Three points may follow from these observations. Firstly, an INR of 1.5 might not be 'enough' to prevent rebleeding. Secondly, the INR does not fully reflect the pathological mechanisms of rebleeding in OAC ICH. Thirdly, further mechanisms than just the derangement of the coagulation system are responsible for ICH in patients on OAC.

To investigate the therapeutic efficacy of rFVIIa versus placebo in patients with spontaneous ICH the Spot Sign for Predicting and Treating ICH Growth Study (STOP-IT) has been started [8]. The INR normalization in patients with Coumadin-related intracranial hemorrhages trial (INCH) will compare the efficacy of PCC and fresh-frozen plasma [9]. These trials will add information on the selective power of the spot sign in spontaneous ICH and the pathophysiological mechanisms behind as well as the treatment of OAC ICH.

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Timolaos Rizos
University of Heidelberg, Department of Neurology
Im Neuenheimer Feld 400
DE-69120 Heidelberg (Germany)
Tel. +49 6221 56 8211, Fax +49 6221 56 5348
E-Mail Timolaos.Rizos@med.uni-heidelberg.de

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Xerostomia after Acute Stroke

Gillian D. Kerr^a, Cameron Sellars^a, Lynsey Bowie^a, Jeremy Bagg^b, M. Petrina Sweeney^b, Peter Langhorne^a, David J. Stott^a

^aAcademic Section of Geriatric Medicine and ^bGlasgow Dental School, Faculty of Medicine, University of Glasgow, Glasgow, UK

Introduction

Dry mouth (xerostomia) is potentially uncomfortable, leads to increased periodontal disease [1] and may contribute to dysphagia. In acute stroke, reduced saliva may lead to abnormal oral bacterial colonisation and contribute to the risk of pneumonia [2].

Methods

We carried out a prospective study of consecutive stroke admissions to the Glasgow Royal Infirmary (June 2004 to November 2005). The study had ethics approval.

Salivary flow rates (unstimulated) were measured on a single occasion by Salivette[®] sampling [3]. The oral cavity was assessed with the Oral Assessment Guide [4], oral flora by imprint culture, and dysphagia using 3 standard assessment tools [5–7]. We recorded pre-morbid modified Rankin Scale (mRS) scores, medical history, medication, National Institutes of Health Stroke Scale (NIHSS) scores and Abbreviated Mental Test scores. Socio-economic status was derived from postcodes using Scottish Neighbourhood Statistics [8]. At a 3-month follow-up, mRS scores were determined.

Data were analysed using SPSS 15.0. Characteristics of patients with dry mouth (salivary flow <1 µl/min) were compared to those with a salivary flow of 1–120 and >120 µl/min (normal salivary flow). Statistical significance was accepted at $p < 0.05$. All probabilities are 2-tailed. To examine the independent risk factors for dry mouth after acute stroke, binary logistic-regression analysis was performed with the dependent variable dry mouth or no dry mouth (salivary flow ≥ 1 µl/min).

Results

Oral status assessment was performed in 368 of 412 eligible patients, 225 (61%) had xerostomia. In univariate analysis (table 1), pre-stroke factors associated with xerostomia included older age, female gender, number of medications and pre-stroke disability; post-stroke associates were: stroke severity, Abbreviated Mental Test score, raised C-reactive protein, urinary tract infection (UTI), other clinical infections and oral *Candida glabrata*. There was no significant association of xerostomia with socio-economic status, oral cavity health, bacterial colonisation, dysphagia, pneumonia or risk of death/disability at 3 months. The independent associates of dry mouth were pre-stroke disability, UTI and oral *Candida glabrata*.

