



VM treatment options in 2016

A Bisdorff-Bresson¹, G Soulez², A Aymard, JL Gérard³, D Salvan, E Sauvaget, B Faucon, M Borsik, JP Saint Maurice¹, F Lemarchand-Venencie, C Laurian, P Herman, E Houdart

Consultation des Angiomes, Hôpital Lariboisière, Paris, France

¹ Sce d'ORL et Neuroradiology de l'hôpital Lariboisière , Paris ² Montréal, Canada ³Hôpital Henri Mondor, Paris





Disclosure	
Speaker name:	
A Bisdorff Bresson	
I have the following potential conflicts of interest to report:	
Consulting	
□ Employment in industry	
Shareholder in a healthcare company	
Owner of a healthcare company	
□ Other(s)	
☐ I do not have any potential conflict of interest	











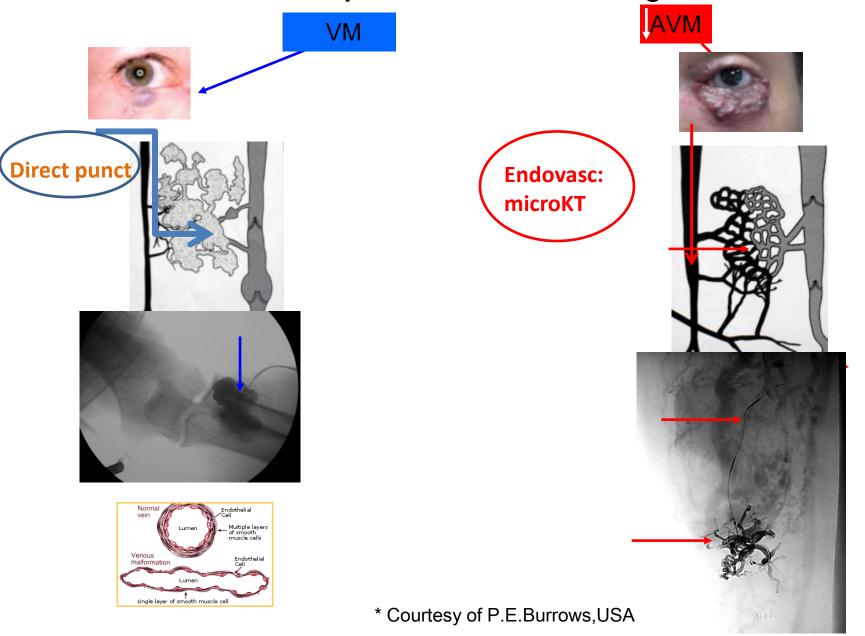








Principle of Vascular malformations TTT Slow flow: direct punct versus High flow



Sclerosant agents

 Liquid Agents : Foam /ETOH/Bléomycine, Doxycycline (résorbable +++)

NON

Résorbable

- Semi liquid Agents: Glue/Onyx/Ethanol gel
- Permanent Agents : Plug/Coils
- Thermic endovenous ablation / Radiofrequency



Agent choice depends on malformation location (deep or superficial) + lesion extension:
Resorbable agent or not??

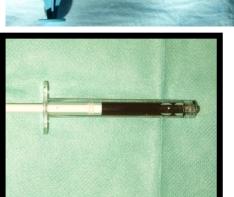
- VM location : cervico facial : sclerotherapy ++++ vs Extemities : sclero versus / surgery ++
- Superficial lesions: prefer: liquid sclerosant agents: ETOH/ Aetoxisclerol/Sotradecol FOAM/ Bleomycine/ Sclerosant gel/ Endovenous laser
- Deep lesions: ETOH /Gel/ Glue /Onyx

Draining veins : Coils /amplatzer/ Glue

No particles embolisation required in VM treatment

Choice: Evaluate **complication risk** 2 d sclerosant agent and lesion location: superficial vs deep















