

ONCOLOGY KNOWLEDGE HUB · TEACHING SLIDES

Brain Tumours

A mini review for clinicians

For postgraduate residents, medical students and practising clinicians

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Learning objectives

- Recognise how brain tumours present in adults and children, and the traps that delay diagnosis
- Work up a brain mass: MRI, search for a primary, tissue and molecular testing
- Name diffuse gliomas by the WHO 2021 integrated diagnosis
- Plan surgery, radiotherapy and systemic therapy for glioma (no doses)
- Choose local therapy for brain metastases
- Manage steroids, seizures and thrombosis safely; plan follow-up

Roadmap

- 1 Burden and groups
- 2 Presentation and traps
- 3 Work-up and imaging
- 4 Molecular classification
- 5 Glioblastoma
- 6 IDH-mutant gliomas
- 7 Brain metastases
- 8 Children
- 9 Supportive care and follow-up
- 10 Evidence and cases

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Burden and groups

Brain tumours in India

- About 32,000 new malignant brain/CNS tumours and 28,000 deaths a year (GLOBOCAN 2022)
- Benign meningiomas and most childhood low-grade gliomas are not counted
- Metastases outnumber primary tumours in adults over 40
- Glioblastoma: commonest malignant primary; median age ~64
- Children: a leading cause of cancer death

Three working groups

Adults (all intracranial tumours): metastases outnumber primary tumours



Children (primary CNS tumours, the commonest solid cancers of childhood)



Adult diffuse glioma

- Defined by IDH, 1p/19q and other markers
- Glioblastoma: commonest malignant primary
- Seizure, focal deficit, raised pressure

Brain metastases

- Lung, breast, melanoma, kidney, GI
- Often found by staging MRI
- SRS and CNS-active drugs changed outcomes

Children

- Half in the posterior fossa
- Vomiting, ataxia, squint, head tilt
- Many cured; late effects matter

Adults: mostly metastases; children: low-grade glioma and medulloblastoma

Risk factors

Established

- Ionising radiation to the head
- NF1, NF2, tuberous sclerosis
- Li-Fraumeni, von Hippel-Lindau, Gorlin
- Lynch and constitutional MMR deficiency
- Age; male sex for glioblastoma

Not established

- Mobile phones
- Power lines
- Head injury
- Stress or studying
- Diet

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Presentation and traps

How brain tumours present

Feature	How often	Key point
Headache	~20% at presentation	Rarely alone; classic triad in ~5%
Seizure	50–80% of primary tumours	Commonest in low-grade, IDH-mutant
Focal deficit	Common in HGG, metastases	Steroid-responsive = oedema
Cognitive change	Frontal, callosal tumours	Mistaken for depression
Raised pressure	Posterior fossa, large tumours	Worse lying down, vomiting

When to scan

Image the brain (MRI with contrast) when

- New headache over 50, or a changed headache pattern
- Headache with vomiting, worse lying down, waking from sleep
- Headache with focal signs, confusion or papilloedema
- First seizure in an adult (not provoked)
- Progressive weakness, speech, vision or personality change
- Any neurological symptom in a person with known cancer
- Child: morning vomiting, ataxia, new squint, head tilt, regression

Common traps in Indian practice

- Ring lesion called tuberculoma or NCC without the full picture
- Steroids started before biopsy when lymphoma is possible
- Seizure treated for years with no scan
- Morning vomiting in a child treated as acidity or worms
- Normal plain CT taken as the end of the work-up
- Confusion in a cancer patient blamed on “weakness”

Isolated headache with a normal neurological examination is rarely a tumour (about 1.5% of headache MRIs); only a small minority of brain tumours present with headache alone.

Red flags are the pattern, the age and the company the headache keeps

A ring is not a diagnosis

Lesion	Imaging and clinical clue	Next step
Metastasis	Grey–white junction, often multiple, much oedema; known or occult primary	CT chest–abdomen–pelvis; biopsy the easiest site
Glioblastoma	Thick irregular rim, necrosis, crosses corpus callosum	Resection or biopsy; IDH, MGMT
Primary CNS lymphoma	Periventricular, homogeneous, restricted diffusion	NO steroids before biopsy; HIV test; slit–lamp
Pyogenic abscess	Thin smooth rim; centre restricts on diffusion	Source, cultures, drainage
Tuberculoma	Conglomerate rings, basal meningitis, chest TB	Tissue or CSF proof where possible
Neurocysticercosis	Cyst with scolex (dot), often several, seizures	Serology, CT calcifications; usually small
Tumefactive demyelination	Open (incomplete) ring, little mass effect	MRI spine, CSF; biopsy if unsure

Diffusion MRI, the setting and tissue sort metastasis, glioma, lymphoma, abscess, TB, NCC

Indian traps

- Seizures treated for years without a scan
- Ring lesion called tuberculoma or NCC without proof
- Steroids before biopsy when lymphoma is possible
- Morning vomiting in a child treated as acidity or worms
- Normal plain CT taken as the end of the work-up

Primary CNS lymphoma: protect the biopsy

- Periventricular, homogeneous, restricted diffusion
- Steroids can melt it and spoil the biopsy
- If stable: withhold steroids, stereotactic biopsy
- HIV test, slit-lamp, CSF, body imaging
- Treated with methotrexate-based chemotherapy, not resection

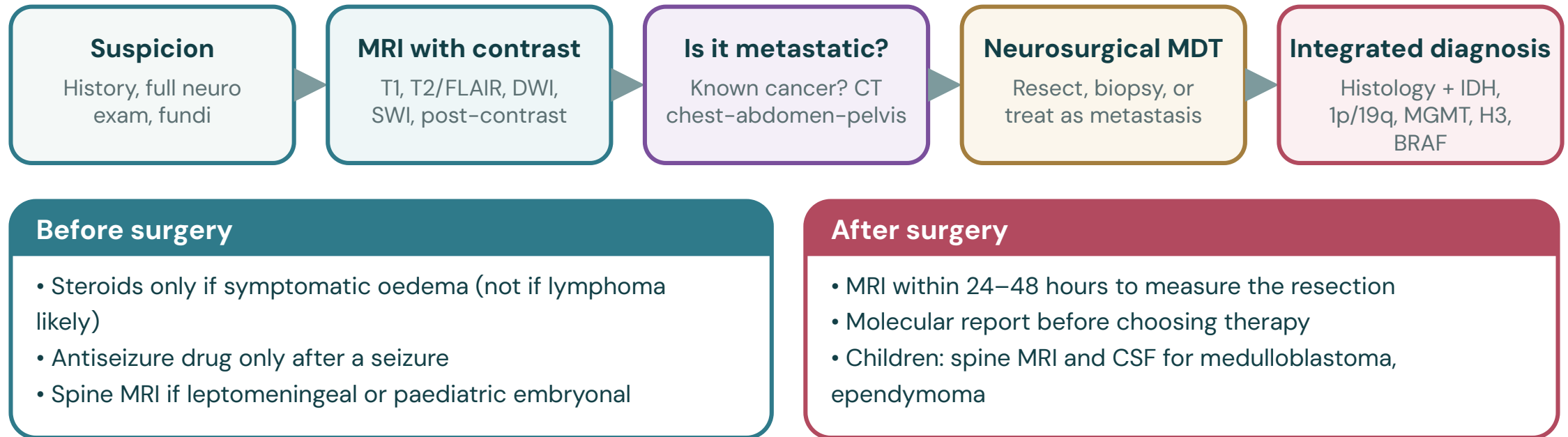
Other common adult tumours

- Meningioma: dural-based, often incidental; observe small asymptomatic ones
- Vestibular schwannoma: hearing loss, tinnitus first
- Pituitary tumours: vision and hormones; headache common
- Colloid cyst: positional, paroxysmal headache; can be fatal
- Ganglioglioma: young, temporal, epilepsy; BRAF V600E in up to 60%