Discussion

We found that xerostomia is very common in acute stroke patients. This study also confirmed factors associated with xerostomia in non-stroke populations (older age, female gender, number of medications, disability). In addition, we identified several as-

Table 1. Clinical characteristics and basic laboratory investigation results of acute stroke patients categorised by salivary flow

	Salivary flow			p value
	<1 µl/min (n = 225)	1–120 µl/min (n = 69)	>120 µl/min (n = 74)	
Patients, %	61.1	18.1	20.1	–
Age, years	68.4 ± 13.9	68.6 ± 13.3	62.4 ± 13.9	0.004
Males:females	96:129	34:35	54:20	<0.001
<i>Pre-stroke characteristics</i>				
Number of medications (median, IQR)	6 (3, 9), n = 219	5 (3, 5), n = 65	5 (1, 7), n = 71	0.006
Smoker	102 (45.3)	38 (55.1)	36 (48.6)	0.362
Diabetes mellitus	46 (20.4)	9 (13.0)	10 (13.5)	0.214
Pre-stroke mRS (median, IQR)	0 (0, 2), n = 207	0 (0, 0), n = 62	0 (0, 0), n = 67	<0.001
Socio-economic deprivation (in lowest 10% of Scottish population)	132 (58.7)	34 (49.3)	46 (62.2)	0.260
<i>Post-stroke characteristics</i>				
NIHSS score (median, IQR)	5 (3, 9), n = 224	4 (2, 10)	3 (2, 6.5), n = 73	0.015
mRS (median, IQR)	3 (2, 4)	3 (2, 4)	2 (1.8, 4)	0.001
AMT score (median, IQR)	8 (4, 9.5), n = 223	8 (4.6, 9.9), n = 68	9 (7, 10), n = 71	0.036
C-reactive protein (geometric mean, 95% CI), mg/l	24.5 (20.5–29.3)	20.4 (15.3–27.6)	14.1 (11.3–17.7)	0.004
Serum urea, mmol/l	6.6 (2.9)	6.3 (3.1)	6.1 (3.1)	0.362
Pneumonia	33 (15), n = 220	10 (14.5)	9 (12.5)	0.837
Urinary tract infection	43 (19.2), n = 224	6 (8.7)	2 (2.7)	0.001
Other infection	30 (13.5), n = 222	6 (8.8), n = 68	2 (2.7), n = 74	0.028
<i>Oral health/flora</i>				
Oral Assessment Guide score (median, IQR)	10 (7, 13), n = 223	10 (8, 12), n = 69	9 (8, 12), n = 74	0.225
Gram-positive bacteria	35 (15.6)	7 (10.1)	9 (12.2)	0.468
Gram-negative bacteria	30 (13.3)	7 (10.1)	8 (10.8)	0.714
<i>Candida albicans</i>	105 (46.7)	35 (50.7)	27 (36.5)	0.191
<i>Candida glabrata</i>	42 (18.7)	6 (8.7)	4 (5.4)	0.006
<i>Markers of dysphagia</i>				
Failed water swallow test	53 (23.6)	13 (18.8)	9 (12.2)	0.101
Logemann total score	1 (0, 5), n = 218	1.5 (0, 5), n = 68	1 (0, 3.5), n = 73	0.252
Daniels severity of dysphagia score	0 (0, 1), n = 218	0 (0, 2), n = 68	0 (0, 1), n = 73	0.622

Results of continuous variables are means ± SD, and categorical variables are given as number and %, except where stated. Data are complete unless otherwise stated. Statistical analysis was by ANOVA (normally distributed continuous variables), Kruskal-Wallis H test (non-normally distributed continuous variables), and χ^2 test (categorical variables).

sociated post-stroke factors: UTI and oral *Candida glabrata* were independently associated. Xerostomia was independently associated with UTI, but not with pneumonia or oral bacterial colonisation. It may be that the association of dry mouth and UTI reflects a common process such as dehydration. There was a significant association of xerostomia with *Candida glabrata*. This is an emerging oral opportunistic pathogen, and now the second most commonly found oral *Candida* spp. [9]. The clinical significance of this finding is not clear.

We believe this to be the first prospective cohort study of salivary flow and acute stroke. Oral health assessments were thorough and used validated tools. However, there are some potential weaknesses. Not all consecutive admissions were included and salivary flow was measured on 1 occasion only.

Further research is needed to establish the time course of post-stroke xerostomia and the longer-term association with patient discomfort, oral health and dysphagia. The associations of xerostomia with UTI and oral *Candida glabrata* also require confirmation. The role of interventions for dry mouth, including artificial saliva, merits investigation.

