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What to exclude when brain death is suspected

Pascale Grzonka, MD [a,*], Kai Tisljar, MD [a], Stephan Rüegg, MD [b,c], Stephan Marsch, MD, DPhil [a,c], Raoul Sutter, MD [a,b,c]

[a] Medical Intensive Care Units, University Hospital Basel, Switzerland
[b] Department of Neurology, University Hospital Basel, Switzerland
[c] Medical faculty, University of Basel, Switzerland

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ABSTRACT

Background: With advances in critical care and organ donation, diagnosis of brain death is gaining importance. We aimed to assess potential brain death confounders from the literature, elucidating clinical presentation and diagnostic approaches in these cases.
Methods: PubMed and Embase were screened using 37 predefined search terms to identify suitable articles reporting cases, case series, or cohort studies in adults.
Results: Out of 4769 articles, 40 case reports or case series describing 45 patients with 19 critical conditions were identified. Mortality was 11% and full recovery 33%. Intoxications (42%; mainly anti-seizure drugs and baclofen) and polyneuritis (37%) were most frequent. Brainstem reflex tests were reported in 96%, apnoea test in 16% and ancillary tests in all but one patient. Full recovery mainly occurred with intoxications. Quality of evidence regarding frequency of confounders is very low and risk of bias high.
Conclusions: Brain death confounders are infrequently reported and formal studies are lacking. Mainly younger patients with polyneuritis and intoxications are described. As outcome, especially in the latter, is often favourable, high awareness and strict adherence to guidelines is crucial. The importance of identifying pathologies compatible with extensive and irreversible brain damage before proceeding to diagnostic tests should be emphasized.

1. Introduction

Brain death is the complete and irreversible loss of brain function necessary to sustain life. Its diagnosis is based on the absence of brainstem reflexes, unresponsiveness to endo- or exogenous stimuli, apnoea, and the exclusion of critical conditions confounding the clinical presentation [1]. With advances in critical care and the emergence of organ donation programs worldwide, early and reliable diagnosis of brain death is gaining importance [www.irodat.org]. Despite this increasing demand, the predefined technical requirements as well as the qualification and number of clinicians to be involved in the diagnostic workup is inconsistent worldwide [2-4]. This issue is mirrored by an international review, revealing that conduct of apnoea tests, time to brain death diagnosis, number of examiners, and predefined clinical scenarios calling for ancillary tests varied among 80 countries [5]. This variability in combination with the lack of systematic studies regarding the frequency and presentation of specific and mostly reversible clinical conditions mimicking brain death further challenges clinicians and puts patients at risk. Hence, heightened awareness in this context is crucial.

The aim of this review was to assess potential conditions mimicking the clinical picture of brain death as reported in the literature, and to elucidate the clinical presentation and diagnostic approaches in these cases.

2. Methods

The predefined key outcomes were the frequency and types of reported conditions potentially acting as confounders in the clinical diagnosis of brain death in adult patients.

We searched the digital library search engines PubMed and Embase using the predefined search term “brain death” in combination with 37 search terms (“pitfalls”, “mimics”, “confounders”, “simulation”, “fake”, “resembling”, “errors in clinical diagnosis”, “misdiagnosis”, “false diagnosis [PubMed]”, “diagnostic problems”, “false positive diagnosis”, “false positive result”, “differential diagnosis”, “imitation”, “clinical criteria”, “clinical examination”, “brain stem reflexes”, “cranial nerves”, “corneal reflex”, “fixed pupils”, “pain”, “gag reflex”, “motor response”, “vestibulo-ocular reflex”, “cough reflex”, “hypothermia”,

* Corresponding author at: Intensive Care Units, University Hospital Basel, Petersgraben 4, 4031 Basel, Switzerland.
E-mail address: PascaleSusanne.Grzonka@usb.ch (P. Grzonka).

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"hypothyroidism", "hypoglycaemia", "intoxication", "alcohol intoxication", "electrolytes [PubMed]/electrolyte disturbance [Embase]", "locked in syndrome", "anaesthetics", "neuromuscular blockade", "hyperammonemia", "drugs", "drug abuse") to identify articles published between January 1960 and January 2018. Details regarding search terms as well as in- and exclusion processes are outlined in Supplementary Tables 1 and 2.

The respective MeSH and Emtree terms were used, if available. Articles written in English and reporting cases, case series, or cohort studies in adult humans were considered. All articles were elected by two independent researchers. Discrepancies regarding the eligibility were resolved by additional discussion to reach consensus.