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Work-up and imaging

From suspicion to diagnosis



MRI, search for a primary when relevant, MDT, tissue with molecular tests

MRI clues

Finding	Suggests
Non-enhancing T2/FLAIR mass	Lower-grade glioma
T2–FLAIR mismatch	IDH-mutant astrocytoma
Calcification, cortical, frontal	Oligodendroglioma
Thick ring, necrosis, crosses callosum	Glioblastoma
Multiple grey–white junction lesions	Metastases
Periventricular, restricts diffusion	Lymphoma (no steroids)

What the neurosurgeon offers

Maximal safe resection

- Goal for accessible gliomas
- Gross total → longer survival
- Awake mapping for eloquent areas
- MRI in 24–48 h to confirm

Stereotactic biopsy

- Deep, multifocal or eloquent
- Lymphoma suspected
- Risk: sampling error, undergrading
- Enough tissue for molecular tests

Shunt / ETV / resection

- Obstructive hydrocephalus
- Posterior fossa tumours in children
- Large metastasis with mass effect
- Diagnostic doubt about a “metastasis”

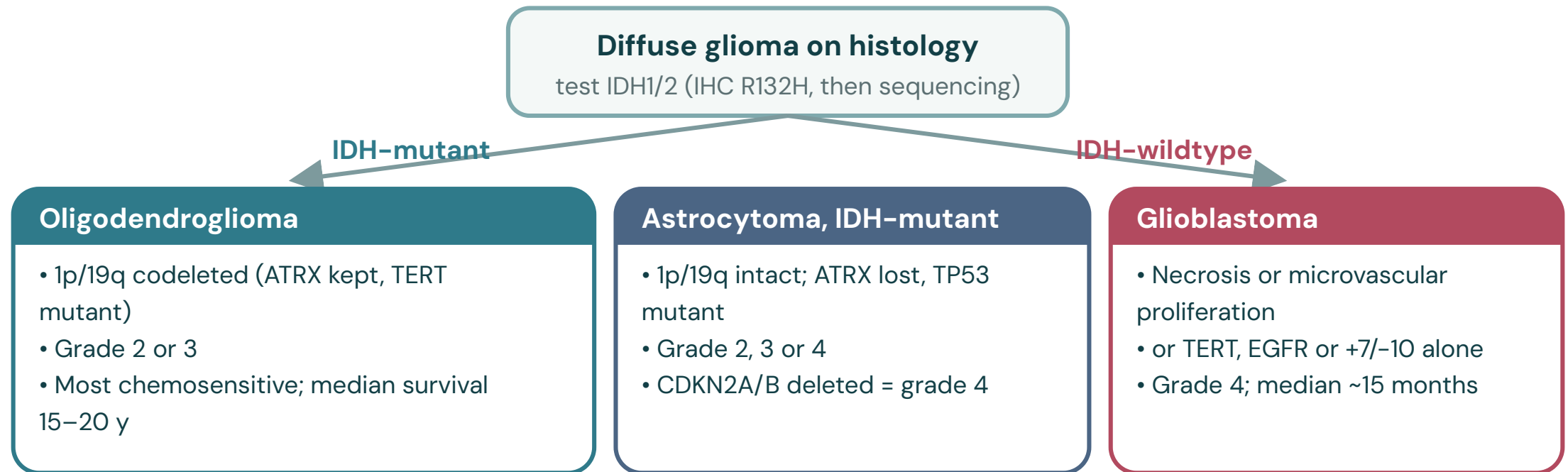
Patchell 1990: in single metastases, 11% turned out not to be metastases at surgery. Tissue is worth having when the story does not fit.

Resection for accessible gliomas; biopsy for deep or lymphoma-like lesions

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Molecular classification

WHO 2021: naming a diffuse glioma



Midline glioma in a child or young adult: test H3 K27 (diffuse midline glioma, grade 4).

Hemispheric high-grade glioma in teenagers and young adults: test H3 G34.

Also test MGMT promoter methylation (glioblastoma: prognostic and predictive) and BRAF V600E where relevant.

IDH, 1p/19q, CDKN2A/B, TERT/EGFR/+7-10, H3

Tests that change treatment

IDH1/2 mutation

Separates astrocytoma/oligodendroglioma from glioblastoma; better prognosis; vorasidenib for residual grade 2

1p/19q codeletion

Defines oligodendroglioma; RT plus PCV or temozolomide; longest survival

MGMT methylation

Glioblastoma: predicts temozolomide benefit; guides treatment in older adults

CDKN2A/B deletion

IDH-mutant astrocytoma becomes grade 4 regardless of histology

TERT, EGFR, +7/-10

IDH-wildtype astrocytoma = glioblastoma even without necrosis

BRAF V600E

Dabrafenib + trametinib (children: first line in LGG; adults: recurrent)

H3 K27 alteration

Diffuse midline glioma, grade 4; dordaviprone at recurrence

MMR deficiency / hypermutation

Germline CMMRD or Lynch; checkpoint inhibitors in selected cases

NTRK or other fusions

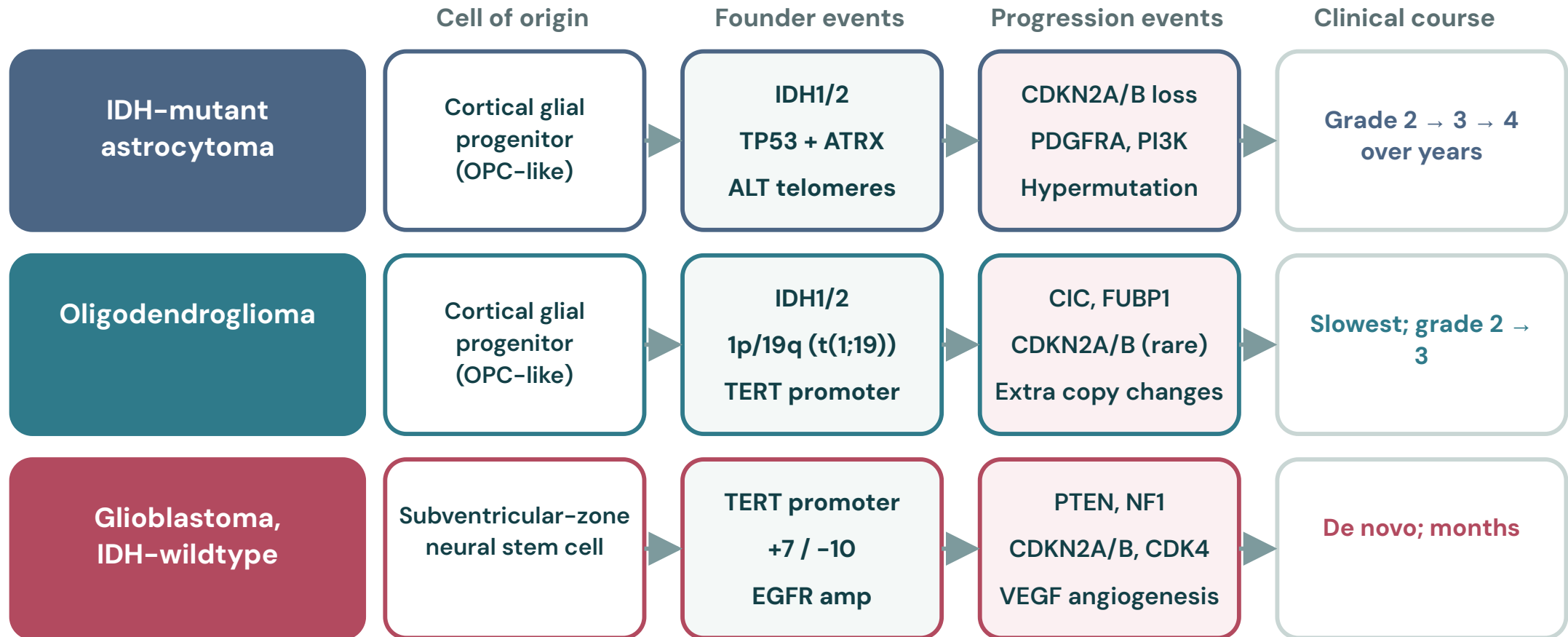
Tumour-agnostic TRK inhibitors (larotrectinib, entrectinib)

