

TIA triage in 2016

Not all that glitters is gold

Disclosures

- No industry related disclosures
- Expert witness work

Overview

- Definition
- Implications
- Guidelines, secondary prevention
- Implementation of guidelines

Definition

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- Key features¹
 - Sudden, as opposed to rapid
 - Absence of nonspecific symptoms

TIA

- *A TIA is a sudden neurological deficit, caused by a vascular problem, that completely resolves.*
 - 20 min to 3 hrs
 - There may or may not be evidence of infarction on MRI/DWI

Case 1

- 82yoM with Atrial Fibrillation (INR 1.6) and old right frontal lobe ischemic stroke.
- P/W sudden speech problems, 19:45, totally resolved by 21:00 in the ED
- NIHSS=0 in the ED
- HCT unremarkable (except old infarct)

TIA: short term risk of stroke

Study (year)	2 days	7 days	30 days
Johnston (2000)	5%		
Lovett (2003)		9%	12%
Gladstone (2004)			8%
Coull (2004)		8%	12%
Kleindorfer (2004)	4%	7%	11%
Correia (2006)		13%	
Average	4.5%	8.5%	11%

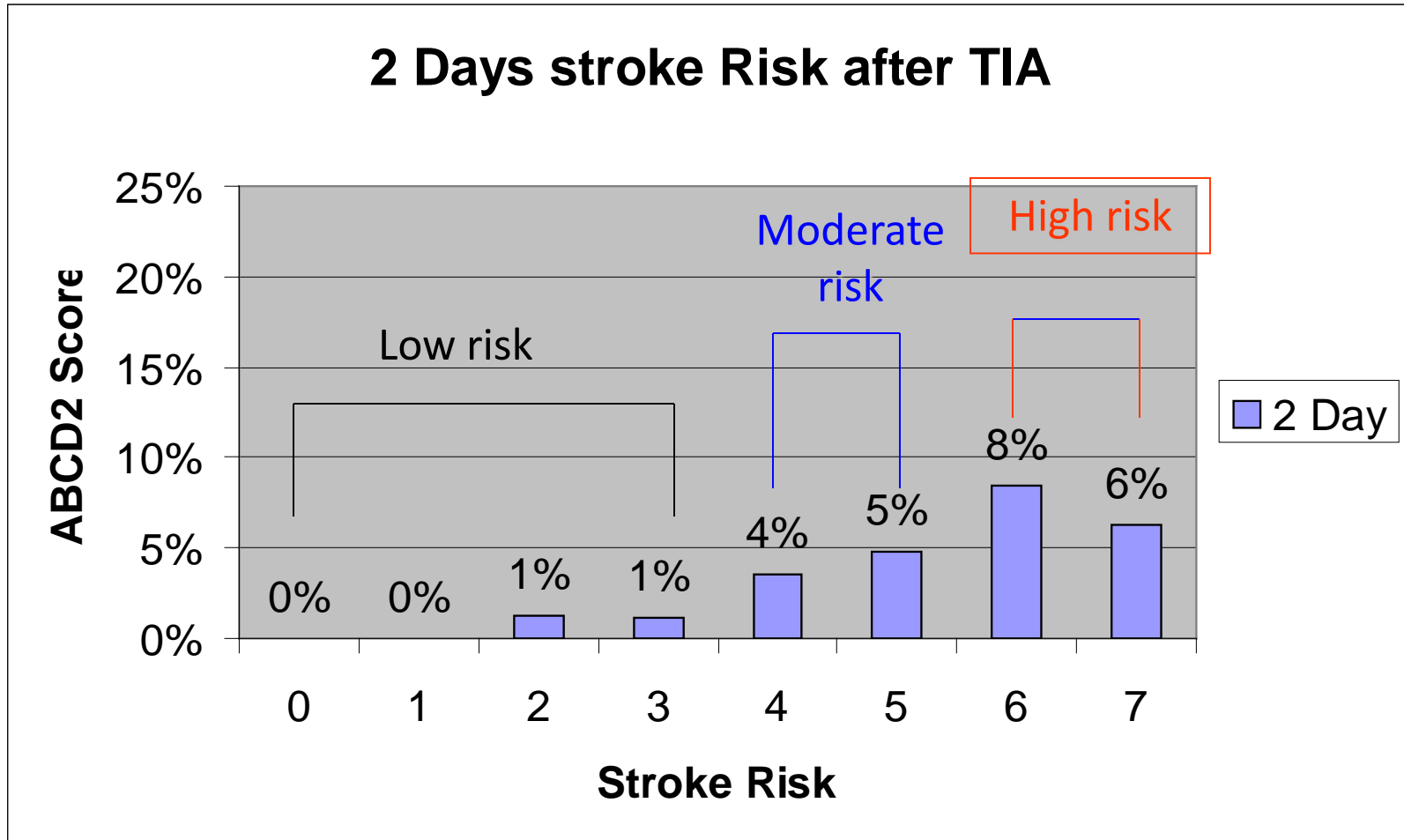
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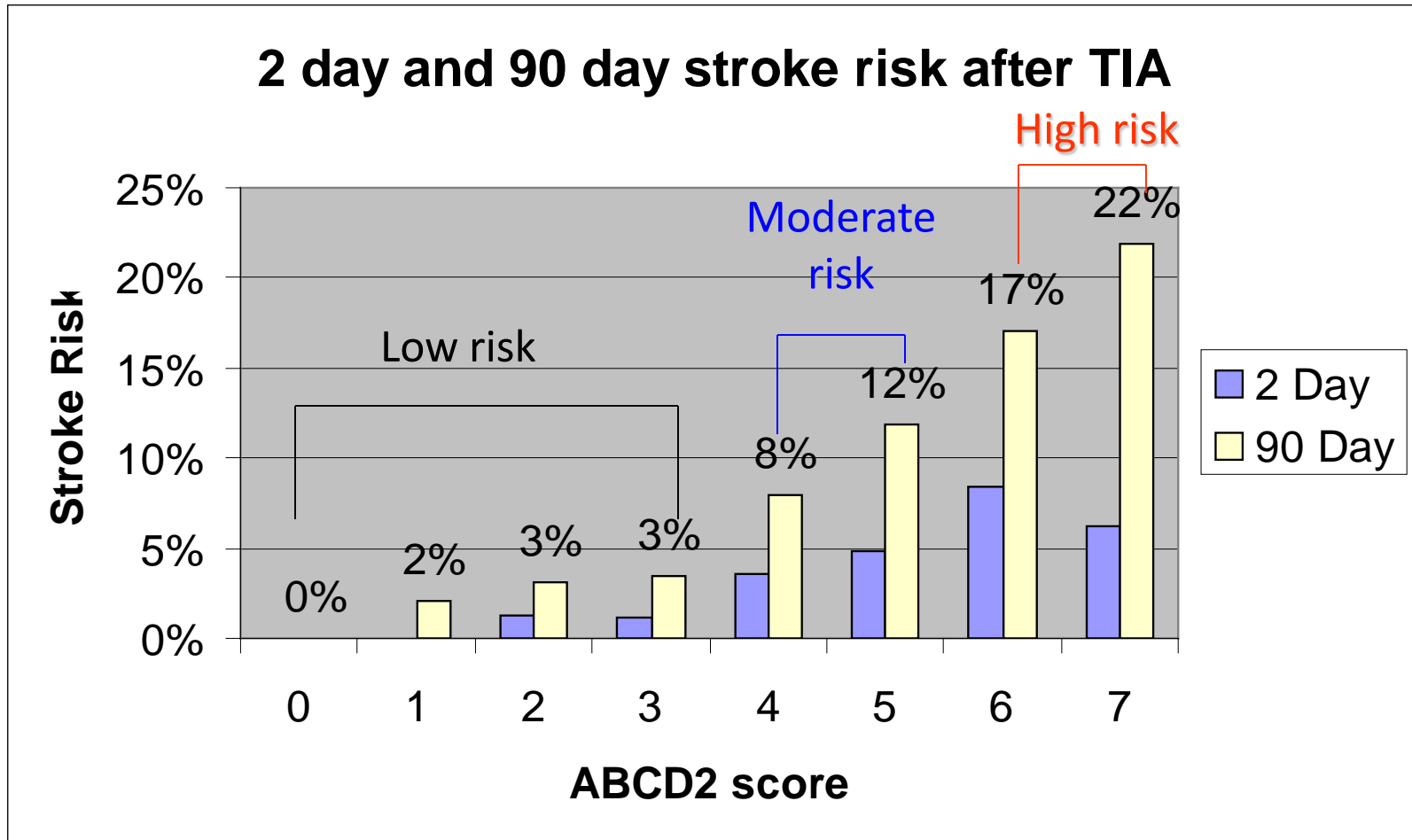
ABCD² score²

Factor, points	OR (95% CI)
Age (≥60yrs = 1 point)	1.4 (1.0-2.1)
BP (≥140 mmHg systolic or ≥90 diastolic → 1 point)	1.6 (1.1-2.2)
Clinical Features: .Unilateral weakness → 2 points .Speech impairment without weakness → 1 point	2.9 (2.0-4.3) <i>1.4 (0.8-2.3)</i>
Duration: ≥60 minutes → 2 points 10-59 minutes → 1 point	2.3 (1.3-4.0) 2.0 (1.0-3.7)
Diabetes (Presence → 1 point)	1.6 (1.1-2.2)

Risk stratification with ABCD² score



Risk stratification with ABCD² score



4799 patients

Case 1

- Who are the 0-8% of patients with TIA, who develop stroke in the first 2 days?