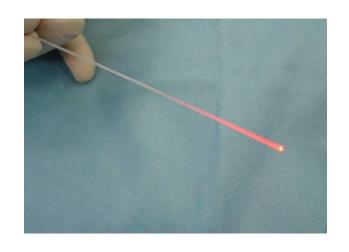
- Skin necrosis
- Transient paresis





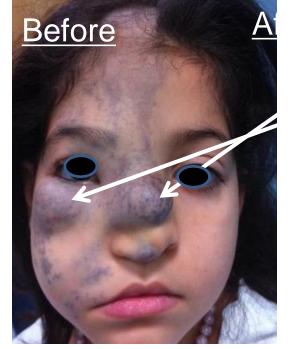


Superficial lesions

























Aetoxisclerol Foam and endovenous laser TTT

Aetoxisclerol 3% foam: 1/3 dilution





GVM

Necrosis Aetoxi 3% foam: 50-50%





II) Endovenous laser treatment









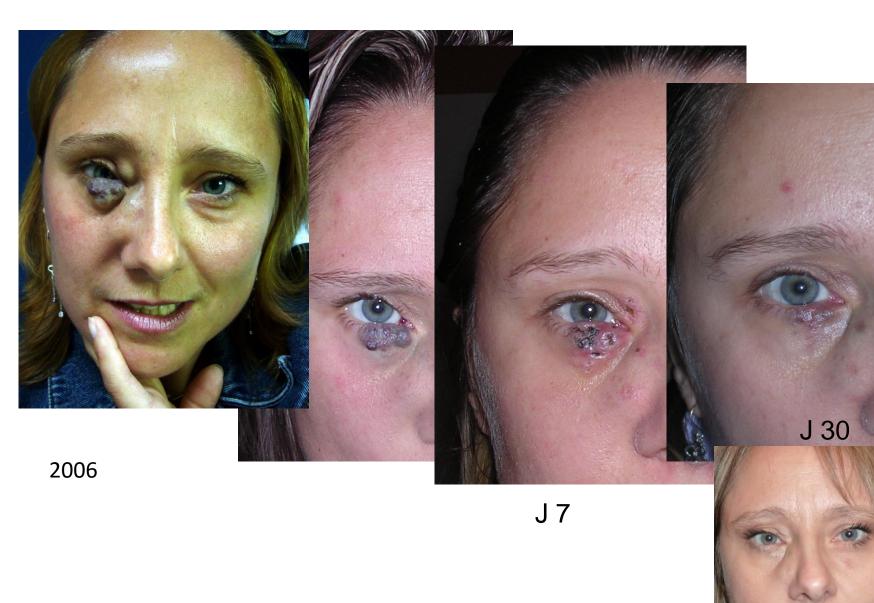






2 MO after 1 Laser session

Dr Larralde, Dr Aillet, Rennes



2012

B Faucon







1 Mo after 1 laser + Aetoxisclero session A Larralde, Rennes

III) GEL





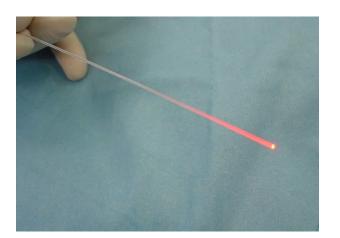














ETOH

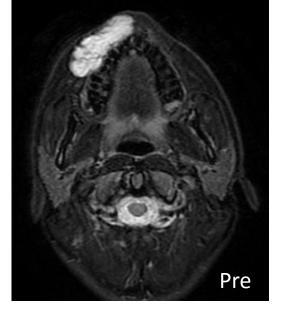
- Requires GA
- Powerfull sclerosant agent
- Less expensive

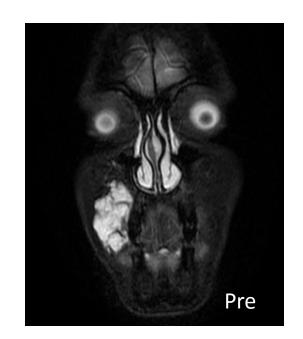
But Major complications: 2d ETOH sclerotherapy