Data regarding brainstem reflex tests, neuroimaging studies, evoked potentials, electroencephalograms (EEGs), and apnoea tests were assessed. In addition, short-term outcomes were compiled. Key data on study design, year of publication, and potentially confounding critical conditions were extracted using standardized data collection forms.

3. Results

From 4769 screened articles (Supplementary Tables 1 and 2), 40 were included with 45 patients described (Fig. 1). Mean age was 43 years (SD 14.3 years). Overall, 19 critical conditions potentially mimicking brain death were identified (Tables 1 and 2).

The frequency of reports regarding specific conditions acting as confounders in the clinical diagnosis of brain death in the literature is presented in Fig. 2. The two most frequent conditions were intoxications (mainly by anti-seizure drugs, baclofen, bupropion, and ethylene glycol) and polyneuritis, followed by infectious and autoimmune encephalitis. Examination of brainstem reflexes was reported in 96% and performance of apnoea tests in 16%, revealing absent spontaneous breathing in 86% of tested patients (Supplementary Table 3).

Ancillary tests were reported in all but one patient, consisting of 35 neuroimaging studies, 37 EEGs, and five studies regarding the integrity of evoked potentials. In the 35 patients in whom neuroimaging studies were performed, imaging was not compatible with brain death in 98%. EEGs were studied in 37 patients (82.2%) and excluded brain death in 93% of examined patients. Overall, in all but one patient with emerging brain oedema following rewarming from hypothermia and one case with incomplete data, reported tests including neuroimaging, evoked potentials and EEGs were not compatible with brain death.

Outcomes of patients with clinical confounders are outlined in Table 3. Mortality was 11% and full recovery was reported in 33%. Critical conditions with full recovery were intoxications (except ethylene glycol). In patients with Guillain-Barre syndrome, most patients survived with moderate to severe disability.

For additional potential confounders, such as electrolyte disorders, hypoglycaemia, hyperammonemia, intoxication with cerebral depressing/nerve blocking agents (others than identified), illicit drugs or "locked in" syndrome, no reports were identified.

4. Discussion

This review of the literature compiles and discusses etiologic, clinical, and neuro-functional characteristics of patients with critical conditions potentially confounding the clinical diagnosis of brain death. It provides detailed analyses of 40 case reports or small case series identified in the literature describing 45 patients with 19 confounders. The quality of evidence in this context is very low, the risk of bias high, and meta-analysis cannot be performed due to the lack of formal studies. However, analyses regarding individual outcomes revealed full recovery to be three times higher than overall mortality, emphasizing the importance of precise and accurate clinical workup to ensure a reliable diagnosis or exclusion of suspected brain death. The two entities