Ask for an integrated diagnosis on every glioma

Molecular rules to remember

- IDH IHC negative under 55 or grade 2–3 → sequence IDH1/2
- 1p/19q must be whole-arm (NGS, methylation, LOH)
- IDH-wildtype + TERT/EGFR/+7-10 = glioblastoma
- IDH-mutant + CDKN2A/B deletion = grade 4
- MGMT methylated in 40–50% of glioblastomas
- Midline glioma in the young: test H3 K27

Three routes, three diseases



IDH mutation is the first (truncal) event in both IDH-mutant routes and is never lost; 1p/19q codeletion follows it. Glioblastoma starts without IDH, usually with TERT.

Cell of origin, founder events and progression events differ

How an IDH mutation makes a glioma

- Mutant enzyme makes 2-hydroxyglutarate (2-HG)
- 2-HG blocks TET demethylases → CpG hypermethylation (G-CIMP)
- Differentiation genes and MGMT silenced
- Insulator failure switches on PDGFRA
- 2-HG suppresses T cells: an immunologically cold tumour
- Rationale for IDH inhibitors (vorasidenib)

Core pathways and oncohistones

RTK / RAS / PI3K growth signalling

- EGFR amplification (~40%), EGFRvIII
- PDGFRA, MET amplification; FGFR–TACC fusion
- PTEN loss (chromosome 10 loss 60–85%)
- NF1 loss; PI3K mutation
- → proliferation and survival

p53 pathway

- TP53 mutation (most IDH–mutant astrocytomas)
- MDM2/MDM4 amplification
- CDKN2A loss removes p14ARF
- → escape from apoptosis
- → genomic instability

RB / cell–cycle pathway

- CDKN2A/B deletion (p16, p15)
- CDK4 or CDK6 amplification (10–15%)
- RB1 loss (13q loss in about a third)
- Usually only one of these per tumour
- → unchecked G1→S entry

Children and young adults: the histone route (epigenetic “oncohistones”)

H3 K27M (H3.3 or H3.1 genes; EZHIP mimics it) blocks the PRC2 complex → global loss of H3K27 trimethylation → developmental genes stay switched on.

Midline (pons, thalamus, spinal cord); DIPG often adds ACVR1 (H3.1) and TP53/PDGFR changes. H3 G34R/V acts in hemispheric tumours of teenagers.

H3 mutations are mutually exclusive with IDH mutation: each defines a separate disease (Sturm 2012). Paediatric LGG instead has a single MAPK driver (BRAF, FGFR1, NF1, MYB).

RTK/RAS/PI3K, p53 and RB in adults; H3 K27M and G34 in children

What each marker is for



Marker	Diagnostic	Prognostic	Predictive	Practical consequence
IDH1/2 mutation	●	●	●	Vorasidenib (grade 2, residual)
1p/19q codeletion	●	●	●	RT + PCV; most chemosensitive
ATRX loss / TP53	●	○	○	Lineage surrogate (astrocytoma)
MGMT methylation	○	●	●	Temozolomide benefit; elderly choice
CDKN2A/B deletion	●	●	○	Upgrades to grade 4
TERT, EGFR, +7/-10	●	●	○	Defines glioblastoma; no targeted drug yet
H3 K27 / H3 G34	●	●	●	Grade 4; dordaviprone (K27M)
BRAF V600E	●	●	●	Dabrafenib + trametinib
BRAF fusion, FGFR, MYB	●	●	●	Tovorafenib, MEK inhibitors; trials
NTRK fusion	●	○	●	Larotrectinib, entrectinib
MMR loss / hypermutation	○	●	●	Germline CMMRD: anti-PD-1; post-TMZ: little benefit

Diagnostic, prognostic, predictive; IDH → 2-HG → hypermethylation → MGMT silenced

Testing pathway in the laboratory

Step 1 • Histology + immunohistochemistry panel on every diffuse glioma: IDH1 R132H • ATRX • p53 • Ki-67

Add H3 K27M and H3K27me3 (midline) • H3 G34 (young, hemispheric) • BRAF V600E/VE1 (young or epithelioid)

IDH1 R132H positive

- ATRX lost / p53 strong → astrocytoma (1p/19q not needed)
- ATRX retained → test 1p/19q (NGS or methylation; FISH with care)
- Then CDKN2A/B (grade 4 if deleted)

IDH1 R132H negative

- Under 55 or grade 2–3: sequence IDH1 (132) and IDH2 (172)
- Midline: H3 K27 alteration
- Hemispheric, young: H3 G34
- Child: BRAF, FGFR1, MYB/MYBL1, NTRK (RNA NGS)

IDH-wildtype adult, low grade

- TERT promoter, EGFR amplification, +7/-10 → glioblastoma, grade 4
- None present and H3 wild-type → astrocytoma, IDH-wildtype, NEC
- Methylation profiling to resolve

Step 2 • Treatment-guiding tests: MGMT promoter methylation (every glioblastoma; key over 65)

BRAF V600E • NTRK/FGFR fusions • MMR and tumour mutational burden when hypermutation or a syndrome is suspected
Unresolved, unusual or paediatric tumour → DNA methylation profiling (also gives copy number and MGMT status).