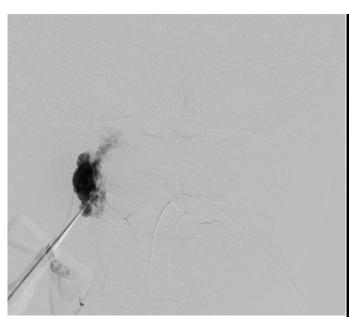
- Venous drainage ++ : DVP /PE
- Transient parlaysis
- PAHT
- Arterial : occlusion (rare)
- Death: 2 cases in France

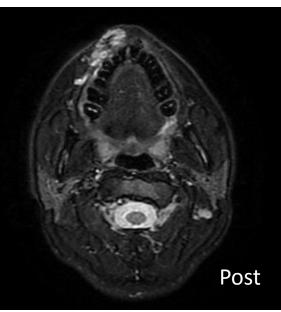
I) ETOH/Lipiodol mixture

Direct puncture : ETOH





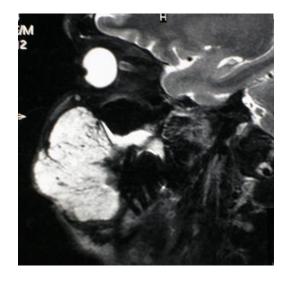






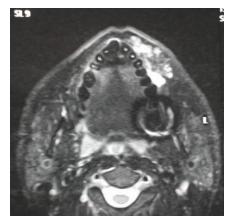
Laribosière Hospital

Before

















after



before

after

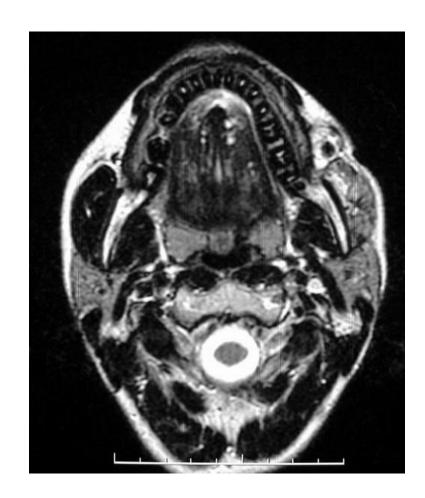




Before After

Before After





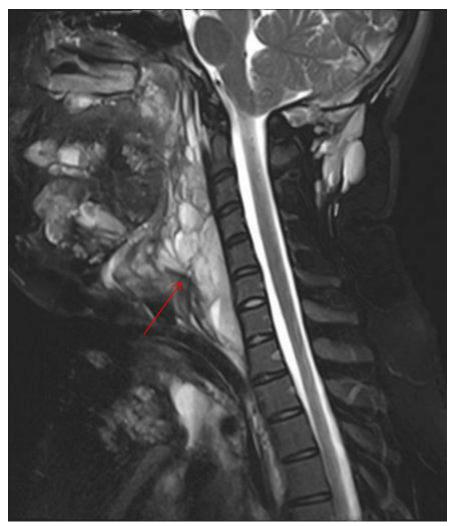
II) Bleomycine = liquid sclerosant agent

Glycopeptide antibiotic produced by the bacterium streptomyces verticillus. Usually, it is used as an **antineoplastic drug** to treat many kinds of cancer, such as lymphoma, cervical cancer, head and neck cancer, and testicular cancer.

Bleomycin A5, also named **pingyangmycin**, is the most commonly used sclerosing agent for the treatment of vascular anomalies in China

Due to potential toxicity related to **pulmonary fibrosis** there is limited experience with Bleomycin for the treatment of VM/LM in North America and Europe .







