TREATMENT OF OPTIC NEURTIS

BASICS TO ADVANCED

TREATMENT

Typical cases which are idiopathic or proven to be due to demyelination are known to recover spontaneously, slowly over time, with restoration of normal vision, including the visual field, though some residual deficit in contrast sensitivity may remain in some cases.

OPTIC NEURITIS TREATMENT TRIAL(ONTT)

- 250 mg intravenously slowly over 30–60 minutes repeated 6 hourly for 3 days,
- followed by oral prednisolone 1 mg/kg/day for 11 days

OPTIC NEURITIS TREATMENT TRIAL(ONTT)

Steroid

Methyl Prednisolone

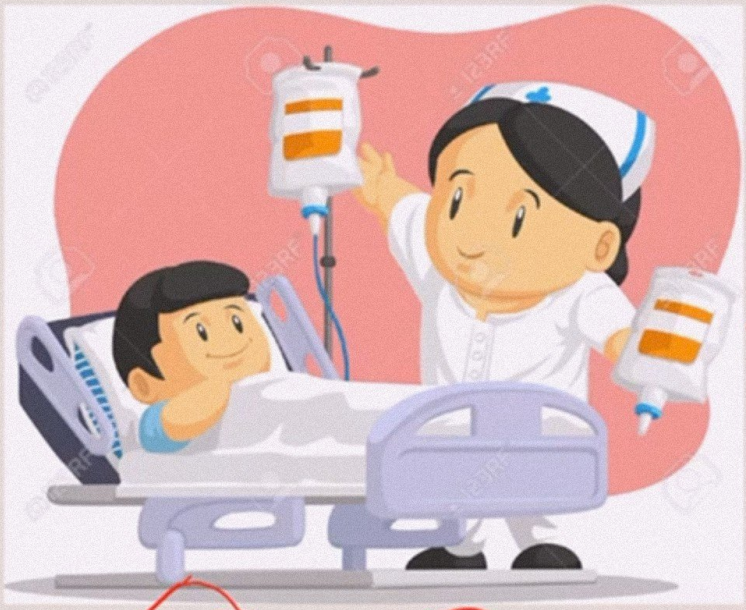
- **250 mg intravenously** slowly over **30–60 minutes** repeated 6 hourly for **3** days,
- followed by oral prednisolone **1 mg/kg/day** for **11 days**

1 GRAM in divided doses X 3 days

250mg IV	250 mg IV	250 mg IV	250 mg IV

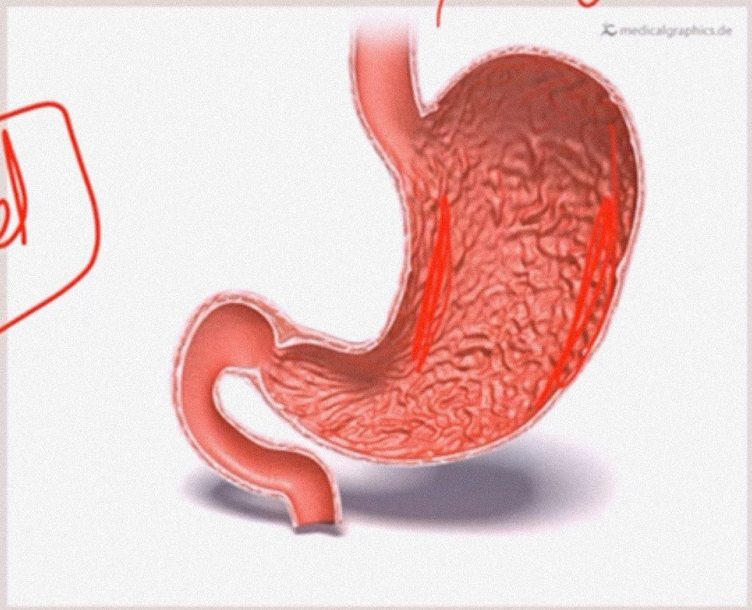
- hastens visual recovery and decreases the likelihood of recurrence, though the long-term visual outcome is no different from that achieved by observation alone, because spontaneous recovery occurs in the natural course in most cases.

- Oral prednisolone, in conventional doses of 1 mg/kg/day, should never be used alone as the recurrence rate has been found to be significantly higher following this regime.