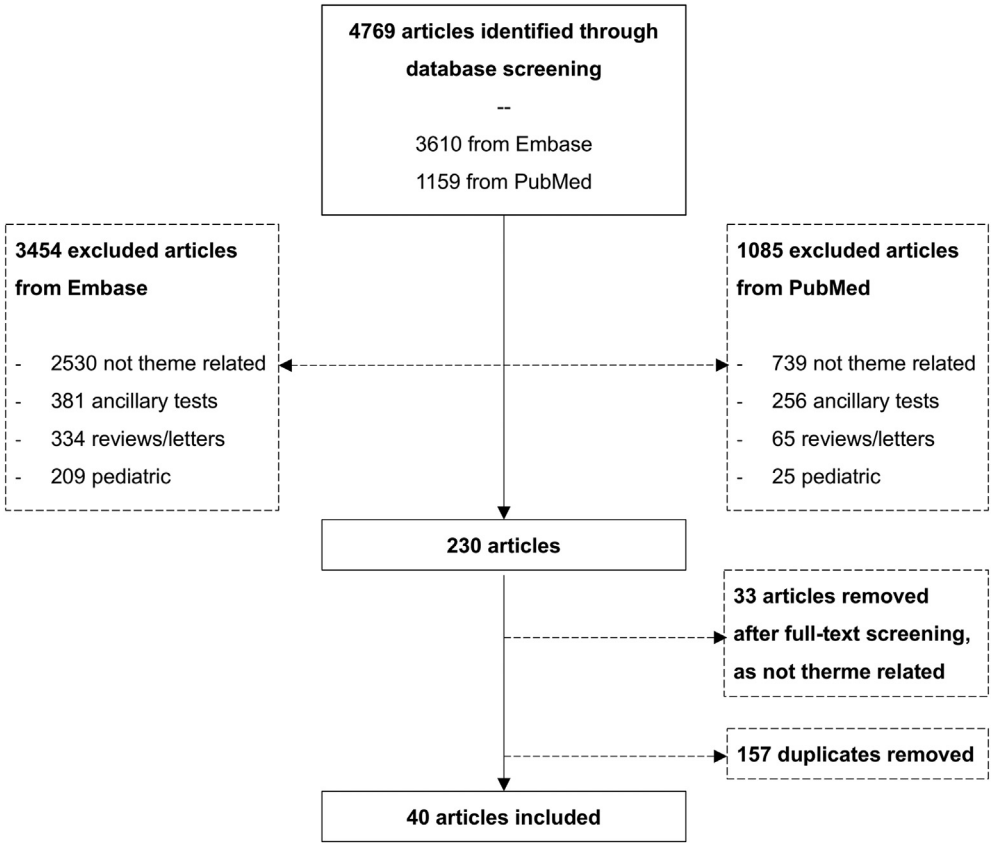

Fig. 1. Flow chart.

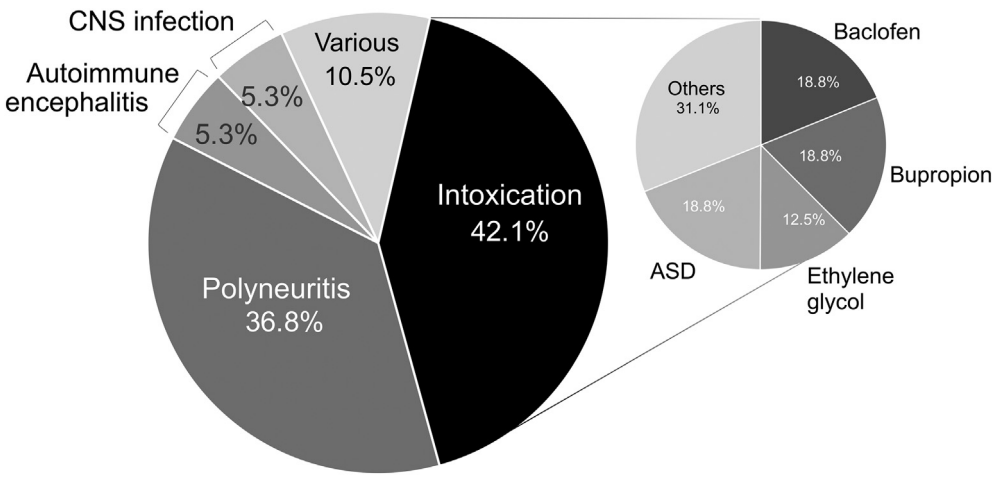

Fig. 2. Proportions of critical conditions mimicking brain death as reported in the literature. CNS = central nervous system; ASD = anti-seizure drugs.

most frequently reported to mimic brain death were acute intoxications and fulminant polyneuritis.

It is alarming that in the majority of case reports included in this review, tests for brain death diagnosis were initiated before the presence of a condition with the potential to cause irreversible brain damage had been established. This constitutes a violation of the most important rule in the process of diagnosing brain death and can lead to unjustified and potentially harmful procedures such as apnoea testing. The need to identify a pathology compatible with brain death before proceeding to further diagnostic tests should be emphasized in all international and institutional brain death protocols.

Table 1
Clinical and diagnostic information of cases reporting polyneuritis, brainstem encephalitis and intoxications mimicking brain death (further details available in Supplementary Table 3).

References	Years of publication	Number of patients; age	Brainstem reflexes	Apnoea tests	Ancillary tests
Polyneuritis (Guillain-Barre syndrome)					
[6-21]	1987 to 2016	18; mean age 47.3 (SD 14.5)	- 11 with absent reflexes - 7 with absent reflexes (but incomplete data)	- 2 without breathing - 16 n.r.	- 13 Imaging studies not compatible with brain death - 4 EPs with potentials, except 1 SSEP with absent brachial plexus potential - 17 EEGs with brain activity
Brainstem encephalitis					
[22,23]	1985 and 1991	4; mean age 37.5 (SD 16.1)	- 2 with absent reflexes - 1 with dilated but reactive pupils - 1 n.r.	- All n.r.	- 4 imaging studies not compatible with brain death - 2 EEGs with brain activity
Intoxications					
Baclofen					
[24-26]	2000 to 2017	4; mean age 48.0 (SD 8.8)	- 2 with absent reflexes - 1 with absent pupillary reflexes - 1 n.r.	- 1 with breathing - 3 n.r.	- 3 imaging studies not compatible with brain death - 3 EEGs: 2 with brain activity, 1 isoelectric
Bupropion					
[27-29]	2012 to 2018	3; mean age 41.0 (SD 10.4)	- All with absent reflexes	- All n.r.	- 3 imaging studies not compatible with brain death - 3 EEGs with brain activity
Ethylene glycol					
[30,31]	2002 and 2012	2; mean age 22.0 (SD 1.4)	- All with absent reflexes	- All n.r.	- All imaging studies not compatible with brain death - All EEGs with brain activity
Anti-seizure drugs					
Carbamazepine [32]	2017	1; age 54	- With absent reflexes	- N.r.	- Imaging not compatible with brain death - EEG with brain activity
Pentobarbital [33]	2015	1; age 40	- With absent reflexes	- N.r.	- EEG isoelectric
Valproic acid [34]	2009	1; age 19	- With absent reflexes	- N.r.	- Imaging not compatible with brain death
Other substances					
Amitriptyline [35]	1991	1; age 46	- With absent reflexes	- N.r.	- N.r.
Indian common krait envenomation [36]	2014	1; age 35	- With absent reflexes	- N.r.	- Imaging not compatible with brain death - EEG with brain activity
Lidocaine [37]	1998	1; age 60	- With absent reflexes	- N.r.	- Imaging not compatible with brain death
Organophosphate [38]	2008	1; age 28	- With absent reflexes, except constricted pupils	- N.r.	- Imaging not compatible with brain death - EEG with brain activity
Succinylcholine [39]	1974	1; age 39	- With absent reflexes	- N.r.	- EEG with brain activity