IHC first; sequencing, copy number and methylation only where they change the answer

Markers that change treatment

Marker	Treatment consequence
MGMT methylated	Temozolomide benefit; TMZ alone in frail elderly
1p/19q codeleted	RT + PCV (or TMZ)
IDH mutant, grade 2 residual	Vorasidenib
BRAF V600E	Dabrafenib + trametinib
NTRK fusion	Larotrectinib, entrectinib
H3 K27M	Dordaviprone at recurrence
Germline MMR deficiency	Anti-PD-1

Molecular pitfalls

- IDH IHC negative $\neq$ IDH-wildtype under 55
- FISH “codeletion” without IDH = partial loss
- ATRX loss also in H3 G34 gliomas
- NOS = test missing; NEC = tested, does not fit
- Treatment-induced hypermutation $\neq$ IO-responsive
- Germline clues: IDH1 R132C, MMR loss, NF1

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Age, function and MGMT decide

IDH-wildtype glioblastoma after maximal safe resection or biopsy

≤70 years, KPS ≥70

- RT 60 Gy + daily temozolomide, then 6 monthly cycles
- MGMT methylated: lomustine–temozolomide an option for young, fit patients
- Add tumour–treating fields if the patient accepts
- Unmethylated: same standard; consider trials

>70 years, KPS ≥70

- Short–course RT (40/15) + temozolomide
- Methylated and frail: temozolomide alone
- Unmethylated: short–course RT alone
- Decide with the family; function first

KPS <70 (any age)

- Methylated: temozolomide alone or short RT
- Unmethylated: short RT or best supportive care
- Early palliative care
- Steroids at the lowest dose that works

Not recommended first line: bevacizumab (no survival gain: AVAglio, RTOG 0825), nivolumab (CheckMate 498), more than 6 adjuvant cycles (GEINO 14-01).

Standard chemoradiation for fit ≤70; short course for older or frail patients

Glioblastoma: what the trials show

Trial	Result	Practice
EORTC/NCIC	Median 14.6 vs 12.1 mo; 5-y 10 vs 2%	RT + temozolomide
Hegi (MGMT)	21.7 vs 15.3 mo if methylated	Test MGMT
EF-14	20.9 vs 16.0 mo with TTFields	Option if accepted
AVAglio / RTOG 0825	PFS up, OS same	No bevacizumab first line
Perry 2017	Short RT + TMZ better in elderly	Standard ≥ 65 fit

What not to do first line

- Bevacizumab (no survival gain)
- Nivolumab in unmethylated tumours (worse survival)
- Dose-dense temozolomide
- More than 6 adjuvant cycles
- RT dose above 60 Gy

Temozolomide safety

Issue	Action
Thrombocytopenia (10–20%)	Weekly counts during RT; hold if platelets fall
Women	2–3 times more haematological toxicity
PJP	Prophylaxis with steroids or lymphopenia
Liver injury	LFTs at baseline and each cycle
Pregnancy	Contraception; fertility counselling

Tumour-treating fields in practice

- Four arrays on a shaved scalp, ≥ 18 hours a day
- Started with adjuvant temozolomide
- Skin irritation is the main side effect
- Not sham-controlled; survival gain ~5 months
- Cost and availability limit use in India

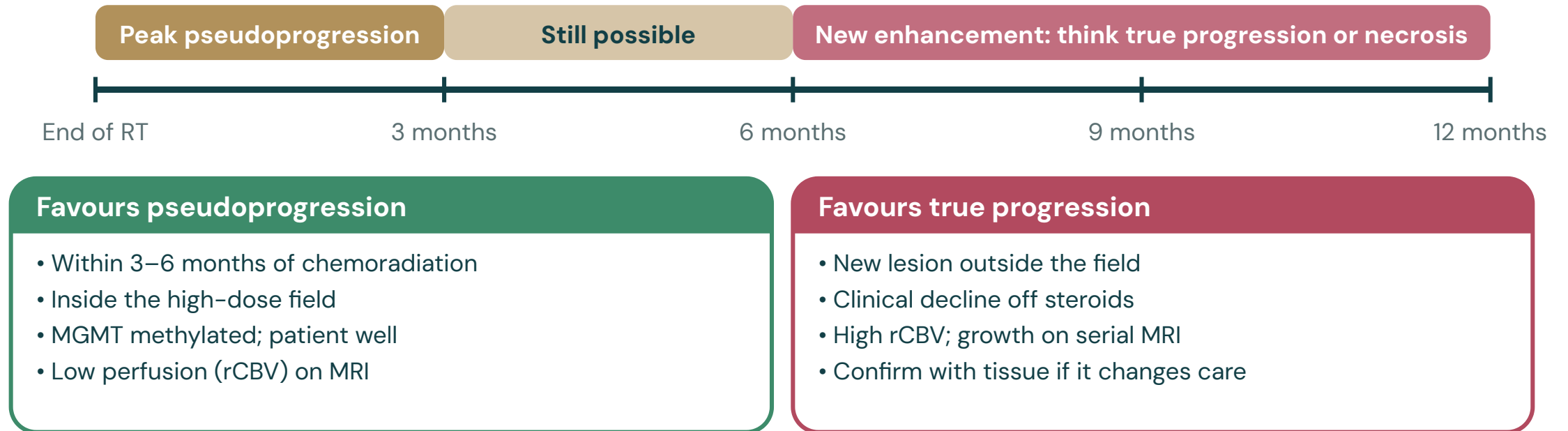
Radiotherapy schedules

Setting	Usual schedule (dose per course, no drug doses)
Glioblastoma, ≤70 y, fit	60 Gy in 30 fractions + daily temozolomide
Grade 3 glioma	59.4 Gy in 33 fractions (or 60/30)
Grade 2 glioma (when RT chosen)	50.4–54 Gy in 28–30 fractions
Older or frail glioblastoma	40 Gy in 15 fractions (or 25 Gy in 5)
1–4 (up to ~10–15) small metastases	Stereotactic radiosurgery, single or 3–5 fractions
Resected metastasis cavity	Cavity SRS rather than whole brain
Many metastases, no SRS option	WBRT 30 Gy in 10 (hippocampal avoidance + memantine)
Medulloblastoma, ependymoma (spread)	Craniospinal irradiation + boost
DIPG	54–59.4 Gy, or 39–45 Gy hypofractionated

Involved-field RT: tumour (enhancement, cavity ± FLAIR) plus a margin; 80–90% of high-grade gliomas recur within 2 cm of the original site.

Involved-field RT; 80–90% of high-grade gliomas recur within 2 cm

Pseudoprogression



Early in-field enhancement: continue and re-image

Re-irradiation

- Focal relapse after a long interval: consider re-RT
- RTOG 1205: re-RT + bevacizumab improved PFS, not OS
- Radiation-induced gliomas: re-RT acceptable after ~5 years
- Weigh necrosis risk and goals

Recurrent glioblastoma

- No therapy clearly prolongs survival; median ~8–9 months
- Re-resection or re-irradiation for focal relapse
- Lomustine; bevacizumab for oedema; temozolomide rechallenge if late and methylated
- BRAF V600E: dabrafenib + trametinib; NTRK: TRK inhibitors
- H3 K27M: dordaviprone
- Palliative care alongside