Admit

gastritis

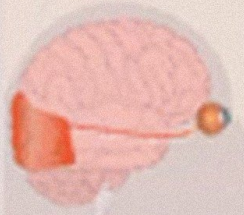


Antacid

BP ↓
ERRS
electrolytes

Is visual recovery in optic neuritis dependent on the timing of treatment?

Optic neuritis (ON): Optic nerve inflammation associated with multiple sclerosis (MS) and autoimmune conditions like neuromyelitis optica spectrum disorders (NMOSDs).



Aquaporin-4 (AQP4) antibody positive disease is a subtype of NMOSD.

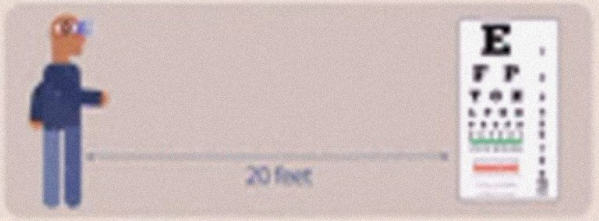
Treatment of ON in NMOSD and myelin oligodendrocyte glycoprotein (MOG) ab positive patients is typically steroid-responsive and time-dependent.

Study question:

Does the timing of intravenous methyl-prednisolone (IVMP) treatment affect visual outcomes in AQP4-IgG and MOG-IgG positive patients with ON?

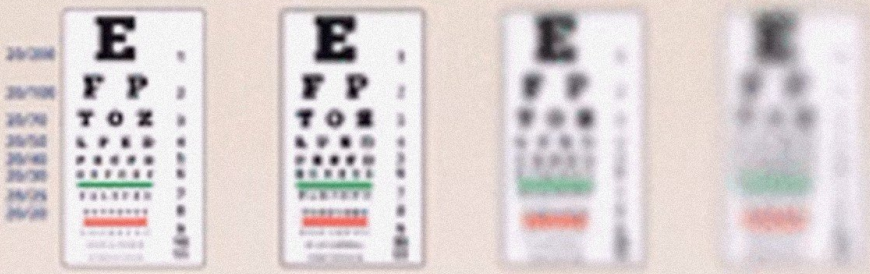
Patient cohort:
27 AQP4-seropositive NMOSD
9 MOG-IgG positive ON

Treatment:
1000 mg IVMP for 3–5 days, then oral prednisone



Best-corrected visual acuity (BCVA) at 3 months.

Patients with AQP4- and MOG-positive ON responded **better** to earlier IVMP treatment.



Patients treated later than **4 days** had an odds ratio of 8.33 of failure to regain **20/20 vision.**

Patients treated later than **7 days** had odds ratio of 10.0 of failure to regain **20/30 vision.**

Early treatment is essential for long-term visual recovery in this group of patients.

Even a 7-day delay in IVMP initiation was detri[illegible]ental to the vision of patients with AQP4 and MOG[illegible]N.

doi:10.1212/NXI.0000000000000572

Neurology
Neuroimmunology

MOG –Ab associated optic neuritis-→ MORE RESPONSIVE TO THE STEROIDS with good recovery

