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Mini Review Article

Cure of Amyotropic Sclerosis

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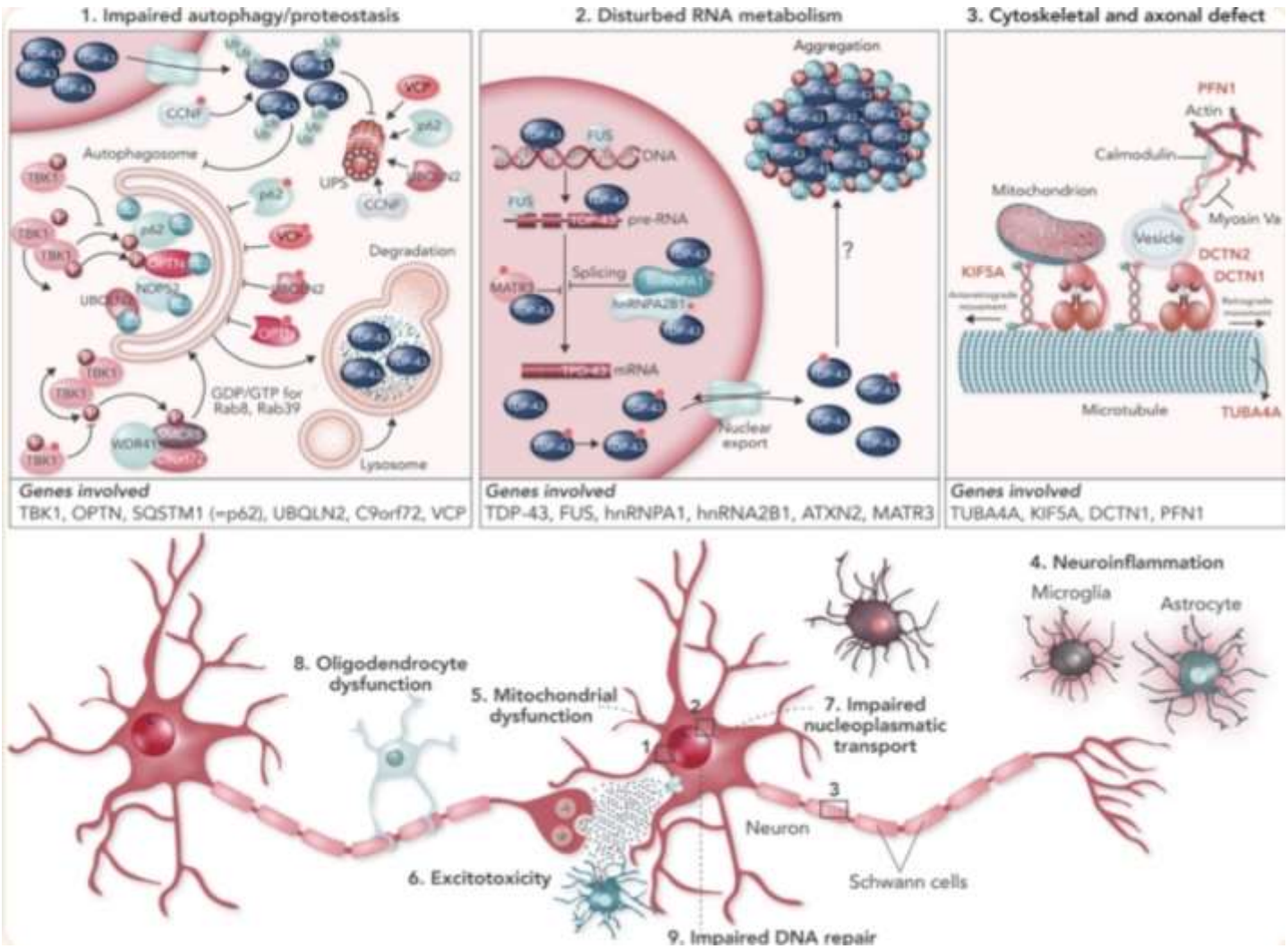
Abstract

It is neurodegenerative disorder affecting primary motor system but in which extra motor manifestation are increasingly recognized .ALS often has a focal onset but subsequently spreads to different body regions, where failure of respiratory muscles typically limits survival to 2–5 years after disease onset. In up to 50% of cases, there are extra-motor manifestations such as changes in behaviour, executive dysfunction and language problems. In 10%–15% of patients, these problems are severe enough to meet the clinical criteria of frontotemporal dementia (FTD).

Keywords: *Amyotrophic lateral sclerosis, sporadic and familial ALS, TDP-43 pathology.*

Aetiology

Figure 1



Grouping of ALS genes in pathologic pathways . (1) Mutations in TBK-1, OPTN, SQSTM1 (= p62), UBQLN2, C9orf72 and VCP affect the protein[1] degradation of pathways and may contribute to TDP-43 accumulation. (2) Mutations in TARDBP, FUS, MATR3, TIA1, hnRNPA1, hnRNA2B1 and[2] ATXN2 gene may all affect RNA metabolism. (3) Mutations in TUBA4A, PFN1, KIF5A and DCTN1 alteration of cytoskeletal dynamics and axonal transport.[3]