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Dr. Gillian D. Kerr
Academic Section of Geriatric Medicine
3rd Floor Queen Elizabeth Building, Royal Infirmary
Glasgow G31 2ER (UK)
Tel. +44 141 211 5088, Fax +44 141 211 4033
E-Mail gilliandkerr@hotmail.com

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The Agony of Choice: Diagnosis of Cervical Artery Dissection by Means of Duplexsonography or Magnetic Resonance Imaging?

Saskia H. Meves^a, Stephan Salmen^a, Peter Mönnings^b,
Aiden Haghighi^a, Ralf Gold^a, Christos Krogias^a

Departments of ^aNeurology, and ^bRadiology, St. Josef Hospital,
Ruhr University Bochum, Bochum, Germany

A sharp rise in the diagnosis of dissection of extracranial brain supplying arteries as a cause of cerebral ischemia in young adults has been noted in the recent past due to increased and intensified clinical awareness as well as advanced technical options [1]. On the basis of 2 cases, we report on the diagnostic challenge to the clinician and the difficulties involved when using noninvasive imaging technologies such as extracranial duplexsonography (ECD) and magnetic resonance imaging (MRI).

Case 1

A 27-year-old female patient complained of diffuse headaches and a slight pain in the left cervical region after a cupboard weigh-

ing about 100 kg had fallen against her head. The neurological examination was unrevealing except for a slight ptosis of the left eyelid and a mild holocranial diffuse headache.

The initial ECD showed no abnormalities. However, due to the described severity of the trauma caused by the weight of the heavy cupboard and the left ptosis, cervical MRI with axial T1-fat-saturated sequences as well as MR angiography were performed. These revealed significant signal increase of the left common carotid artery in the fat-saturated sequences, whereas the MR angiography showed no abnormalities (fig. 1a, b). There was a visible extension of the signal increase into the internal carotid artery in follow-up MRIs, but the follow-up ECDs were unable to detect any flow or vessel wall abnormality.

Case 2

A 38-year-old male patient reported that he had felt a sudden slap in his neck and a sudden attack of rotatory vertigo the day before. Due to the persistence of an unsteady gait and a new onset of numbness of the left upper arm, he came to our emergency department. Except for the diffuse hypesthesia of the left upper arm, the neurological examination detected no other abnormalities.

ECD showed increased flow velocities of 220 cm/s, raised pulsatility and a diastolic backflow in the V3-segment of the right vertebral artery. The following MRI with fat-suppressed images on day 2 after the initial symptoms showed hypoplasia of the right vertebral artery, but the diagnosis of a dissection could not be confirmed (fig. 1c). Within 2 days the ECD abnormalities normalized. A MRI follow-up 2 weeks later reconfirmed the diagnosis of dissection (fig. 1d).

Discussion

These 2 cases demonstrate that dissections can be missed by either ECD or MRI when used as single method. Our first patient came with 1-sided neck pain, along with a partial Horner's syndrome. Even though she showed no signs of focal cerebral ischemia and ECD was normal, we carried out a MRI because her symptoms and the severe trauma [2] clinically suggested the diagnosis of dissection. Schievink [1] has stated that the classic triad of localized pain, Horner's syndrome and ischemia is found in less than one third of patients with carotid dissection, but the presence of any 2 elements of this triad strongly implies the diagnosis of dissection. The clinical presentation of the second patient (sudden unilateral neck pain, symptoms of vertebrobasilar ischemia) has led to the diagnosis of vertebral dissection. It was confirmed by means of ECD but not by the initial MRI. MRI confirmation was obtained 2 weeks later.

ECD has its limitations in demonstrating dissection in patients in whom intramural hematoma causes only slight morphological changes of the vessel wall or hemodynamic-nonrelevant lesions [3]. Thorough ultrasound examinations reveal roughly 13% negative findings in the initial examination of cervical artery dissection [4]. The sensitivity of ultrasound for the detection of cerebral artery dissection has been found in general to be 92% for both vascular territories [5], yet only 69% in patients without cerebral ischemia but with isolated Horner's syndrome [6].

The visualization of vessel hematoma can be difficult to depict by MRI during the first 3 days, as reflected in case 2. During this phase of the dissection, the rim of the acute intramural hematoma consists of deoxyhemoglobin and may not reveal hyperintensity, but rather appear isointense relative to the adjacent muscle [7, 8].