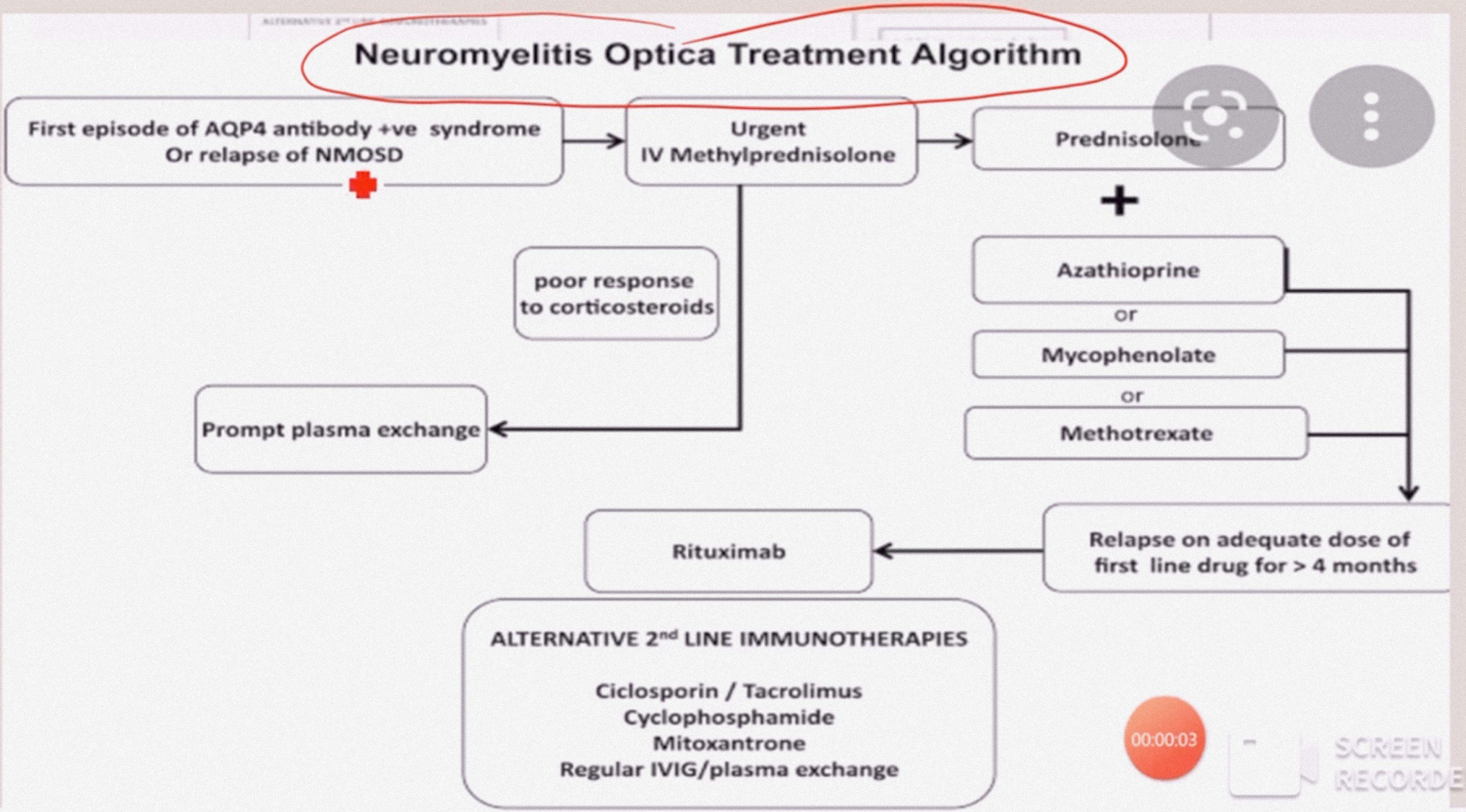
BUT

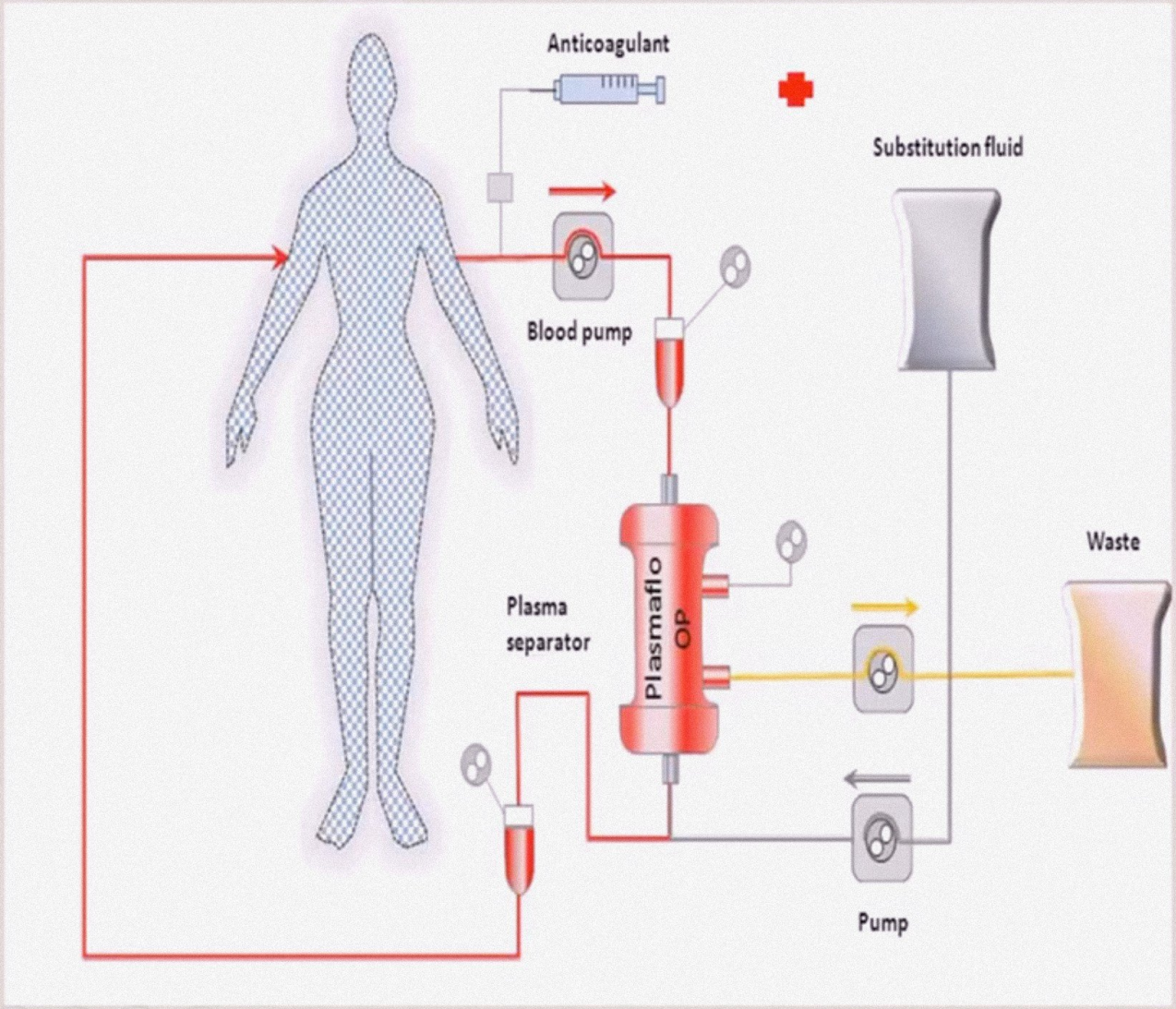
More relapses