Pathogenesis

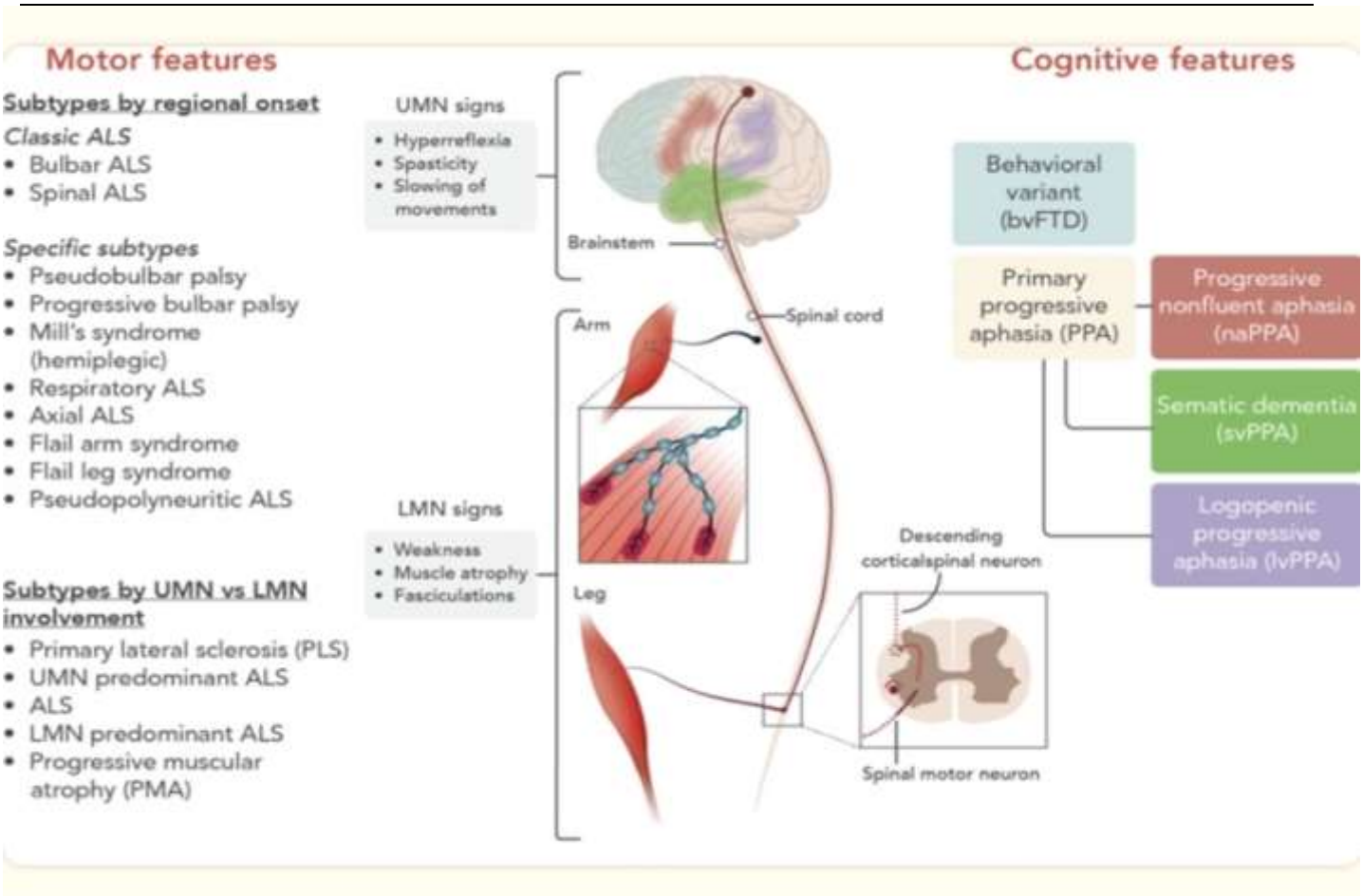
Neuropathological signof ALS is characterized by loss of the neuromuscular connection, axonal retraction and subsequent cell death of UMNs (upper motor neurons) and LMNs(lower motor neurons), surrounded by astrogliosis[4] and microgliosis, with ubiquitin-positive inclusions being observed in surviving neurons.[5] TDP-43 is main component of these inclusions in more than 95% of ALS[6] patients . TDP-43 is an RNA- and DNA-binding protein involved in multiple processes such as transcription, splicing, micro RNA maturation, RNA transport and stress granule formation. In[7] line with its nuclear and cytoplasmic functions, TDP-43 can shuttle between the nucleus and the cytoplasm, but its localization is mainly nuclear. Unlocalization to the[8] cytoplasm, leading to nuclear depletion of TDP-43 along with cytoplasmic protein aggregation,[9] is a hallmark of ALS.

Clinical presentation

Figure 2

It shows the phenotypic representation of the Amyotropic sclerosis [10]

Cure of Amyotropic sclerosis



Stemcell brain therapy

Implantation of the progenitors neurons lead to development of the neurons and affected neurons can be replaced by new neurons through stem cell brain therapy and lead to development and cure of the Amyotropic sclerosis

Dose 1) stem cell neuron progenitors cell injected through spinal needle through cervical puncture

Dose 2) Nutritional dose which gives nutrition to the cell

Dose 3) monoclonal antibodies against the deformed or degenerated neurons .

Discussion

1.) Aetiology of Amyotropic sclerosis

2.)clinical feature of Amyotropic sclerosis

3.) Cure of Amyotropic sclerosis

Conclusion

Cure of Amyotropic sclerosis is found and pathogenesis and aetiological studies of Amyotropic sclerosis

Reference

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