EEG = electroencephalogram; SSEP: somatosensory evoked potentials.
N.r. = not reported; SD = standard deviation.

Table 2
Clinical and diagnostic information of cases reporting infections and other less frequently reported conditions mimicking brain death (further details available in Supplementary Table 3).

References	Years of publication	Number of patients; age	Brainstem reflexes	Apnoea tests	Ancillary tests
Infections					
Botulism [40]	2017	1; age 43	- With absent reflexes	- N.r.	- Imaging not compatible with brain death - EEG with brain activity
CMV encephalitis [41]	2016	1; age19	- With absent reflexes	- Without breathing	- Imaging not compatible with brain death - EEG with brain activity
Various					
Diffuse leptomeningeal carcinomatosis [42]	2015	1; middle-aged	- With absent reflexes	- N.r.	- Imaging not compatible with brain death - EEG with brain activity
Hypothyroidism [43]	2003	1; age 62	- With absent reflexes	- Without breathing	- Imaging not compatible with brain death
Hypothermia [44]	2011	1; age 55	- With absent reflexes	- Without initial breathing, but breathing later on	- Subsequent exams after second breathing stop: - Imaging compatible with brain death - SSEP with absent evoked potentials - EEG isoelectric
Vasculitis of the central and peripheral nervous system [45]	2011	1; age 33	- With absent reflexes	- Without breathing	- Imaging not compatible with brain death - EEG with brain activity

CMV = cytomegalovirus; EEG = electroencephalogram; SSEP: somatosensory evoked potentials.
N.r. = not reported.

4.1. Intoxications and brain death

As intoxications were among the conditions most frequently diagnosed as an alternative to suspected brain death and associated with a high rate of full recovery, it is the opinion of the authors that screening for intoxications should be an integral component of all workup algorithms in this context.

The major toxins described were ethylene glycol, baclofen, bupropion, and anti-seizure drugs. There are, however, two major caveats to be mentioned in this regard: First, routine laboratory toxicological screening tests do not include ethylene glycol and its indirect detection mainly depends on the calculation of the osmotic gap; second, the term “drug screening” is a misnomer since it implies a comprehensive screening for all toxins and drugs, which is not the case. Often,

Table 3
Outcomes in patients with critical conditions mimicking brain death (further details available in Supplementary Table 3).

References	Years of publication	Number of patients; age	Outcomes
Polyneuritis (Guillain-Barre syndrome)			
[6-21]	1987 to 2016	18; mean age 47.3 (SD 14.5)	- 1 mild disability - 2 moderate disabilities - 9 severe disabilities - - 2 deaths
Brainstem encephalitis			
[22,23]	1985 and 1991	4; mean age 37.5 (SD 16.1)	- 1 full recovery - 1 moderate disability - 1 severe disability - 1 n.r.
Intoxications			
Baclofen			
[24-26]	2000 to 2017	4; mean age 48.0 (SD 8.8)	- All with full recovery
Bupropion			
[27-29]	2012 to 2018	3; mean age 41.0 (SD 10.4)	- All with full recovery
Ethylene glycol			
[30,31]	2002 and 2012	2; mean age 22.0 (SD 1.4)	- All with severe disabilities (incl. deafness)
Anti-seizure drugs			
Carbamazepine [32]	2017	1; age 54	- Full recovery
Pentobarbital [33]	2015	1; age 40	- Full recovery
Valproic acid [34]	2009	1; age 19	- Full recovery
Other substances			
Amitriptyline [35]	1991	1; age 46	- Full recovery
Indian common krait envenomation [36]	2014	1; age 35	- Full recovery
Lidocaine [37]	1998	1; age 60	- Mild disability
Organophosphate [38]	2008	1; age 28	- Full recovery
Succinylcholine [39]	1974	1; age 39	- Full recovery
Infections			
Botulism [40]	2017	1; age 43	- Severe disability
CMV encephalitis [41]	2016	1; age 19	- Full recovery
Various			
Diffuse leptomeningeal carcinomatosis [42]	2015	1; middle-aged	- Death
Hypothyroidism [43]	2003	1; age 62	- Death
Hypothermia [44]	2011	1; age 55	- Death
Vasculitis of the central and peripheral nervous system [45]	2011	1; age 33	- Mild disability