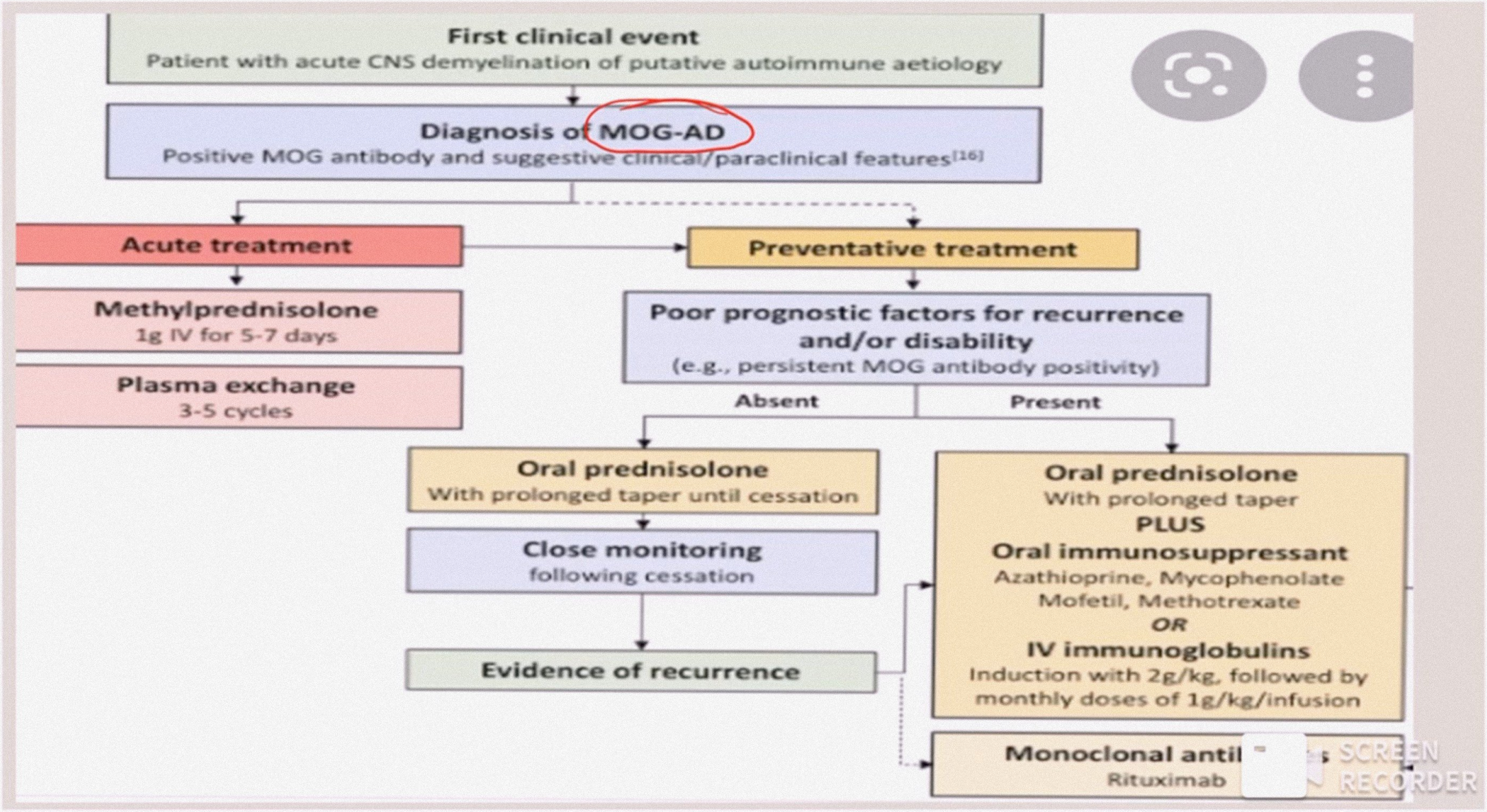
ROLE OF LIFELONG IMMUNOSUPPRESSION