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IDH-mutant gliomas

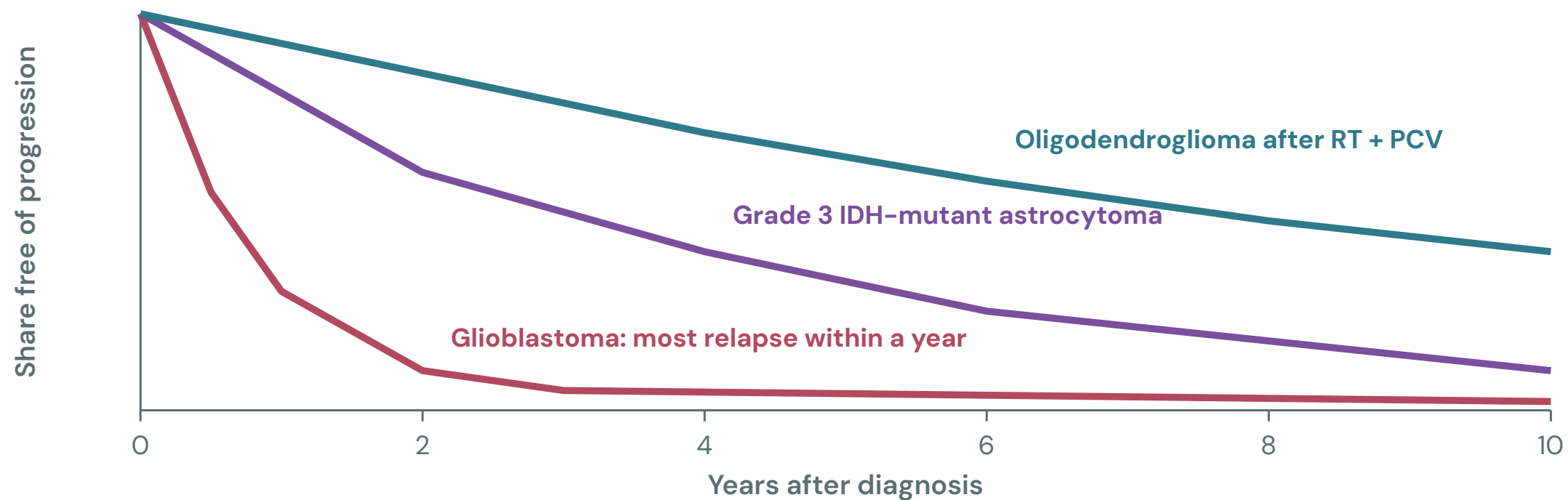
IDH-mutant gliomas

Situation	Plan	Evidence
Grade 2, residual, controlled	Vorasidenib or watch	INDIGO: PFS 27.7 vs 11.1 mo
Grade 2 needing treatment	RT + PCV or temozolomide	RTOG 9802: 13.3 vs 7.8 y
Astrocytoma grade 3	RT + 12 cycles temozolomide	CATNON final
Oligodendroglioma grade 3	RT + PCV (or temozolomide)	RTOG 9402 / EORTC 26951
Astrocytoma grade 4	RT + temozolomide	Extrapolated

Oligodendroglioma pearls

- Seizure is the commonest first symptom
- Calcification, frontal, cortical
- Most chemosensitive glioma
- Median survival 15–20 years with modern therapy
- Very large field: chemotherapy first
- Procarbazine supply: temozolomide as alternative

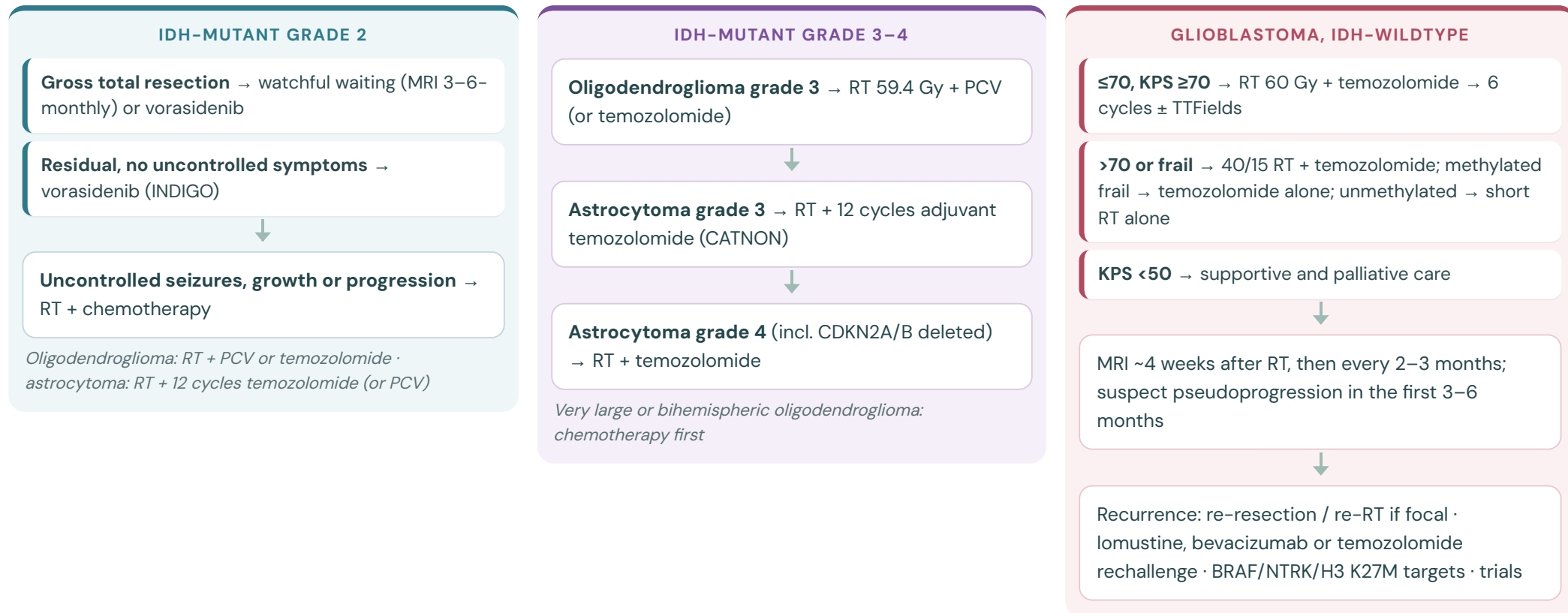
When relapses happen



Tenfold difference in pace between glioma types

Algorithm: adult diffuse glioma

Suspected glioma on MRI: neuro-oncology MDT · steroids only if symptomatic · antiseizure drug only after a seizure · **maximal safe resection** (biopsy if deep or diffuse) · MRI in 24–48 h · integrated diagnosis: IDH, 1p/19q, CDKN2A/B, MGMT



