

Critical Review Form

Therapy

A transient ischaemic attack clinic with round-the-clock access (SOS-TIA): feasibility and effects, *Lancet-Neurology* 2007; 6: 953-960

Objectives: To evaluate the effect of a hospital-based TIA clinic available 24 hours/day 7-days/week with standardized clinical assessment followed by initiation of a comprehensive stroke prevention program.

Methods:

Family physicians, Neurologists, Ophthalmologists and EM physicians were informed of the SOS-TIA clinic availability via leaflets. By phoning the clinic with potential patients, they reached a stroke nurse by 9a – 5p or a Neurologist 5p – 9a. They were admitted to SOS-TIA immediately after the phone call if they had focal symptoms of brain or retinal dysfunction that were of sudden onset and presumed to be related to ischaemia and if the patients had subsequently made a total recovery”. (p.954)

A vascular neurologist was responsible for the decision to exclude patients and their standardized clinical assessment occurred within 4-hours. At the discretion of the vascular neurologist, testing included brain imaging (MRI and/or CT), carotid and transcranial dopplers, ECG and echocardiography, blood cell counts, glucose, lipid profile and C-reactive protein were also obtained.

After evaluation the neurologist contacted the referring physician to discuss the diagnosis and most appropriate treatment. Targets included BP <140/90 in non-diabetic patients and LDL <100 mg/dl. Follow-up was obtained by face-to-face Neurology office re-evaluation or nurse telephone. The primary outcomes were stroke at 90-days and stroke/MI/vascular death at 1-year with these outcomes confirmed via medical record reviews whenever possible. Endpoints were confirmed by a consensus of 2 Neurologists. The authors used the ABCD² score to compare the outcomes of their cohort with historical groups. Finally, the authors analyzed subgroups of those presenting with 24 h of symptom onset, ABCD² ≥ 4 or those with motor or speech impairment testing > 10 days for post-SOS-TIA outcomes. Stroke unit admission criteria included high-grade arterial stenosis, low blood flow in MCA, potential cardiac embolic source (SBE, prosthetic valve, ACS, aortic dissection), crescendo TIA, or suspected paroxysmal arterial fibrillation.



Guide		Comments
I.	Are the results valid?	
A.	Did experimental and control groups begin the study with a similar prognosis (answer the questions posed below)?	
1.	Were patients randomized?	No – this is not a randomized trial so subject to multiple forms of bias including selection, ascertainment and co-intervention bias.
2.	Was randomization concealed (blinded)?	No – not randomized
3.	Were patients analyzed in the groups to which they were randomized?	No randomized, analyzed as one group.
4.	Were patients in the treatment and control groups similar with respect to known prognostic factors?	No treatment and control group.
B.	Did experimental and control groups retain a similar prognosis after the study started (answer the questions posed below)?	
1.	Were patients aware of group allocation?	Yes – all allocated to some intervention.
2.	Were clinicians aware of group allocation?	Yes.
3.	Were outcome assessors aware of group allocation?	Yes.
4.	Was follow-up complete?	“1052 (97%) patients were followed up for a median of 16 months (IQR 12-19) after presentation at the TIA clinic, and the remaining 33 patients (3%) were lost to follow-up”. (p.957)
II.	What are the results (answer the questions posed below)?	

1.	How large was the treatment effect?	<ul style="list-style-type: none"> • 1085 patients were admitted to the SOS-TIA clinic between Jan 2003 and Dec 2005 averaging 30/month & 22% did not have a TIA. • 87% were seen by a Neurologist within 24 h of the telephone call. • 53% were seen within 24 h of symptom onset, 61% within 48 h, and 75% within 7 days. • For TIA without brain tissue damage, the median symptom duration was 10-minutes IIQR 3-30 min) c/w 15 minutes for TIA with brain damage and 30-minutes for non-ischemic diagnosis. • 99% had brain imaging, 71% had MRA, 61% echo, and 95% had brain imaging, arterial exploration and echo. • Causes were identified in 41%, 64%, and 74%, respectively for those with definite TIA without brain damage, minor stroke, and definite TIA with brain damage. • 26% were admitted to stroke unit with median LOS 4 days. • Among those with definite TIA, possible TIA, or minor stroke anti-thrombotic therapy was given immediately in 98%. • 76% of atrial fibrillation with definite TIA received oral anticoagulants • Anti-HTN treatment was started/modified in 24% definite TIA and 43% of definite TIA with brain damage. • Lipid lowering agents were started in 45% patients. • The leading etiology of TIA was atherothrombosis (24 – 38%) followed by cardioembolic (10 – 15%), though among TIA without new brain lesion 59% remained unknown. • Among all patients the 90-day stroke rate was 1.24% (95% CI 0.72 – 2.12) compared with ABCD² predicted rate of 5.96%. This represents a 4.72% ARR and NNT = 21. If we assume all 33 lost to follow-up had a stroke the 90-day stroke rate would increase to 4.4% and NNT = 64. • Among TIA patients without new lesion the 90-day stroke rate was 1.34% c/w ABCD² predicted 6.13%
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		<ul style="list-style-type: none"> The 1-year risk of MI or vascular death was 1.1%, lower than those reported in separate meta-analyses. The median LOS was less than 1-day c/w other Paris hospital TIA patients LOS of 6-8 days.
2.	How precise was the estimate of the treatment effect?	Narrow inter-quartile ranges and 95% CI as reported above.
III.	How can I apply the results to patient care (answer the questions posed below)?	
1.	Were the study patients similar to my patient?	Not entirely. These were a mix of primary, specialist, and emergency patients.
2.	Were all clinically important outcomes considered?	No – the authors did not assess patient satisfaction or QOL.
3.	Are the likely treatment benefits worth the potential harm and costs?	Yes, if similar infrastructures can be designed elsewhere to expedite TIA evaluation and evidence-based intervention to reduce post-TIA stroke rates and a portion of the \$57 billion spent annually in the US alone.

Limitations

1. **Non-randomized, non-blinded trial so subject to multiple forms of bias including:**
 - a) **Selection bias** – the sickest subjects were likely referred to and admitted to local hospitals. If so, the healthier subject enrolled in SOS-TIA might be expected to display a more favorable prognosis than the historical cohort of ABCD².
 - b) **Co-intervention bias** – awareness of the study by patients, families, and clinicians might produce a Hawthorne-effect of interventions which normally would not have occurred outside the study setting. For example, clinicians aware that their patient’s outcomes are being monitored might spend a few extra minutes at the next office visit to discuss med compliance and review signs/symptoms of stroke.

- c) **Ascertainment bias.** – investigators believing in the results of SOS-TIA might have searched diligently for risk factors and outcomes via index interview or phone follow-up that normally would not have been discovered outside the study setting.
2. **No follow-up data on patient or referring physician compliance with recommendations.**
 3. **Insufficient data for separate analysis of EM patients.**
 4. **Median follow-up interview at 16-months subject to recall bias.**

Bottom Line

A TIA clinic available around-the-clock appears to reduce post-TIA and post-minor stroke subsequent 90-day and 1-yr stroke rates compared with historical cohorts with NNT=21. Future randomized trials should confirm these results in alternative settings while describing the expense and local acceptance of such rapid access clinics. Additionally, patients' important outcomes such as satisfaction and QOL should be reported. Any rapid access clinic would benefit from a community education arm since 42% of patients do not seek medical attention within 24-hours of symptom onset.