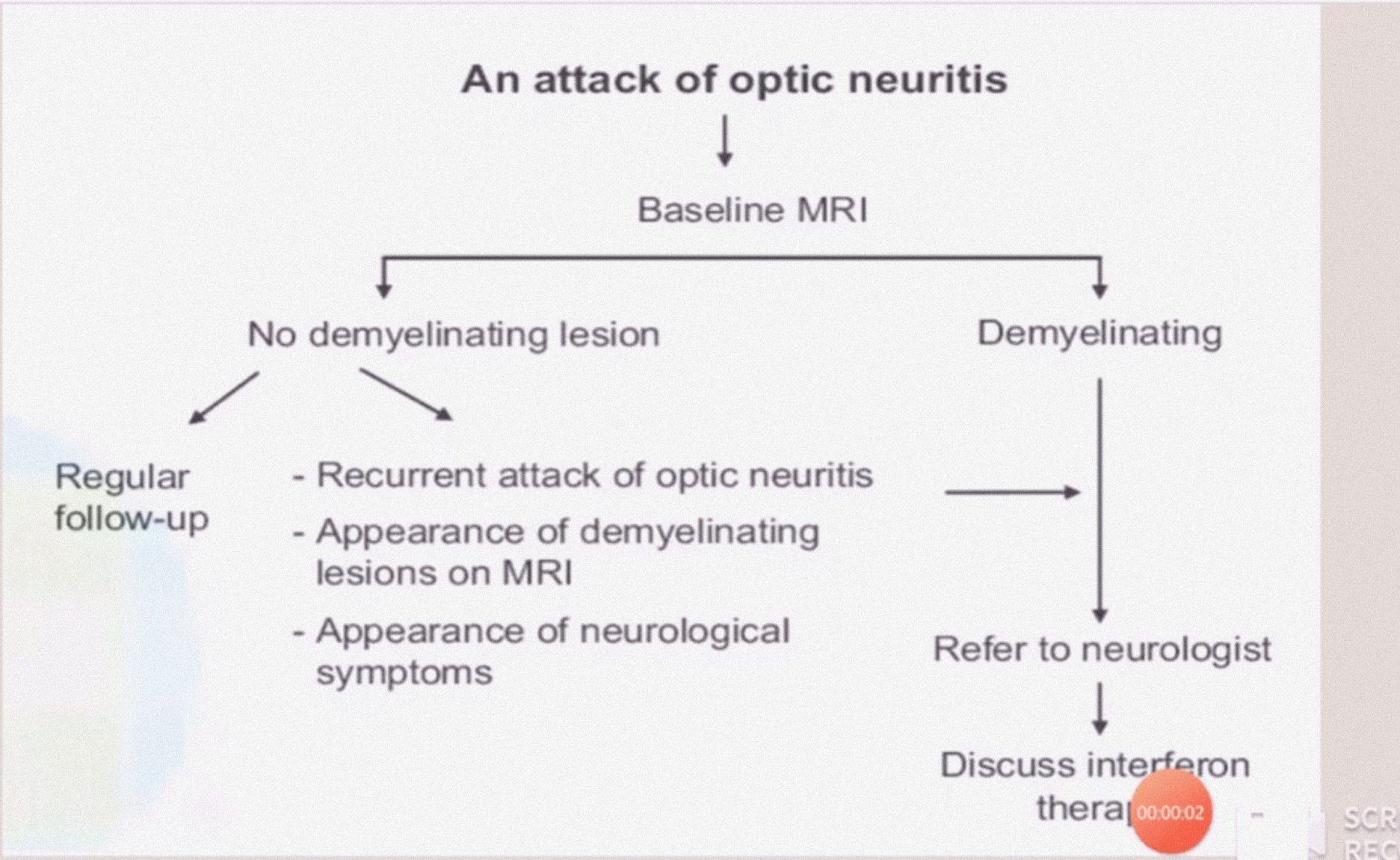
AQP4 –Ab has worse prognosis and is MINIMALLY RESPONSIVE TO STEROIDS

