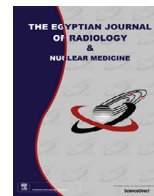




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## Original Article

## “Facial vascular anomalies; MRI and TRICKS-MR angiography diagnostic approach”

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## ABSTRACT

**Purpose:** Evaluate role of MRI and TRICKS-MR angiography in diagnosis of facial vascular anomalies.**Material and methods:** This study included 22 patients (mean age 9 years) with proved facial vascular anomalies on basis of interventional/surgical procedures (n = 19) or clinical follow up (n = 3). They underwent MRI examination with TRICKS-MRA. Images were evaluated for lesion location, size, feeding arteries and draining veins.**Results:** AVM was diagnosed in 15 patients (68.2%), hemangioma (5) patients (22.7%) and low flow venous malformation (2) patients (9.1%). TRICKS-MRA was accurate in diagnosis of 25 feeding arteries out of total 27 included in the study with 92.6% positive predictive value, 100% negative predictive value and 93.1% accuracy. AVMs were treated with sclerotherapy (26.7%), embolization (40%) and combined embolization and surgery (33.3%). Surgery was done in 2 hemangiomas (40%) while the other three patients (60%) underwent clinical follow up for 2 years with stationary course. The 2 patients with venous malformation underwent successful sclerotherapy.**Conclusion:** MRI & TRICKS-MRA provide excellent diagnostic data for assessment of facial vascular anomalies. TRICKS-MRA adds precise delineation of the vascular mapping, regarding feeding arteries and draining veins which helps in planning of therapeutic approach and reduces needing for invasive DSA.© 2017 The Egyptian Society of Radiology and Nuclear Medicine. Production and hosting by Elsevier. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## 1. Introduction

Vascular anomalies include broad spectrum of lesions involving all parts of the body, esp. the head and neck [1]. In 1996, the International Society for the Study of Vascular Anomalies (ISSVA) adopted Mulliken and Glowacki classification [2], dividing vascular anomalies into vascular tumors and vascular malformations, in combination with Jackson et al. [3] modification, subcategorized vascular malformations according to their flow dynamics as low-flow or high-flow malformations.

Vascular tumors include **hemangioma**, haemangioendotheliomas, tufted angiomas and sarcomas. Slow-flow vascular malformations including capillary malformations (CM), **venous malformations** (VM), lymphatic malformations (LM), capillary and venous malformations (CVM), capillary lymphatic and venous

malformations (CLVM), and high-flow malformations including arteriovenous fistula (AVF) and arteriovenous malformations (AVM) [1].

Diagnostic imaging plays an important role in the differential assessment of lesions and in planning the therapeutic approach [4].

Magnetic resonance imaging (MRI) serves as the principal imaging modality to diagnose and plan treatment for patients with cutaneous vascular anomalies [5]. T2-weighted images are mainly used to evaluate the extent of the abnormality; GRE images are used to identify the hemodynamic nature of the condition (high- vs low-flow lesion), and contrast-enhanced images are used to determine the extent of the malformation and to distinguish slow-flow vascular anomalies (VM versus lymphatic malformation [LM]). Many MR angiographic techniques have been developed for assessment of facial vascular anomalies [5,6].

Characteristic MRI findings of **haemangioma** include a focal, lobulated soft-tissue mass that is isointense relative to muscle on T1-weighted images and hyperintense on T2-weighted images. It has diffuse and homogeneous contrast enhancement and dilated feeding and draining vessels within and around the mass [7].

**AVMs** are characterized by abnormal connections between arteries and veins. These malformations have a central confluence

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of tortuous and dysplastic vessels (called a nidus), where the arterial blood is shunted to veins. On MRIs, the anomaly appears as enlarged vascular channels associated with dilated feeding and draining vessels “flow voids without parenchymal staining”. A discrete soft-tissue mass is typically absent. However, perilesional signal abnormalities, soft-tissue enhancement and/or mass-like features, atypical findings that makes these lesions more difficult to differentiate from hemangioma. These uncommon findings have been attributed to edema or a fibrofatty matrix within the surrounding tissues. MRA and MRV may be useful to confirm the high-flow nature of the lesion and to map out the feeding and draining vasculature [8].

The venous malformation (VM) is a spongy mass that is composed of abnormal veins (veins with a relative lack of smooth muscle cells in their walls). VMs are high-signal-intensity lesions on T2-weighted images and low-signal-intensity lesions on T1-weighted MRIs; they have lobulated margins and multiple, rounded, signal voids that represent phleboliths. Enhancement is patchy and central in VMs. Prominent or dysplastic draining veins may be identified with MRI [9].