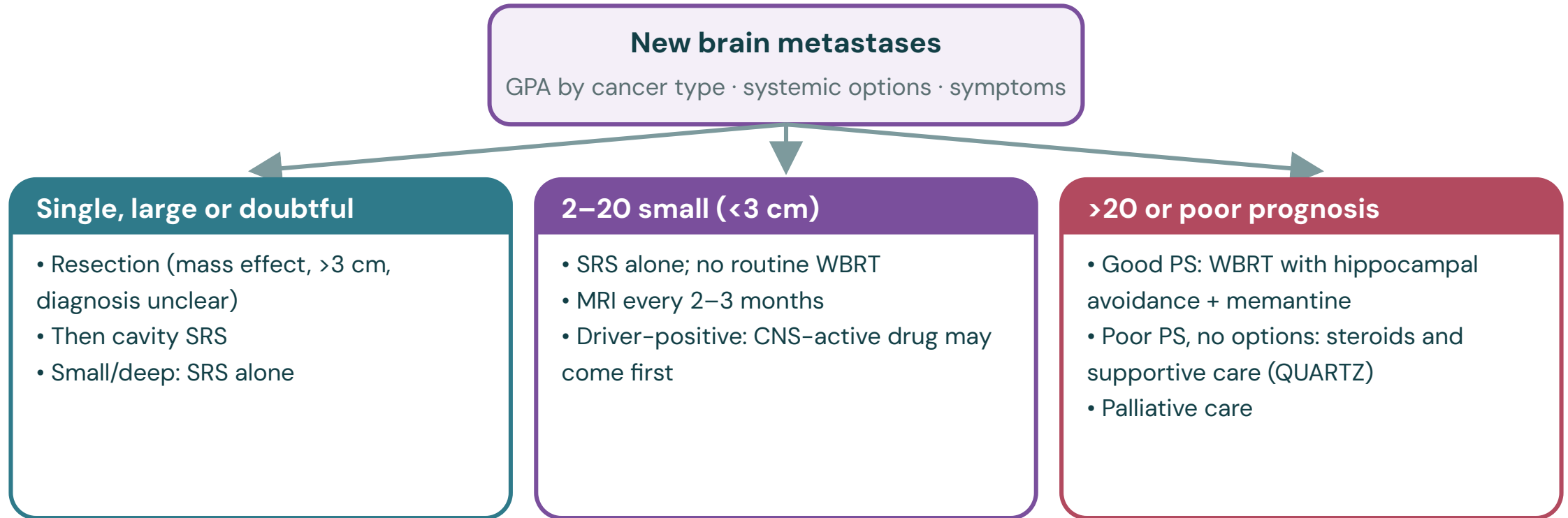
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Brain metastases

Brain metastases: principles

- Commonest brain tumour in adults
- Prognosis by disease-specific GPA (8–16 months, longer with CNS-active drugs)
- Genotype the primary or metastasis
- Steroids only for symptoms
- No prophylactic antiseizure drugs

Choosing local therapy



Adding WBRT halves intracranial relapse but does not prolong life and harms memory (EORTC 22952, NO574, JROSG 99-1).

Number, size, symptoms and systemic options decide

Metastases: the trials

Trial	Question	Answer
Patchell 1990	Surgery + WBRT vs WBRT	40 vs 15 weeks
N0574	SRS ± WBRT	WBRT: more decline, no OS gain
Mahajan	Cavity SRS vs observation	Local control 72 vs 43%
N107C	Cavity SRS vs WBRT	Less cognitive decline
QUARTZ	WBRT vs supportive care	No difference

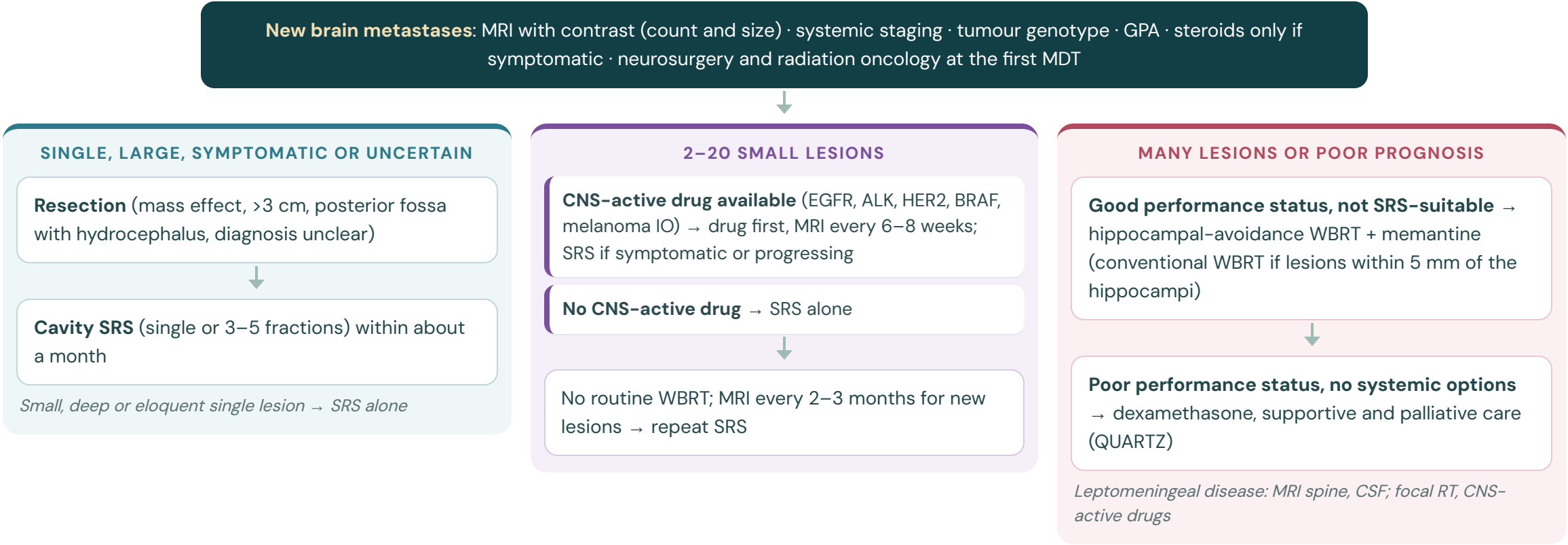
CNS-active systemic therapy

- EGFR: osimertinib; ALK: alectinib, lorlatinib
- HER2 breast: tucatinib-based regimens
- Melanoma: ipilimumab + nivolumab; BRAF/MEK
- Small asymptomatic lesions: drug first, MRI at 6–8 weeks
- SRS for symptomatic or progressing lesions

Leptomeningeal disease and necrosis

- Leptomeningeal: MRI brain and spine, CSF cytology
- Focal RT, CNS-penetrant drugs, selected intrathecal therapy
- Radiation necrosis after SRS: 10–25%, more with IO or TKIs
- Perfusion MRI, spectroscopy or tissue to tell from relapse
- Symptomatic necrosis: steroids or bevacizumab

Algorithm: brain metastases

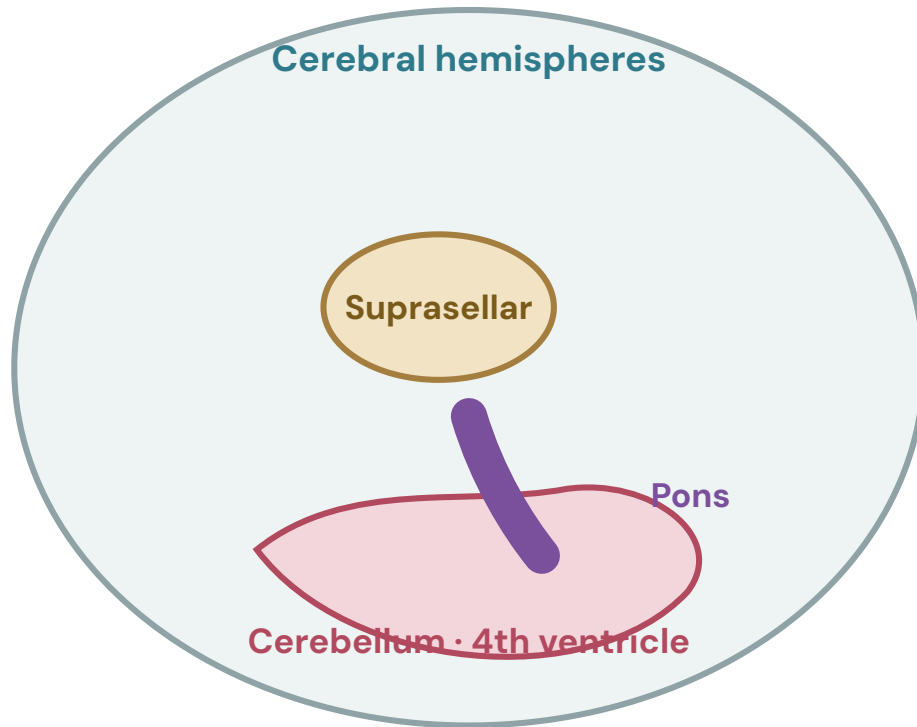


All patients: no prophylactic antiseizure drugs · DOAC or LMWH for VTE (individualise in melanoma, RCC, thyroid, choriocarcinoma) · taper steroids early, especially before immunotherapy · driving advice · discuss goals of care.

