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·讲座·

Interventional catheter methods for complex tetralogy of fallot and "Critical" PS/pulmonary atresia with intact interventional septum

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TOF is generally accepted as the index lesion used for evaluating the overall quality of a congenital cardiac program. To achieve good results, the evaluation and management of TOF requires an intimate collaboration between all members of the cardiac team involved in the care of these children, especially the interventional catheterizer and the surgeon. Each program has to develop its own approach, relying on the collective strengths of their staff. Any weak link in this chain will often have a profoundly negative effect upon the patient, which may be difficult to correct later.

Whereas the cath lab was the sole source of the anatomic and physiologic data for surgical planning, much of the information, especially intracardiac anatomy and physiology, can now be acquired with echocardiography, and if necessary, enhanced with MRI and CT. The role of diagnostic angiography is reserved for the elucidation of complex and small vascular structures beyond the resolution & specificity of less invasive methods, such as pulmonary artery branches, cornonary arteries and arterial collaterals. This anatomy becomes particularly important when staging procedures for TOF in the infant with small pulmonary arteries or atresia, when is the greatest potential for enhancing arterial size and vascular bed growth. Our approach is to perform early correction which will insure antegrade flow into the small pulmonary arteries, by opening the outflow tract(patch or conduit), and when feasible, closing the VSD. Aortic to pulmonary arterial shunts are avoided. Direct access into the pulmonary arteries allows additional catheter-based interventions. Which may include balloon dilation and stenting, which avoiding any deformity from peripheral shunts. Aortic collaterals perfusing lung segments competing with antegrade flow into the branch pulmonary arteries may need Giannturco coil occlusion. Those with small pulmonary arteries may require multiple ballooning/ stenting interventions to increase flow for "rehabilitation" and preserving RV function. Alternatively, some surgeons perform "unifocalization", which for the interventionist achieves the same direct antegrade access into the pulmonary arterial tree. Our eventual goals are to recruit the maximal functional pulmonary vascular bed with the lowest resistance, balancing the ventilation-perfusion ratio, while minimizing the RV pressure and volume overload. A residual ASD and/or VSD, either deliberately left as a palliative "fenestration" or an unwanted patch leak, can later be occluded with a device in the cath lab.

The natural history of RV to PA conduits has been disappointing. Frequently, these develop stenosis at the homograft valve or from an intimal peel. In these situation, short-term extension of conduit life can be accomplished with ballooning and/or stenting. If the conduit has an acute bend, usually from sternal compression, non-surgical relief is difficult to accomplish. For those chronically volume loaded and dysfunctional RVs, xenograft valves mounted into stents have been percutaneously delivered during catheterization, providing a competent pulmonary valve, leading to diminished symptoms. Early results look promising, and in select cases may prolong conduit longevity, obviating the need for surgical reoperation.

A more aggressive interventional approach has evolved for the treatment of pulmonary atresia with an intact interventricular septum. If the diagnostic evaluation reveals an adequate RV morphology and size for a two-ventricle approach, along with membranous pulmonary valve atresia and a non-RV dependent coronary circulation, an attempt at valve perforations is warranted. Frequently, the valve can be crossed gently with a guide wire, followed by progressive balloon dilations. If the valve is resistant to mechanical perforation, radio

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frequency or excimer laser is utilized to burn a hole through the membrane, followed by progressive dilations, with a resultant drop in RV pressure. This technique has had a significant morbidity and mortality, which has been diminishing as experience increases, shunting or outflow patching. Many of these will then require later interventions for re-dilation, shunt and/or foramen ovale closure. The initial selection of infants who will benefit from this catheterization approach, and who will continue with a two-ventricle circulation, is evolving, as the techniques and instrumentation.

Through a collaborative team approach, our techniques have advanced at both ends of the spectrum, form older adults with congenital heart disease to the newborn and soon, maybe to the fetus. Attempts have been made to open the RVOT in fetal pulmonary atresia with IVS to allow future RV and PA growth. Can the same be done in fetuses with TOF & PA?

TOF

Preoperative Intervention:

Indications for diagnostic cath in simple TOF

Pulmonary arterial anatomy

Coronary arterial anatomy

Multiple VSDs

Arch abnormalities & vascular rings-22q 11 microdeletion

Ballooning & stenting PDA

Ballooning pulmonary valve

Defining PA anatomy & alterative sources of PBF with pulmonary atresia

Selective arterial angiography

Pulmonary venous wedge angiography

Complimentary imaging techniques-MRI & CT

V/Q nuclear scanning

Postoperative Intervention: Palliative

Coiling residual aorta to PA shunts

Rehabilitating branch pulmonary arteries

Ballooning branch through RV to PA conduit

New equipment: Cutting balloons, stent designs

Coiling aortic collaterals that compete with antegrade PA flow

Occluding fenestrated VSDs-clamshell device

Postoperative Intervention: Corrective

Physiologic evaluation-shunt, gradients, regurgitation

Ballooning & stenting branch PA stenosis

Occluding residual VSDs-patch leaks

Muscular VSDs

Ballooning & stenting RV to PA conduit

Valve replacement of RV to PA conduit

Coronary revascularization

Is it too far off when TOF could be corrected in the cath lab

PA & IVS

Preoperative Intervention:

RV morphology (tripatite), volumes, z-scores

Tricuspid regurgitation and TV morphology

Pulmonary artery size and anatomy

Coronary artery pattern, circulation dependency, sinusoids

Atretic valve perforation: wire, radio-frequency, excimer laser

Balloon dilation

PDA stenting

Postoperative Intervention: Corrective

PFO closure

Blalock shunt closure

Repeat balloon dilation

Pulmonary artery dilation (surgical shunt sites)

Reference-TOF

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·病例报告　Case report·

肝细胞癌动脉化疗栓塞术后并发脑梗死一例

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Cerebral infarction aften transaterial shemoembolization for hepatocellular carcinoma a cage report　*WANG Han, WANG Jing-bing, ZHANG Gui-xiang, WANG Lin-chuan. Shanghai First People's Hospital, Jiaotong University, Shanghai* 200080, *China*

【**Key words**】 Hepatocellular carcionma; Complication; Cerebral infarction

肝细胞癌动脉栓塞治疗的并发症有很多,但栓塞术后并发脑梗塞的病例却十分罕见,我们在工作中遇到 1 例,现报道如下。

患者男,47 岁。始因右上腹不适感伴乏力 1 个月于 2003 年 4 月入院,收治我科介入病房。入院后经上腹部 CT 检查:肝右叶前上段大小约 10.3 cm × 8.9 cm × 9 cm 类圆形肿块,不均匀强化,门静脉期强化仍较明显,病变实质不均匀强化,包膜尚完整,其中央可见较小低密度不强化影;门静脉及其分支内未见异常密度影;腹膜后未见肿大淋巴结。经皮经肝穿刺病灶取活检证实为肝细胞癌。随后共在我科行动脉化疗栓塞术 4 次,每次间隔 2 个月。

2003 年 10 月患者第 4 次到我科行动脉化疗栓塞治疗,造影证实肿瘤血管并排除动-静脉分流后,经导管自肝固有动脉注入超液化碘油及 THP 混悬剂 10ml,自右膈下动脉注入超液化碘油及 THP 混悬剂 20 ml,复查造影见肿瘤栓塞彻底。术后返病房后即予常规预防感染及护肝处理。术后 5 h 患者诉右手感麻木,查体:神清,伸舌居中,右上肢肌力略减退,予低分子右旋糖酐 + 丹参静滴。用药后患者感症状好转。术后 12 h 患者出现烦躁,小便失禁。查体:BP120/80mmHg,神清,反应迟钝,查体欠合作,右鼻唇沟变浅,伸舌右偏,双瞳等大,对光反射(+),左半身痛觉减退,左上肢肌张力增高,左上、下肢肌力Ⅱ°,左下肢膝反射亢进,双侧巴氏征(-)。立即予留置导尿,脱水及营养神经治疗,并查头颅 CT 及 MRI。CT 显示:双侧额叶、顶叶灰质密度相对增高,双侧丘脑密度略增高(图 1)。MRI 见双侧小脑半球、双侧枕叶、额叶、顶叶皮层下白质和两侧丘脑斑片状异常信号(图 2,3)。术后 30 h,患者出现咳嗽症状,无痰,并感心前区不适。急症胸片未见异常,心电图示窦性心动过速,心率 100 次/min,QRS 低电压。予低流量吸氧及止咳对症处理,患者咳嗽及心前区不适症状很快消失。经脱水及营养神经治疗,术后第 3 天开始患者病情逐渐好转,左侧肢体感觉及肌力逐渐恢复。术后第 10 d 予拔除导尿管,患者能自行排尿,左侧肢体感觉恢复,肌张力正常,肌力Ⅳ°+,并能自行下床行走。术后第 12 d 予出院。术后 1 个月复查头颅 MRI 所见仍可见双侧小脑半球、双侧枕叶、额叶、顶叶皮层下白质和两侧丘脑斑片状异常信号,但表现已不明显(图 4)。

讨论　原发性肝癌动脉栓塞术后并发症有多种,但并发脑梗死甚为罕见,李小宝等[1]曾报道 1 例此类病例。脑梗死发生在膈下动脉碘油栓塞治疗后。膈下动脉常是原发性肝癌的重要供血动脉,经多年的临床应用,经膈下动脉化疗栓塞治疗肝癌已成为是一种安全有效的方法[2]。但由于膈下动脉供血区域的特点,使得其进行栓塞治疗时有发生特定并发症的风险,如呃逆、胸痛伴咳嗽和少量咯血、膈肌麻痹、心绞痛伴心电图异常、可逆性脊髓损伤等[3]。本例患者为巨块型肝细胞癌,第 4 次动脉化疗栓塞术后并发脑梗死。分析原因,对膈下动脉碘油乳剂栓塞仍应被列为"首要嫌疑"。考虑是在行膈下动脉栓塞治疗时,超液化碘油经微小的膈下动脉-膈下静脉(或膈下动脉-心包膈静脉)短路微量流失,进入

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