Treatment of VM will depend on the sites involved and the extent of the VM. Sclerotherapy has become the current mainstream treatment for small, focal venous malformation. It can be used alone or combined with surgery and/or laser therapy. Multifocal or extensive VM are rarely cured but can be controlled with multiple treatments [10].

AVMs are the most challenging lesions to manage. Diagnostic imaging are prerequisites for anatomical assessment and analysis of the nidus, the site of arterio-venous shunting. Integrate surgical and non-surgical interventions should be utilized for optimum care. Treatment usually involve sclerotherapy, endovascular or percutaneous embolization, surgical excision, or some combinations of these modalities [6,11].

The approach to treatment of hemangiomas should be individualized, based upon the size of the lesion, morphology, location, presence or possibility of complications, potential for scarring or disfigurement, the age of the patient, and the rate of growth or involution at the time of evaluation and the potential risk of treatment. Active nonintervention (serial observation) is the mainstay of therapy for many uncomplicated, localized hemangiomas because hemangiomas involute spontaneously after the first year of life. Topical and intralesional corticosteroids, Pulsed-dye laser, Surgical therapies can also be used for management [12,13].

Although, MRA may allow useful information about feeding vessels of lesions, but they produce static images with prolonged acquisition time and cannot detect small vessels. On other hand Contrast-enhanced MRA improved the signal from the inflowing blood through the T1-shortening effect of gadolinium. It has drawbacks of being associated with high dose of contrast medium in addition to early venous filling with arteries overlapping [5].

While routine MRI protocols can lead to the correct diagnosis in the majority of cases, the imaging appearances can sometimes be nonspecific or confusing [5]. Time-resolved imaging of contrast kinetics (TRICKS) is a recently introduced technique of CE-MRA that acquires multiple 3D volumes during the passage of contrast agent bolus, so one can obtain dynamic filling of the arteries and veins similar to digital subtraction angiography (DSA) [13,14]. Many studies have demonstrated the advantage of this technique in assessment of the peripheral, intra and extracranial as well as spinal vascular lesions [15–20].

## 2. Aim of the study

The aim of this study was to evaluate the role of MRI and TRICKS-MR angiography in diagnosis of facial vascular anomalies

using the surgical/angiographic data of the patients through their medical files as gold standard reference.

## 3. Patients & methods

This prospective study was performed upon 30 consecutive patients referred to MRI unit at our institute during the period between June 2014 till June 2016. Ethics committee approval and informed written consent were obtained. Privacy & confidentiality of all patient data were guaranteed. All data provision were monitored and used for scientific purpose only.

### 3.1. Patients

#### 3.1.1. Inclusion criteria

- Patient with suspected facial vascular malformation on clinical bases (facial soft compressible swelling with overlying skin discoloration ( $n = 12$ ), dilated subcutaneous vessels ( $n = 9$ ) or pulsatile swelling ( $n = 8$ ).
- No age restriction.

#### 3.1.2. The exclusion criteria

- General MRI contraindication; claustrophobic patients & extreme overweight.
- Contraindication to contrast agents; impaired renal function with GFR  $<30$  mL/min/1.73 m<sup>2</sup>.

Three patients were excluded from the study because of poor image quality secondary to inadequate sedation. Five patients were excluded because of lack proved diagnosis whether on interventional/surgical procedures or clinical follow up basis.

The remaining 22 patients with proved facial vascular anomalies were included in the study; 19 patients underwent interventional/surgical procedures and 3 patients underwent clinical follow up with stationary course. They were 12 males and 10 females with age ranged between (2 months–23 years). Sedation was used in 13 patients; oral chloral hydrate was used in (7) infants under 2 years, while IV propofol (Diprivan) was used in its sedative dose 1–1.5 mg/kg in 6 children.

### 3.2. Method

All patients of our study were subjected to the followings;

- Full history taking.
- Clinical examination: Including general examination for other similar lesions and local examination for the facial anomaly or swelling.
- Laboratory investigations with stress upon the renal function tests.
- Doppler U/S examination for accessible lesions.
- MRI and TRICKS-MRA Examination.

#### 3.2.1. MR examinations

MRIs were performed by using a closed MRI machine 1.5-T magnet (Signa Excite HD; GE Healthcare, Milwaukee, USA). Patients were examined in the supine position using head and neck phased array coil (eight channel head array). The following sequences were acquired: T1-weighted images (TR; 405–750, TE; 9–18 ms) and T2-weighted images (TR; 4440–6340, TE; 96–100 ms), STIR sequence (TR; 3800–6500, TE; 37–55 ms) or T2 Fat Sat (TR 2850–4000, TE 80–120).

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