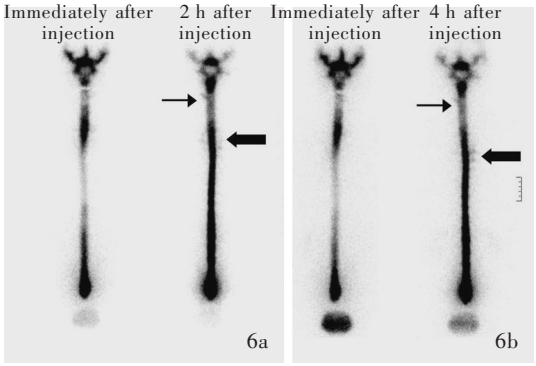

图6 脑池和脊髓池造影术所见[4] 6a 注射对比剂2 h后可见对比剂渗出，提示脊膜漏（细箭头所示为颈髓，粗箭头所示为胸髓） 6b 注射对比剂4 h后可见对比剂渗出，尤以胸髓明显（细箭头所示为颈髓，粗箭头所示为胸髓）

Figure 6 Radionuclide cisternography findings [4] Two hours after injecting contrast agent, contrast exudation could be seen, suggesting dural leakage at upper cervical vertebral level (thin arrow indicates) and middle thoracic vertebral level (thick arrow indicates, Panel 6a). Four hours after injecting contrast agent, contrast exudation could be seen, especially in the thoracic cord (thin arrow indicates cervical cord, thick arrow indicates thoracic cord; Panel 6b).

（VHA）是近年提出的自发性低颅压特征性影像学表现，表现为自发性低颅压患者颅内静脉与Galen静脉之间夹角＜79°，具有诊断提示作用，其灵敏度为88%、特异度为92%（图7）[15]。

5. 其他 2016年，Fichtner等[36]对自发性低颅压患者行经眶超声检测，结果显示，伴体位性头痛患者较不伴体位性头痛患者站立位时视神经鞘直径更小[（4.84±0.99）mm对（5.53±0.99）mm，P=0.044]，且体位变化对伴体位性头痛患者的影响更大，从而为临床提供一种快速、廉价且无创的诊断技术。

二、治疗方法

1. 一般支持治疗 卧床休息（头低脚高位）、大量补液、腹部束带和对症处理是目前普遍认可的支持治疗方法[7-10]。部分脑脊液漏可以自发停止，故此类患者无需治疗也可以康复。

2. 等渗生理盐水静脉滴注 其原理与大量补液相同[7-9]，每日补液量保持在1000～2000 ml。

3. 激素静脉滴注 有学者推荐，予糖皮质激素如地塞米松静脉滴注治疗自发性低颅压，效果较好，但其作用机制尚待进一步研究，可能与减轻炎症反应、消除脑组织水肿和硬膜下积液、促进脑脊液再吸收等有关，故未能作为首选治疗方法[9,24,37]。