Before
Courtesy PE Burrows, USA

<u>After</u>



Long

Pricy \$\$\$





III) Sclerogel=ETOH gel in between liquid /non resorbable material





Less painfull ETOH, slowly resorbable

III) Deep IMVM: Calf region non resorbable agent might be used



ONYX

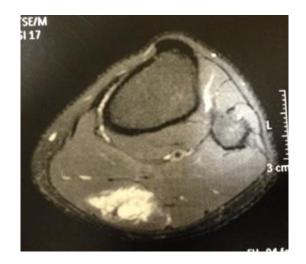












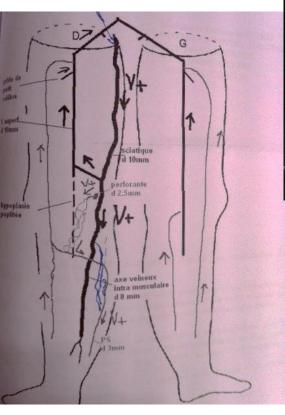




IV) Non resorbable agent: NBCA cast



Truncular VM



Milka Greiner

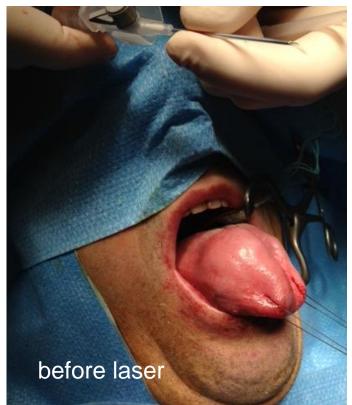


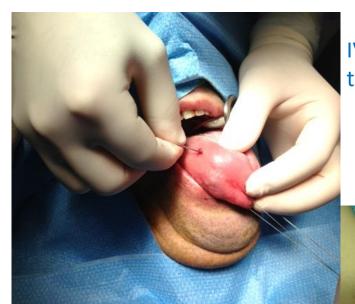
Prior glue embo





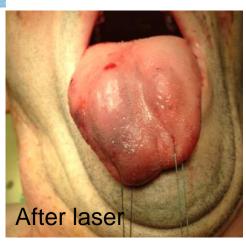






IV) Endovenous laser treatment

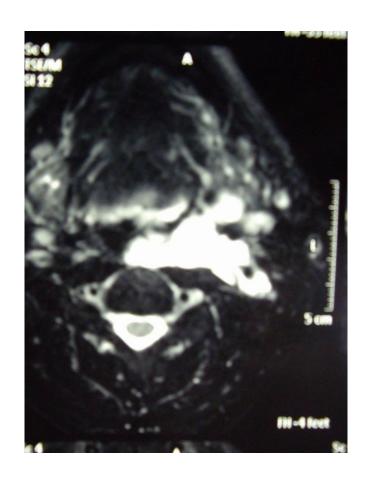






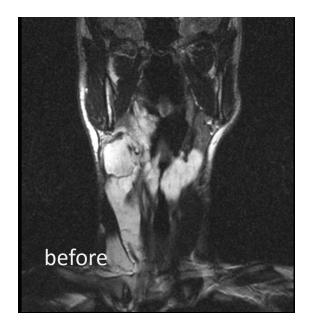
IV) Endovenous laser treatment





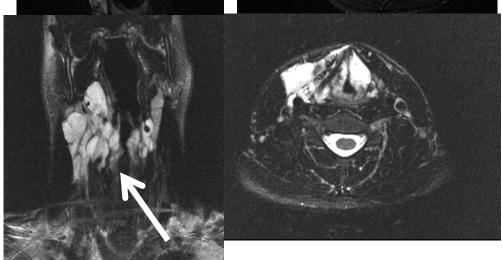
Before and after endovascular laser treatment : post wall> lateral wall







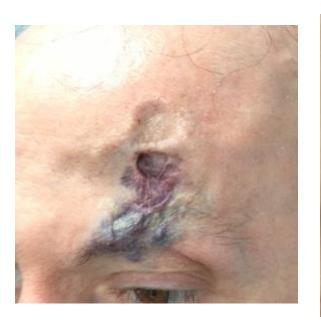




BUT: Surgery remains is still a good TTT option for some locations

Indications

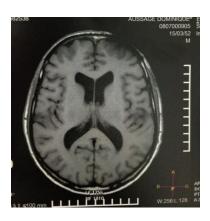
Large extensive lesions
Connections large draining veins with deep system
Thromboses lesions