PLASMA EXCHANGE
LONG TERM IMMUNOSUPRESSION (rituximab, Azathioprine


Neuromyelitis Optica Treatment Algorithm
First episode of AQP4 antibody +ve syndrome
Or relapse of NMOSD
Urgent
IV Methylprednisolone
Prednisolone
+
poor response
to corticosteroids
Azathioprine
or
Mycophenolate
or
Methotrexate
Prompt plasma exchange
Relapse on adequate dose of
first line drug for > 4 months
Rituximab
ALTERNATIVE 2nd LINE IMMUNOTHERAPIES
Ciclosporin / Tacrolimus
Cyclophosphamide
Mitoxantrone
Regular IVIG/plasma exchange
00:00:03
SCREEN
RECORDER


Anticoagulant
Substitution fluid
Blood pump
Plasma
separator
Plasmaflo
OP
Waste
Pump


First clinical event
Patient with acute CNS demyelination of putative autoimmune aetiology
Diagnosis of MOG-AD
Positive MOG antibody and suggestive clinical/paraclinical features[16]
Acute treatment
Methylprednisolone
1g IV for 5-7 days
Plasma exchange
3-5 cycles
Preventative treatment
Poor prognostic factors for recurrence and/or disability
(e.g., persistent MOG antibody positivity)
Absent
Present
Oral prednisolone
With prolonged taper until cessation
Close monitoring
following cessation
Evidence of recurrence
Oral prednisolone
With prolonged taper
PLUS
Oral immunosuppressant
Azathioprine, Mycophenolate Mofetil, Methotrexate
OR
IV immunoglobulins
Induction with 2g/kg, followed by monthly doses of 1g/kg/infusion
Monoclonal anti
Rituximab


An attack of optic neuritis
Baseline MRI
No demyelinating lesion
Demyelinating
Regular follow-up
- Recurrent attack of optic neuritis
- Appearance of demyelinating lesions on MRI
- Appearance of neurological symptoms
Refer to neurologist
Discuss interferon
00:00:02
SCREEN RECORDER