CMV = cytomegalovirus.
N.r. = not reported; SD = standard deviation.

current practice is to limit the screening to few substances, including ethanol, benzodiazepines, opioids, acetaminophen, salicylates, and other specific classes of therapeutic drugs [46]. In light of these limitations, it seems plausible that several toxins are not detected and underreported as mimics of brain death. Hence, clinicians are urged to explicitly search for further substances if routine toxicological screenings remain inconclusive.

4.2. Polyneuritis and brain death

Fulminant polyneuritis represented the second most frequently reported condition mimicking brain death. In contrast to the favourable outcomes with intoxications, recovery from polyneuritis was frequently linked to moderate or severe disability. To what degree the reports adhered to predefined disability scoring systems could not be assessed, leading to potential inconsistency in reporting individual outcomes. In addition, detailed information regarding long-term recovery was not available. Most cases described Guillain-Barre syndrome, an acute immune-mediated polyneuropathy, but some reports did not clearly determine the type of polyneuritis. At first glance, it may surprise that clinicians could misinterpret the clinical scenario of polyneuritis as brain death, since the history and clinical course of polyneuritis is usually distinct. However, two major scenarios should be taken into account: First, patients' histories may sometimes not be available at initial presentation; and second, admission can be late in the course of disease, so hypoxic-ischemic brain injury from respiratory failure may constitute an alternative aetiology, which is associated with a high mortality. Finally, it is again worrisome that in two cases apnoea tests were conducted even though neuroimaging was incompatible with brain death. It may be argued that apnoea tests were performed to further emphasize the confounding effect of polyneuritis in this context. However, it is the authors' opinion that in such cases putting patients at risk by apnoea testing, even for educative purposes, is inappropriate and should be avoided.

4.3. Further limitations

The critical conditions identified by this review are likely to represent the most frequent mimics while other less well known confounding scenarios may be underreported and/or under-recognized, such as with electrolyte disorders, hypoglycaemia, hyperammonemia, intoxication with cerebral depressing/nerve blocking agents (others than identified), illicit drugs or “locked in” syndrome. In addition, details regarding the diagnostic workup in individual cases were not always consequently reported and subjected to the quality of the reports. For example, most case reports included in this review did not outline the exact time point and chronological order of the performed tests. For example, prognostic work up in patients surviving cardiac arrest without complete return to premorbid neurofunctional baseline should not be performed before neuro-protective measures including controlled hypo- or isothermia is completed and patients are fully rewarmed. However, if and to what degree protocols were followed regarding timing and chronological order of prognostic tests could not be sufficiently analysed in the identified cases.

Another limitation is the restriction of included articles written in English. This review was not registered.

5. Conclusions

Critical conditions mimicking brain death are infrequently reported and formal studies in this context are lacking. Identified cases mainly describe younger patients with polyneuritis and intoxications. Favourable outcome in most patients with acute intoxication calls for high awareness and strict adherence to guidelines. The latter is crucial to avoid unnecessary and potentially harmful procedures, such as apnoea testing, and to prevent unjustifiable care withdrawal. The importance of identifying pathologies compatible with extensive and irreversible brain damage before proceeding to diagnostic tests should be emphasized. Studies are urgently warranted to uncover more potentially confounding conditions mimicking brain death, and to evaluate the optimal diagnostic procedures and adherence to established protocols.

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Not applicable.

Consent for publication

Not applicable.

Availability of data and material

All data generated or analysed during this study are included in this published article [and its supplementary information files].

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Authors' contributions

PG did the screening of the literature. PG and RS elected and analysed the articles and wrote the draft of the manuscript. All authors contributed significantly to the final version of the manuscript. It was read and approved by all authors.

Declaration of Competing Interest

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