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Children

Location predicts the tumour



Posterior fossa (~half)

Pilocytic astrocytoma, medulloblastoma, ependymoma, DIPG (pons).
Morning vomiting, ataxia, head tilt, hydrocephalus.

Suprasellar / optic

Craniopharyngioma, optic pathway glioma (NF1), germ cell tumour.
Vision loss, short stature, polyuria, early puberty.

Hemispheres

Low-grade glioneuronal tumours (seizures), high-grade gliomas.
Seizures, weakness, behaviour change.

Infants

Large head, bulging fontanelle, sunset eyes, loss of milestones.
Embryonal tumours, ATRT.

Half in the posterior fossa; symptoms follow location

Image a child when

- Headache waking from sleep or on waking
- Headache worse lying down, or age ≤ 6 years
- Persistent morning vomiting
- New squint, papilloedema, nystagmus, reduced acuity
- Ataxia, head tilt, regression of milestones
- Large head, bulging fontanelle in an infant

Paediatric low-grade glioma

Situation	Plan
Resectable	Gross total resection → surveillance
BRAF V600E	Dabrafenib + trametinib first line
BRAF fusion	Chemotherapy or tovorafenib
NF1 optic glioma	Treat for vision loss; avoid RT
Outcome	~90% alive at 20 years; half progress

Medulloblastoma and ependymoma

- Medulloblastoma: WNT, SHH, group 3, group 4
- Surgery, risk-adapted craniospinal RT, cisplatin-based chemo
- Infants: chemotherapy to delay or avoid RT
- Ependymoma: maximal resection then focal RT
- Post-RT chemotherapy did not help (ACNS0831)
- PFA (H3K27me3 loss) is higher risk

High-grade glioma and DIPG

- HGG >3 y: focal RT + temozolomide → lomustine
- BRAF V600E: dabrafenib + trametinib; CMMRD: anti-PD-1
- DIPG: MRI diagnosis; RT only proven therapy
- Hypofractionated RT non-inferior (Zaghloul)
- Median survival under 1 year; early palliative care

Radiotherapy in children

- Avoid cranial RT under 3 years where possible
- Focal conformal or proton RT for LGG and ependymoma
- Craniospinal RT + boost for medulloblastoma
- Anaesthesia for young children: hypofractionation helps
- Plan late-effects follow-up from day one

Algorithm: children

Red flags (morning vomiting, ataxia, squint, head tilt, regression, headache ≤ 6 years) → urgent MRI (CT if unavailable) → paediatric neurosurgery the same day if hydrocephalus · steroids for oedema · staging: spine MRI, CSF, AFP/ β -hCG where relevant · tissue + molecular (BRAF, H3, groups)



LOW-GRADE GLIOMA / GLIONEURONAL

Resectable → gross total resection → MRI surveillance (cure in most)



Unresectable or progressive



BRAF V600E → dabrafenib + trametinib

BRAF fusion / NF1 → carboplatin–vincristine or vinblastine; tovorafenib or MEK inhibitor

Optic pathway glioma in NF1: treat for vision loss, not size alone

EMBRYONAL AND EPENDYMOMA

Medulloblastoma → resection → craniospinal RT by risk group + chemotherapy (≥ 3 years); chemotherapy-based under 3



Ependymoma → maximal resection (second look if residual) → focal RT; < 1 year: chemotherapy bridge

Germline testing: SHH medulloblastoma (TP53, SUFU, PTCH1), ATRT (SMARCB1)

HIGH-GRADE GLIOMA AND DIPG

Hemispheric HGG → maximal resection → focal RT + temozolomide → lomustine

BRAF V600E → dabrafenib + trametinib after RT · CMMRD → anti-PD-1

DIPG (diagnosed on MRI) → RT (conventional or hypofractionated) · steroids · trials · early palliative care

All children: paediatric neuro-oncology centre · fertility preservation where possible · school and neurocognitive support · lifelong late-effects clinic (endocrine, hearing, growth, second tumours) · family genetic counselling when a syndrome is suspected.

Late effects in survivors

- Neurocognition and school performance
- Growth hormone, thyroid, cortisol, puberty
- Hearing loss (cisplatin), vision
- Second tumours, vasculopathy after RT
- Fertility; psychosocial support

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Supportive care and follow-up

Supportive care that matters

Steroids (dexamethasone)

- Only for symptomatic oedema; none if asymptomatic
- Load then 8–16 mg/day for severe, 2–4 mg/day for mild
- Taper to the lowest dose; watch glucose, myopathy
- Avoid before biopsy if lymphoma possible

Seizures

- No prophylaxis if never had a seizure
- After a seizure: levetiracetam (non-enzyme-inducing)
- Avoid phenytoin/carbamazepine with chemo, TKIs
- Rescue benzodiazepine at home near end of life

Venous thrombosis

- No routine prophylaxis except after surgery
- Treat DVT/PE: DOAC preferred, else LMWH
- Higher bleed risk: melanoma, RCC, thyroid, choriocarcinoma
- Non-contrast CT before starting

Infection and stomach

- PJP prophylaxis if steroids or lymphopenia on temozolomide
- H2 blocker rather than PPI with glioblastoma
- Vaccinate; check sugar on steroids
- Early palliative and rehabilitation input

Steroids for symptoms; antiseizure drugs after seizures; DOACs for VTE

Steroids

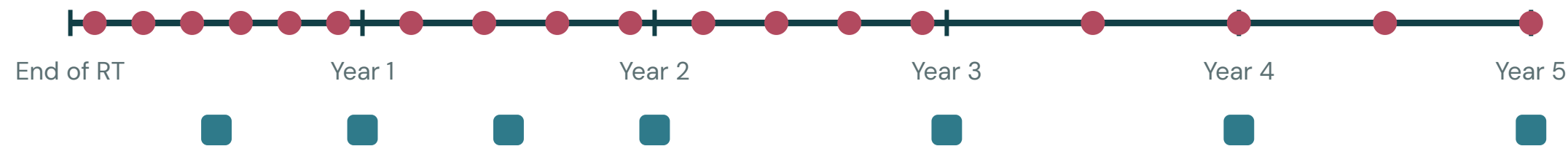
Do	Avoid
Treat symptomatic oedema only	Steroids for asymptomatic oedema
Taper to the lowest dose	Indefinite high doses
Check glucose, watch myopathy	Steroids before biopsy if lymphoma possible
H2 blocker if needed	Routine PPI with glioblastoma

Seizures and thrombosis

Problem	Approach
Never had a seizure	No prophylaxis
After a seizure	Levetiracetam
Chemotherapy or TKI	Avoid enzyme inducers
DVT/PE	CT, then DOAC (or LMWH)
Prophylaxis	Only after surgery

A simple follow-up rhythm

High-grade: MRI ~4 weeks after RT, then every 2–3 months for 2–3 years



- High-grade glioma: MRI + clinic (steroid dose, seizures, cognition, mood, blood counts on temozolomide)
- Low-grade glioma: MRI every 3–6 months for 5 years, then 6–12-monthly for life (more often on vorasidenib early)

Metastases after SRS: MRI every 2–3 months for a year. Children: MRI per protocol; endocrine, hearing, growth, school and fertility review. Treat the person: driving and work advice, epilepsy safety, carer support, advance-care planning when appropriate.