4. 硬膜外注射生理盐水 将10～20 ml生理盐水直接注射至硬膜外隙，但该方法为有创性操作，易导致神经根损伤甚至感染。

5. 自身硬膜外静脉血补片 该方法于1960年首次应用于临床并沿用至今，被认为是治疗各种病因导致低颅压综合征的最有效方法，治疗有效率较高[7-9]，首次治疗有效率＞30%[12,38]，总体有效率＞70%[39]。甚至有学者认为，自发性低颅压典型表现出现后应即刻采用该方法而无需等待其他诊断结果[12]，但也有25%患者经该方法治疗后未见症状改善[30]。2016年，Zheng等[40]采用MRI增强脊髓池造影术证实该方法有效，治疗前可见明显脑脊液漏，但治疗后消失。自身硬膜外静脉血补片治疗数分钟后患者临床症状即可缓解，具体方法是抽吸数毫升至数十毫升自体血注射至硬脊膜下隙，当血液扩散至8～10个椎体节段时，增加椎管或脑脊液压力的同时脑脊液漏部位出现纤维沉积，填补硬脑膜缺损[4,41]。2013年，Huang等[33]对比1例自发性低颅压患者行自身硬膜外静脉血补片治疗前后的MRI表现，发现治疗后15天硬脑膜静脉窦扩张恢复正常、硬脑膜强化征象减弱，治疗30天内下垂脑现象消失。2016年，Karm等[42]观察104例行自身硬膜外静脉血补片治疗的自发性低颅压患者，认为脑池造影术中核素较早在膀胱显影的患者可能需要不止一次治疗，仅存在下垂脑现象的患者常首次治疗即获得较好效果。自身硬膜外静脉血补片治疗对腰椎穿刺致低颅压综合征的效果优于自发性低颅压，首次治愈率达90%，第2次即可以使几乎所有的腰椎穿刺致低颅压综合征患者好转[9]。可能是由于自发性低颅压患者脑脊液漏口较分散且数量较多，而腰椎穿刺后脑脊液漏口数量少且多集中于硬膜外注射穿刺点附近。Murphy等[43]对2例多发性脑脊液漏患者进行单次多部位自身硬膜外静脉血补片治疗，效果较好。自身硬膜外静脉血补片治疗后可能再次出现低颅压，但是多为指向性，对乙酰唑胺反应良好，其他并发症还包括腰背部疼痛、颅内积气等，但是总体较一般支持治疗起效迅速且并发症较少[44]。近年陆续有学者报告，于显微内镜或CT介导下行自身硬膜外静脉血补片治疗，首次治疗有效率即＞70%[38,45-46]，改进传统自身硬膜外静脉血补片治疗，值得神经科医师借鉴。

6. 脑血管扩张药 嘱患者吸95%氧气和5%二氧化碳，每小时吸5～10分钟，或尼莫地平10 ml/d静

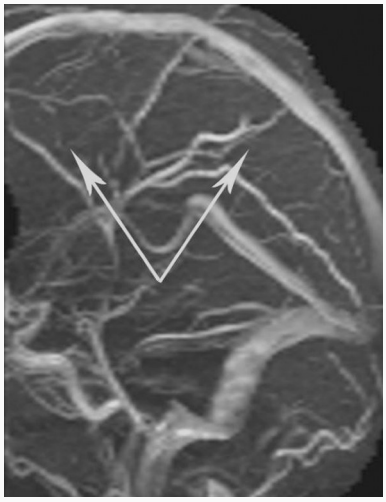

图7 MRV显示，颅内静脉与Galen静脉之间夹角为70°（箭头所示），对自发性低颅压的诊断起提示作用[15]

Figure 7 MRV demonstrated the included angle between intracranial vein and Galen vein was 70° (arrows indicate), making a suggestion on the diagnosis of SIH[15].

脉滴注，以达到扩张脑血管、降低脑血管阻力、增加脑脊液分泌的目的。

7.其他药物 咖啡因、麻黄素、罂粟碱、新斯的明、毛果芸香碱、乌洛托品、促肾上腺皮质激素、镇痛药、维生素A等[10,37]均可以用于自发性低颅压的治疗。目前，咖啡因的治疗效果业已得到公认，其他药物亦有治疗成功的文献报道，但仍存争议[7-8]。

8.外科手术 若脑脊液漏口长期无法愈合或关闭，症状持续未缓解甚至加重，可予外科手术，包括硬脊膜外右旋糖酐注射术、经皮纤维蛋白胶注射术等[10,46]，但是由于难以准确定位脑脊液漏或脊膜缺损部位，给手术带来一定困难。

三、并发症

自发性低颅压可以合并颅内静脉窦血栓形成、昏迷和硬膜下血肿等[10,44]。硬膜下血肿或硬膜下积液较为轻微，无明显症状，严重者可能产生占位效应和中线移位，压迫脑组织，需外科手术干预。反射性高颅压可能发生于成功关闭脑脊液漏后，由于部分患者无症状或症状轻微而忽视，故其实际发生率远高于诊断率，严重者可以发生视乳头水肿，部分患者误以为是低颅压综合征复发。大多数反射性高颅压患者病程有自限性，即使予乙酰唑胺治疗也可能需要较长时间恢复。此外，值得注意的是，合并假性脑瘤的自发性低颅压患者在治疗脑脊液漏后可能造成假性脑瘤症状再次出现，需予乙酰唑胺或外科手术治疗。颅内静脉窦血栓形成发生率较低，多见于活动性脑脊液漏患者头痛性质改变时，可予抗凝治疗。双上肢肌萎缩通常与蛛网膜外隙积液有关，表现为双上肢无力或肌萎缩，而感觉障碍不明显，应注意与运动神经元病（MND）相鉴别。脑表面铁沉积较为罕见，但仍有少数患者发展为自发性低颅压的远期并发症，可继发于脑脊液漏或神经根撕裂，合并蛛网膜外隙积液。