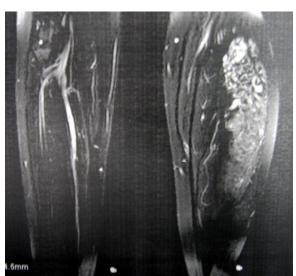




Excision surgery Frontal VM ,B Faucon, D Bresson

Excision surgery of Calf VM



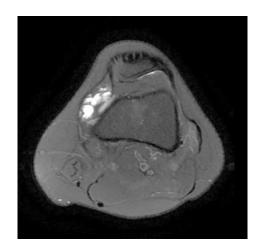




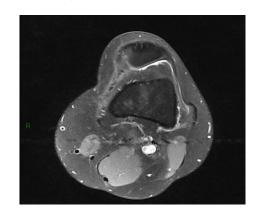


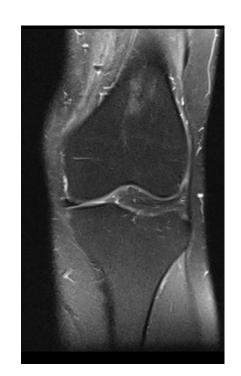


CI Laurian



RequiresPre op US +++ , **no** preop tourriquet ++





Excision surgery of IMVM





Cl. Laurian

The Journal of Clinical Investigation

R

Rapamycin improves TIE2-mutated venous malformation in murine model and human su

Elisa Boscolo,¹ Nisha Limaye,² Lan Huang,¹ Kyu-Tae Kang,¹ Julie Soblet,² Melanie Uebelhoer,² Antonella Me Emmanuel Seront,⁴ Sophie Dupont,⁴ Jennifer Hammer,⁵ Catherine Legrand,⁶ Carlo Brugnara,ˀ Lauri Eklund, Joyce Bischoff,¹ and Laurence M. Boon²,⁵

Vascular Biology Program and Department of Surgery, Boston Children's Hospital, Harvard Medical School, Boston, Massachusetts, USA. ²Human Molecular Genetics Université catholique de Louvain, Brussels, Belgium. ³Oulu Center for Cell-Matrix Research, Biocenter Oulu and Department of Medical Biochemistry and Molecular Boulu, Finland. ⁴Centre du Cancer, Department of Pediatric Oncology, and ⁵Center for Vascular Anomalies, Division of Plastic Surgery, Cliniques Universitaires Saint Luc, Brussels, Belgium. ⁶Institute of Statistics, Biostatistics, and Actuarial Sciences, Université catholique de Louvain, Louvain-la-Neuve, Belgium. ⁷Department of Labora Boston Children's Hospital, Harvard Medical School, Boston, Massachusetts, USA.

Venous malformations (VMs) are composed of ectatic veins with scarce smooth muscle cell coverage. Activation the endothelial cell tyrosine kinase receptor TIE2 are a common cause of these lesions. VMs cause deform local intravascular coagulopathy, and they expand with time. Targeted pharmacological therapies are not avacondition. Here, we generated a model of VMs by injecting HUVECs expressing the most frequent VM-causing TIE2-L914F, into immune-deficient mice. TIE2-L914F-expressing HUVECs formed VMs with ectatic blood-fill that enlarged over time. We tested both rapamycin and a TIE2 tyrosine kinase inhibitor (TIE2-TKI) for their expression and for their ability to inhibit mutant TIE2 signaling. Rapamycin prevented VM growth, while

no effect. In cultured TIE2-L914F-expressing HUVECs, rapamycin effectively reduced mutant TIE2-induced A

Conclusion

Agent choice depends on VM location and extension

Resorbable or non resorbable

Endovenous laser treatment can be an option

Surgery remains an option in some cases

Futur Medical treatment : Rapamycine ? Others ?

Mail: annouk.bisdorff@aphp.fr



A Aymard
M Borsik
C Degrugillier-Chopinet
C Laurian
L Drouet
M Wassef
N Leclerc

Our Mentors:



JJ Merland et MC Riché



Odile Enjolras