Case 1

- The patient's ABCD² score =4
 - 2-day risk of stroke is 4%
 - 90-day risk of stroke is 8%
- The mechanism of the TIA is probably known a-fib, while sub-therapeutic on warfarin

Case 1

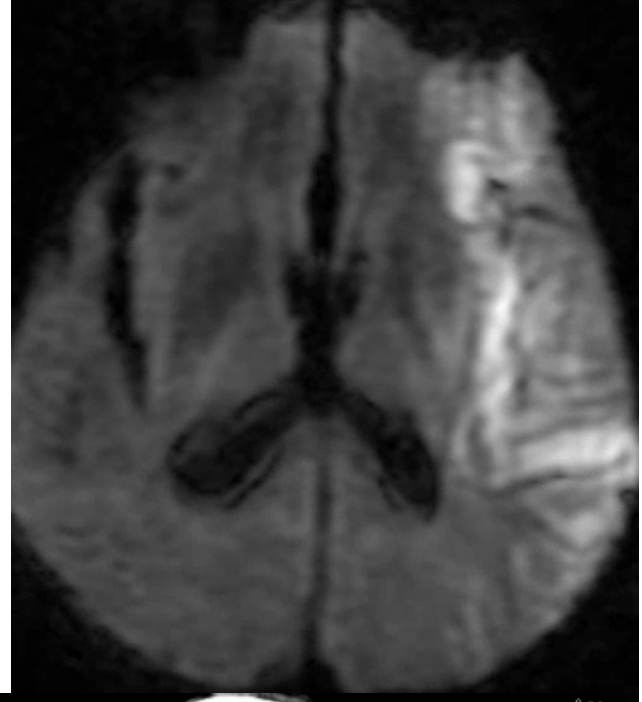
- We know the cause
- We have the treatment
- Our management options:
 - A) Obtain immediate vascular imaging in ED
 - B) Admit to hospital, observation, workup, anticoagulation
 - C) Discharge home with “bridge” anticoagulation
 - D) Discharge to home without bridge, but augment warfarin dose and recheck INR in 48hrs

Case 1

- *82yoM with Atrial Fibrillation (INR 1.6) and old right frontal lobe ischemic stroke.*
- *P/W sudden slurred speech, 19:45, totally resolved by 21:00 in the ED*
- *NIHSS=0 in the ED*
- *HCT unremarkable (except old infarct)*
- **Cr = 1.5, CTA deferred, pt admitted for MRA**

Case 1

- Admitted, MRA pending
- Discovered in room, aphasic, right hemiparetic
- NIHSS = 22



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280

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Case 1

- Despite successful technical revascularization of a left M2 occlusion, the patient's deficits did not improve.
- Based on prior medical problems, was already dependent (mRS 2), the family withdrew care and the patient expired.

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- *Despite successful technical revascularization, of a left M2 occlusion, the patient's deficits did not improve.*
- *Based on prior medical problems, was already dependent (mRS 2), the family withdrew care and the patient expired.*
- The patient went from no deficit to death.

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- **Is the ABCD score a tool we can use to triage the TIA patient?**

Case 1

- *The left M2 occlusion caused the large stroke*
- *Was the left M2 occlusion present initially when his NIHSS resolved to 0?*
- *The ABCD score predicted low risk of stroke, so why did this happen?*
- *Is the ABCD score a tool we can use to triage the TIA patient?*
- **What is the cause of stroke following TIA?**

What causes TIA in general?

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- Cardioembolism
 - Atrial fibrillation
 - Cardiomyopathy
 - Paradoxical
 - Septic
- Athero-thrombo/embolism
 - Arch
 - Extracranial ICA, VA
 - COW: MCA, iICA, BA
- Dissection
 - ICA, VA
- “Lacunar”
lipohyalinosis
 - Small vessel
- Cerebral Venous
Thrombosis

Hypothesis

- One feature that characterized Case 1 is the presence of “large vessel occlusion” (LVO)

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- *One feature that characterized Case 1 is the presence of “large vessel occlusion” (LVO)*
- Hypothesis: LVO is associated with increased risk of stroke following TIA, regardless of ABCD² score.