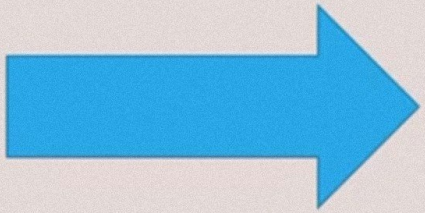
INTERFERON BETA

IFNβ/α

- Cytokine in the interferon family produced by mammalian cells .
- It balances the expression of the proinflammatory and anti-inflammatory agents in brain and reduces the number of inflammatory cells that cross the blood brain barrier
- Reduces the population of T helper cells 17 population

CHAMPS STUDY

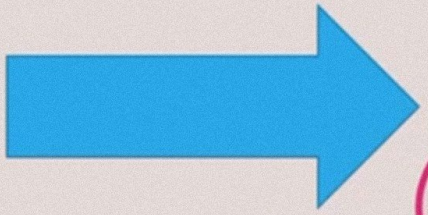
30 micrograms of interferon a1B



Cumulative probability of developing MS was 35 % vs 50 %

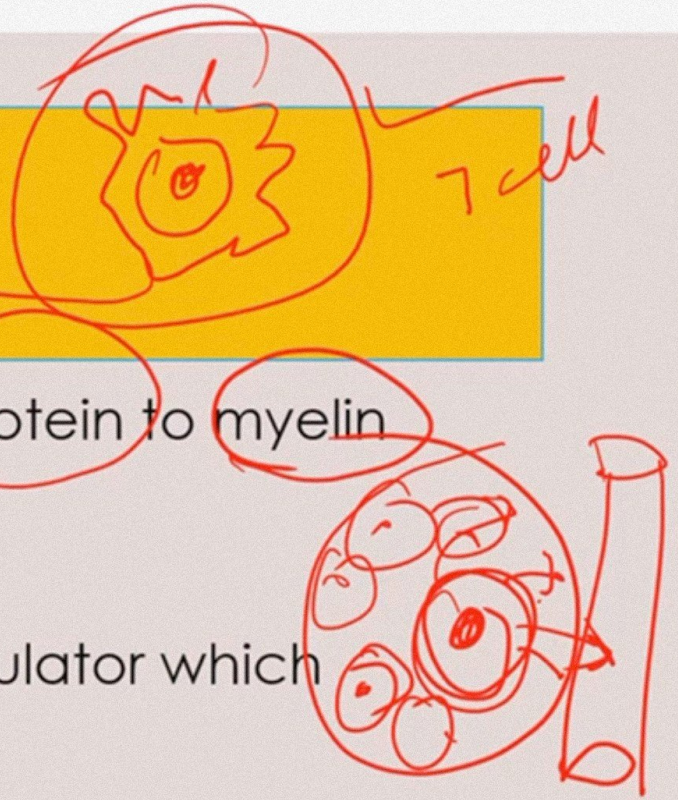
CHAMPIONS STUDY

EARLY TREATMENT
WITH interferon a1B



Fewer relapses
Fewer conversion to
MS

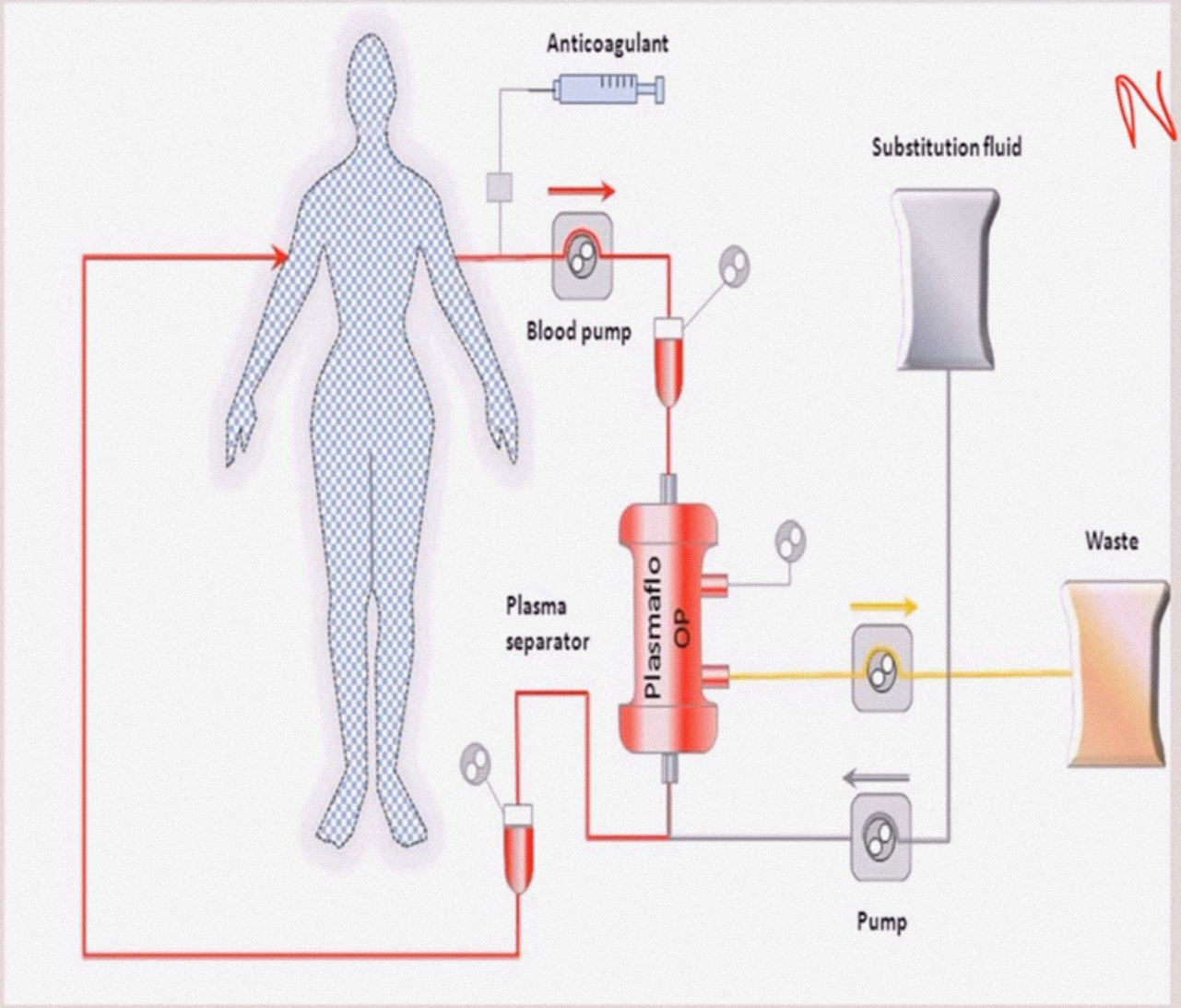
OTHER NEWS DRUGS



- **GLATIRAMER ACETATE (COPAXONE):** decoy protein to myelin basic protein
- **FINGOLIMOD :-** sphingosine 1 phosphate modulator which sequesters lymphocytes in lymph nodes
- **NATALIZUMAB (Tysabri):** Humanised monoclonal antibody against the cell adhesion molecule alpha 4 integrin

Table 4. Drugs Used in the Management of ON

Drug	Indication	Adverse Effects
Methylprednisolone	Typical and atypical ON	Mood changes, insomnia, dyspepsia, facial flushing, weight gain, hyperglycemia, osteoporosis, hypertension
Interferon beta 1a Interferon beta 1b	Prevent conversion of ON to MS in high-risk patients as determined by MRI	Flulike symptoms, injection-site reactions, headache, depression, suicidal ideation, severe hepatic injury (rare)
Glatiramer acetate	Prevent conversion of ON to MS in high-risk patients as determined by MRI	Increased risk of infection, injection-site reactions, cancer, nausea, hypertension
Azathioprine	1st-line NMO	Hepatotoxicity, severe leukopenia, nausea, vomiting
Rituximab	1st-line NMO	Fever, chills, hypotension, lymphopenia
Methotrexate	2nd-line NMO	GI toxicity, hepatotoxicity, leukoencephalopathy
Mitoxantrone	2nd-line NMO	Infection, hematologic toxicity, cardiotoxicity, GI adverse events, alopecia, injection-site reactions
Mycophenolate mofetil	2nd-line NMO	GI, respiratory, and dermatologic adverse events; infection; urotoxicity; hematologic toxicity; cardiotoxicity
Tocilizumab	3rd-line NMO	GI toxicity, serious infections, neutr[illegible]ia, thrombocytopenia, elevated liver enzymes, lipid abno[illegible]es, hypersensitivity reactions



NMO
Ab.