MRI drives follow-up; interpret early changes with care

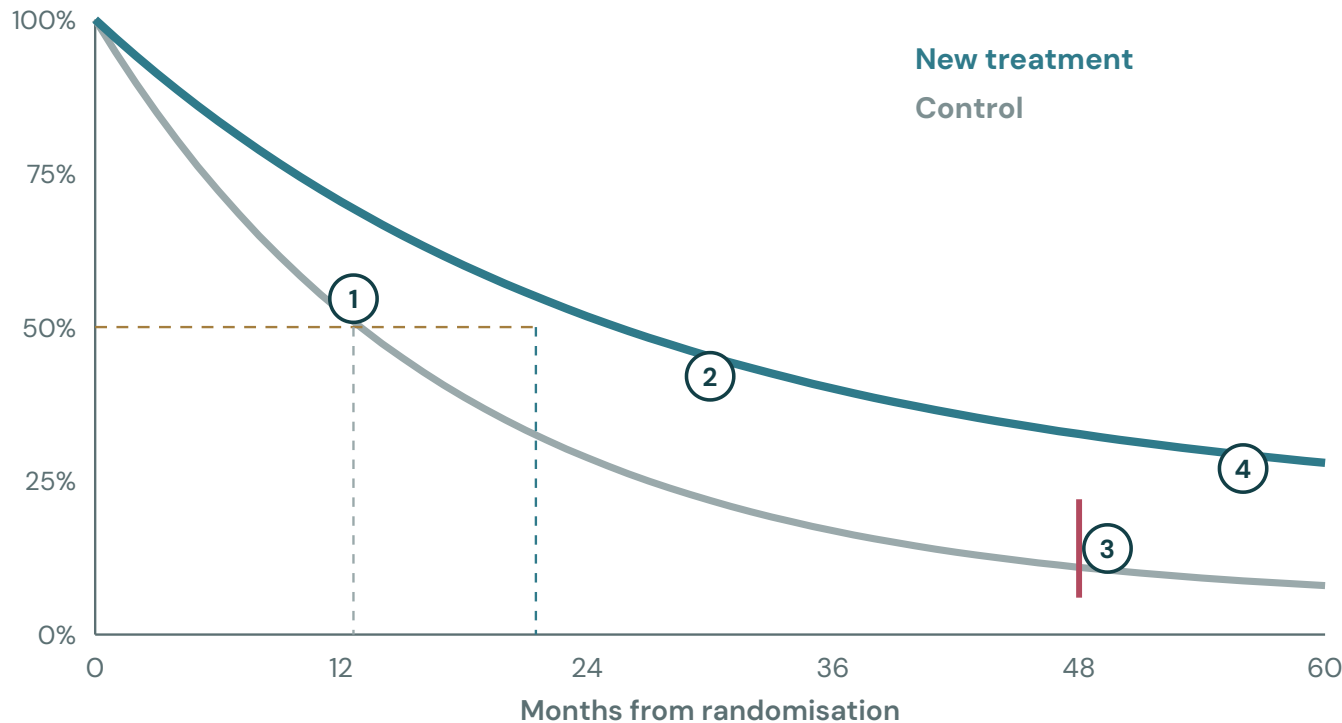
Living with a brain tumour

- Driving advice in writing after a seizure
- Rehabilitation and return to work or school
- Fertility and contraception
- Carer support
- Goals of care and a home seizure plan near the end of life

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Evidence and cases

Reading a survival curve



- 1 Median**
The time at which half the patients have had the event. A median gain of a few months can hide a long tail of survivors.
- 2 Hazard ratio**
Relative risk of the event at any moment (0.70 = 30% lower risk). It is not an absolute difference.
- 3 Absolute difference**
The vertical gap at a fixed time (here about 20 points at 4 years).
 $NNT = 100 \div \text{gap}$.
- 4 The tail**
A plateau suggests some patients gain long-term control: the typical immunotherapy pattern.
- 5 Crossover and PFS**
If control patients get the new drug later, overall survival differences shrink; a PFS gain alone may not mean longer life.

Median, hazard ratio, absolute gap and the tail

Case 1: seizure in a young engineer

- Non-enhancing frontal mass, calcified
- Awake resection, small residual
- IDH-mutant, 1p/19q codeleted, grade 2
- Vorasidenib vs watchful waiting
- RT + PCV if it grows

Case 2: glioblastoma, methylated

- 58-year-old, word-finding difficulty
- Gross total resection; MGMT methylated
- RT + temozolomide ± TTFields
- New rim enhancement at 2 months
- Pseudoprogression: continue and re-image

Case 3: older adult, unmethylated

- 76-year-old, butterfly glioblastoma
- Biopsy only; KPS 70
- Short-course RT alone
- Early palliative care
- Function and family goals first

Case 4: the "tuberculoma" that grew

- Periventricular, diffusion-restricting lesions
- Transient response to steroids
- Taper steroids, stereotactic biopsy
- Primary CNS lymphoma
- Methotrexate-based therapy; stop ATT

Cases 5 and 6: metastases

- EGFR lung, 3 small lesions → osimertinib + MRI; SRS in reserve
- Breast cancer, 3.5 cm cerebellar lesion → resection + cavity SRS
- DVT later → DOAC after CT
- No WBRT, no seizure prophylaxis

Cases 7 and 8: children

- 6-year-old "treated for worms": pilocytic astrocytoma, cured by resection
- 9-year-old with vision loss: hypothalamic LGG, BRAF V600E
- Hydrocortisone before thyroxine
- Dabrafenib + trametinib first line

Practice pearls for Indian clinics

- MRI for a first adult seizure
- Biopsy a lesion that grows on antituberculous treatment
- No steroids before biopsy if lymphoma likely
- Send IDH, 1p/19q and MGMT with the first block
- Hypofractionated RT for long-distance travellers

Take-home messages

- Molecular markers name and grade gliomas
- MGMT and age guide glioblastoma treatment
- RT + chemotherapy for IDH-mutant gliomas needing treatment; vorasidenib can delay it
- SRS, not WBRT, for most metastases
- Children: test BRAF; mind late effects
- Steroids and antiseizure drugs only when needed

THANK YOU

Questions and discussion

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Full mini review, algorithms, MCQ bank and case vignettes: drharitha.co.in/professionals

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