Coutts et al (2008)³

- Prospective cohort study
- 180 pts with TIA or minor stroke (NIHSS 0-3), followed for 90 days
- Intracranial Vessel Occlusion n=24 (13%)
- In the 24 with LVO, 46% had symptomatic clinical progression
 - Compared with 6% without LVO, RR 7.9 (3.7-17.1)
 - Presence of LVO **raised the risk of stroke 8-fold**

Coutts et al (2009)⁴

- Retrospective cohort, mild AIS or TIA, 2002-07
- Had CT + CTA i/e
- 297 pts identified, 90 day follow up
- Outcome “poor” mRS \geq 2 at 90d or d/c to other than home (rehab or nursing home)
- CTA: presence or absence of large vessel occlusion or severe (>50%) stenosis in either the circle of Willis (COW) or extracranial circulation

Coutts et al (2009)⁴

- In the poor outcome category, 27/57 (48%) had an LVO/SS
- COW + LVO/SS in 17/57(30%) of poor outcome vs 45/400(11%) of good outcome, $p=0.0006$
- Extracran + LVO/SS in 10/57 (18%) of poor outcome vs 35/400 (9%) of good outcome, $p=0.054$
- **The presence of LVO/SS is correlates with a poor outcome, especially if the lesion is in the Circle of Willis.**

Smith et al (2009)

- STOPStroke⁵, prospective cohort
- Stroke/TIA and CT/A
- 33 months, 735 enrolled, n=97 TIA
- Of the TIA cases, 13 (13%) had LVO
- Overall, +LVO associated with 4.5-fold increased odds of death (95%CI 2.7-7.3, $p < 0.001$) and absent LVO +3.2-fold increased odds of good outcome (95% CI 2.2-4.2, $p < 0.001$)
- They did not have enough TIA cases to study TIA specifically

Predictors of risk of early stroke

- Higher ABCD² score
- Presence of LVO/SS

Predictors of risk of early stroke

- *Higher ABCD² score*
- *Presence of LVO/SS*
- But, does high ABCD² = LVO ?

Predictors of risk of early stroke

- *Higher ABCD² score*
- *Presence of LVO/SS*
- But, does high ABCD² = LVO ?
 - There is little data
 - Arterial dissection
 - First presentation of atherosclerotic disease

Management Guidelines⁷

- Class 1 Evidence
 - Patients with TIA should preferably undergo neuroimaging evaluation within 24 hours of symptom onset.
 - Noninvasive imaging of the cervicocephalic vessels should be performed.
 - Patients with suspected TIA should be evaluated as soon as possible.

Management Guidelines⁷

- ECG should occur as soon as possible; prolonged cardiac monitoring is useful in patients with an unclear
- Echocardiography (at least TTE) is reasonable, especially in patients in whom no cause has been identified by other elements of the

Prevention Guidelines⁸

- Antiplatelet medication
- BP control
- HMG CoA reductase inhibitors
- Revascularization for symptomatic carotid stenosis
- Anticoagulation for afib

What's lacking

- Hospitalization
 - Admit, observation, or discharge
 - Selection
- Testing
 - Urgency of vascular imaging
 - Urgency of echo, rhythm analysis, blood work
- Ranta and Barber (2016)⁹

Model 1

- Admit all TIA cases to hospital or ED observation unit, with input from a stroke specialist.
- Pro: expert-driven, ability to observe and treat in case of worsening
- Con: clinical benefit unproven; no cost benefit

Model 2

- Sanders et al (2012)¹⁰
- ED assessment
 - Persistent signs, crescendo TIA, other medical issues: admission
 - Otherwise TIA clinic same or next day
- Vascular imaging: same or next day CDUS
 - Afib: anticoagulation, priority for TIA clinic
 - No afib, +CDUS ICAS>50%: CTA and priority to TIA clinic

Model 2

- 468 patients with 90 day follow up
- 150 control “admission method”
- Outcome = stroke at 90 days
- Stroke 1.50% (7/468) in Model 2, vs 4.67% (7/150) in “admission method”

Model 3

- SOS-TIA, Lavallee et al (2007)¹¹
- Specialized 24/7 clinic
- Not clear who saw the pts, but 24/7 vascular neurologist available at least by phone
- CDUS, TCD and ECG
- If high suspicion, then urgent TTE
- Direct communication bw VN and PCP re secondary prevention strategy
- Discharge to home