四、预后

自发性低颅压多呈良性，数天至数月后缓解，难治性或复发较少，其中多数遗留体位性头痛[10]。有研究显示，自发性低颅压复发率约为10%，内科保守治疗复发率约7.7%、外科治疗约38%，且头部MRI阳性患者临床预后较好[12]。Mcgrath等[47]报告1例妊娠15周时发生自发性低颅压的女性患者再次妊娠16周时复发，具体机制尚未明确，可能与妊娠有关。

五、总结

自发性低颅压是中枢神经系统少数可以完全治愈的疾病，其影像学检查和治疗方法尽管已较为成熟，但仍有需要研究的领域。未来应在此基础上着重进行针对病因学和临床预后的相关研究，以提高对疾病的认识。

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WFNS Congress Beijing 2019

Time: September 9–12, 2019

Venue: Beijing, China

Website: http://www.wfns2019.org/

The WFNS Congress Beijing 2019 will be held on September 9–12, 2019 in Beijing, China under the auspices of the World Federation of Neurosurgical Societies (WFNS), which is hosted by the Chinese Medical Doctor Association and Chinese Medical Association.

Founded in 1955, The WFNS is a professional and scientific non - governmental organization comprised of 130 members including 5 continental associations, 119 national or regional neurosurgical societies and 6 affiliate societies. WFNS is the highest academic organization of neurosurgery and the family of all neurosurgeons around the world. The WFNS Congress plays an important role in enhancing medical technology, strengthening academic exchanges and promoting collaborative research and exploration in neurosurgery and related disciplines.

"Glorious Neurosurgery" is the theme of WFNS Congress Beijing 2019. We will hold the opening ceremony on the Great Wall in the golden season. The conference hall is adjacent to the "Bird´s Nest", the main venue of the 2008 Summer Olympics and the 2022 Winter Olympics. Apart from a perfect scientific program, we will work hard to organize a wealth of cultural activities and very interesting tours for you and your companions. We will also invite 150 young neurosurgeons from the developing countries especially along the "Belt and Road" regions to attend the congress free of registration fee, food and accommodation. Furthermore, we will provide international return fares and a month-long clinical training afterwards in Beijing to 50 of them free of charge in food and accommodation.

Fifth European Stroke Organization Conference

Time: May 22–24, 2019

Venue: Milan, Italy

Website: http://eso-conference.org/2019/

The 5th European Stroke Organization Conference (ESOC) will take place in Milan, Italy, on May 22–24, 2019. ESOC 2019 will build on the enormous success of the last four European Stroke Organization (ESO) Conferences. ESOC is Europe´s leading forum for discussing and disseminating the latest advances in stroke care.

Over 1800 abstracts were submitted to ESOC 2018 in Gothenburg. In the large clinical trials sessions, results from 10 major randomized controlled trials (RCTs) were presented, many of which with accompanying high impact publications. Our delegate numbers continue to grow year on year and we are confident ESOC 2019 will be the largest yet.

One of the highlights of ESOC 2018 was the presentation of the "European Action Plan 2018–2030" which builds on the experience and the format of the previous Helsingborg Declarations. This document was written by ESO in cooperation with the patient organization Stroke Alliance for Europe (SAFE), with the involvement of the World Health Organization (WHO).

ESOC 2019 will see presentations of major clinical trials, state-of-the-art talks by renowned clinicians and researchers and receive updates on the latest guidelines. We will be joined by the Italian Stroke Organization.