Model 3

- 643 pts had “definite TIA”
- Overall 1.24% (0.72-2.12) risk of stroke at 90 days, compared with estimated 5.96% based on ABCD² scores for this cohort

Model 4

- Rothwell et al (2007)¹², EXPRESS study
- Compared routine TIA outpatient referral (med 3 days, CDUS, TTE, no immediate initiation of meds) to same-day open-access clinic (M-F) with immediate initiation of meds.
- Reduction of 90-day stroke from 10.3% to 2.1%, HR 0.2., 0.08-0.49, $p = 0.0001$)

Model 5

- Olivot (2011)¹³ TWO ACES stud
- Prospective study, 224 pts that came to ED
- ABCD² = 0-3: sent from ED to TIA clinic (<48hr), MRA i/e or CTA i/e between ED and TIA clinic
- ABCD² = 4-5: CTA i/e
 - CTA i/e lesion >50% stenosis, admission
 - Otherwise, TIA clinic

Model 5

- Pts referred to TIA clinic had a stroke rate of 0.6% at 7 days (same at 90 d!), compared with expected 4.0, 7.1 % risk based on ABCD² score, $p < 0.034$, $p < 0.001$

Model 6

- Wasserman et al (2010)¹⁴
- ED visit, dx of TIA
 - Outpt CDUS, TTE, FLP
- $ABCD^2 \geq 6$: 7 day f/u in TIA clinic
- $ABCD^2 = 3-5$: 14 day f/u in TIA clinic
- $ABCD^2 < 4$: >14 day f/u

Model 6

- >1000 pts, outcome stroke at 90 days

ABCD2 score	Strokes	Predicted	P
<4 (n=321)	3, 0.9% (0-1.98)	3.1	0.0364
4-5 (n=469)	18, 3.8% (2.1-5.58)	9.8	<0.0001
≥ 6 (n=192)	10, 5.2% (2.07-8.35)	17.8	<0.0001
Combined	31, 3.2% (2.07-4.25)	9.1	<0.0001

Model 7

- Ranta et al (2015)15
- PCP with web-based decision tool triage to TIA clinic vs “usual care” by PCP
 - Web based tool
 - High risk ABCD2>3, 2 or more recent events, presence of afib → referral to TIA clinic 24 hrs
 - Low risk → TIA clinic within 7 days

Model 7

- 90-day stroke outcomes
- Intervention, 2/172 (1.2%)
- Control, 5/119 (4.2%)
- $P = 0.098$

Comparison

Model	Pro	Con
1	<ul style="list-style-type: none">-Full and immediate vascular imaging-Ability to intervene if patient worsens	<ul style="list-style-type: none">-Unproven clinical benefit, higher cost
2-7	<ul style="list-style-type: none">-Avoids most hospitalization, cost savings	<ul style="list-style-type: none">-There is clinical benefit when compared with the ABCD² risk, ie <u>compared with doing nothing.</u>

What is the goal?

- Cost savings?
- Reduction of stroke risk?

Hypothetical goals

- Reduction of cost, hospitalization
- Elimination (not reduction) of risk of stroke
 - This must include imaging of all cervicocranial vessels, knowing what we know about LVO and its strong correlation with poor outcome.

The UPMC-Mercy Model

- Brain and vascular imaging in ED
 - MRI brain, MRA head and neck
- If no infarct, and no LVO/SS, then observe 24h:
 - ABCD² >3, observe inpatient, complete workup
 - ABCD² <3, observe in “clinical decision unit” in the ED, then discharge home

Beyond Mercy

- If there is no afib and no LVO/SS, then
 - Antiplatelet
 - Address BP, glucose, LDL
 - Discharge home
 - Outpatient Holter
 - Follow up in Stroke clinic, next business day

References

1. Cerebrovasc Dis 2008;26: 630-635
2. Lancet 2007; 369:283-292
3. Stroke 2008; 39: 2461-2466
4. Intl. J. Stroke, 2009; 4: 448-453
5. Stroke 2009; 40: 3834-3840
6. Neurohospitalist 2011; 1(4): 187-199
7. Stroke 2009; 40: 2276-2293
8. Stroke 2014; 45: 2160-2236
9. Neurology 2016; 86: 947-953
10. Stroke 2012; 43: 2936-2941
11. Lancet Neurology 2007; 6:953-60
12. Lancet 2007;370:1432-42
13. Stroke 2011; 42:1839-1843
14. Stroke 2010; 41:2601-2605
15. Neurology 2015;84:1545-1551