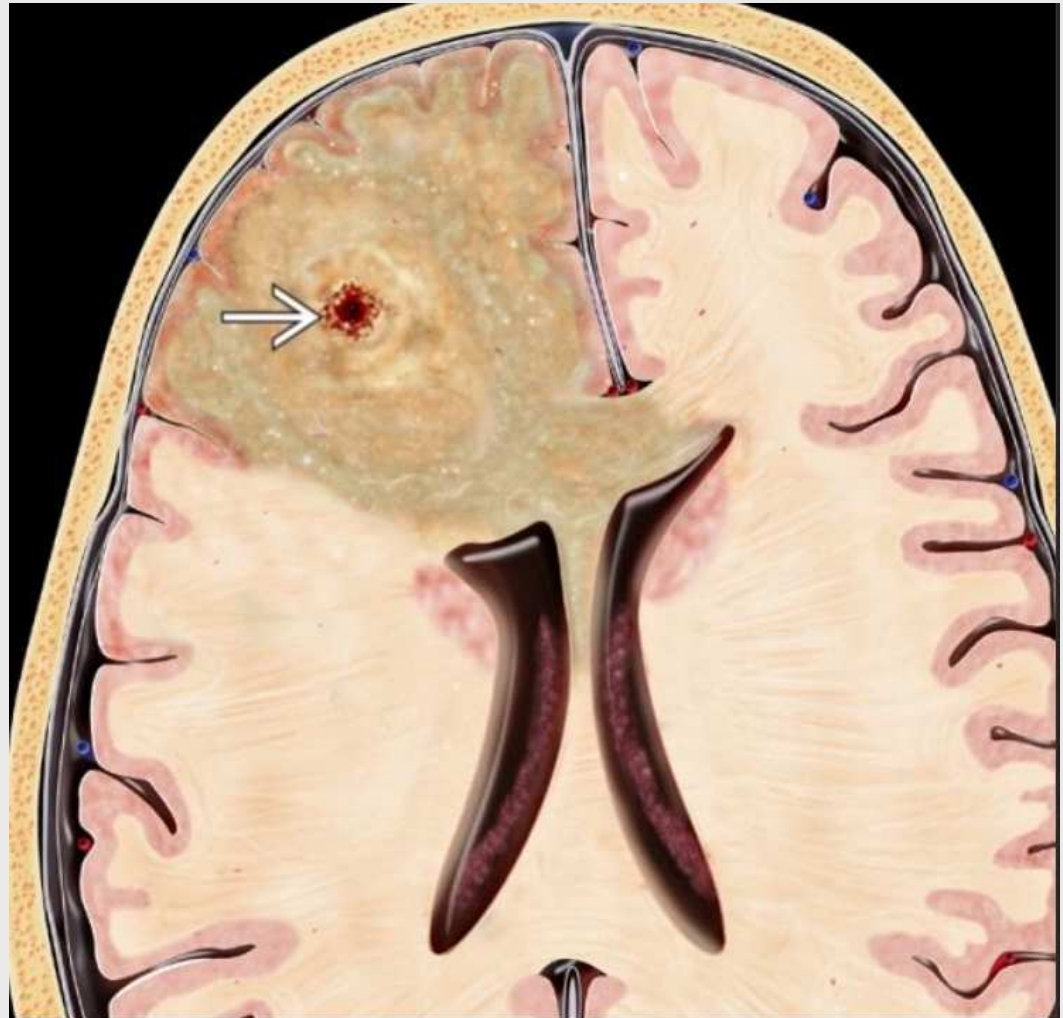
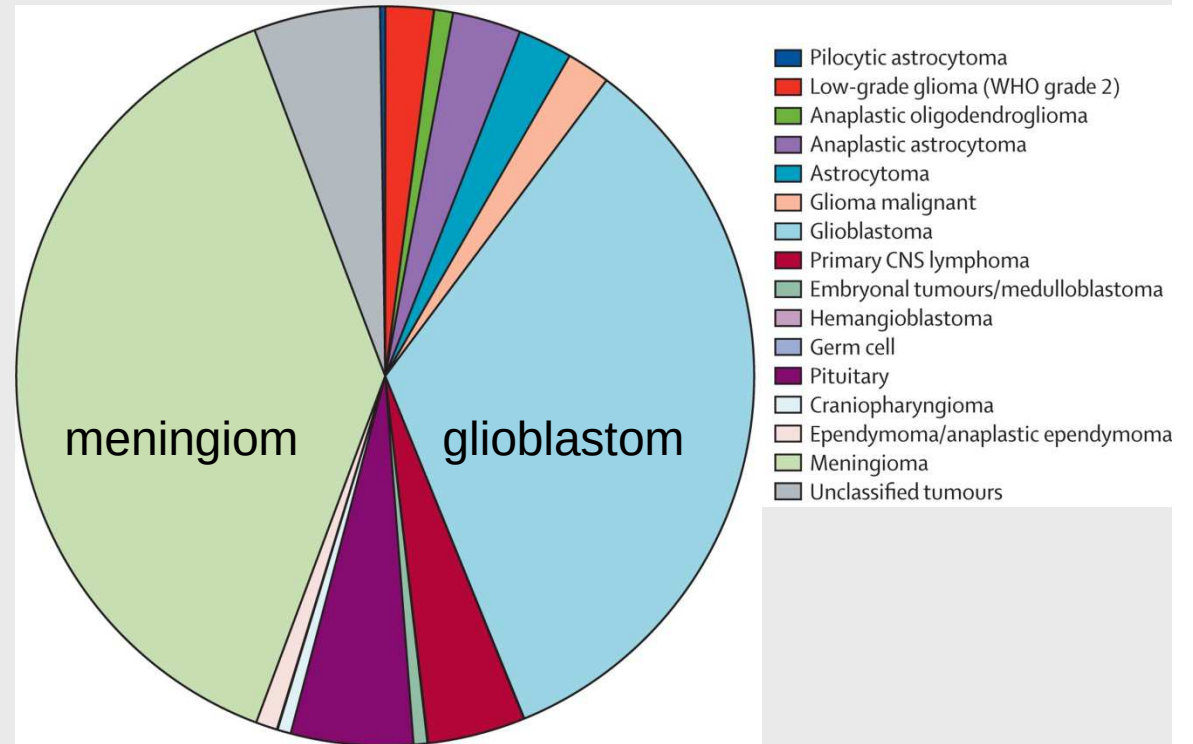


MR diagnostikk av hjernetumor

Kjell Arne Kvistad
seksjonsoverlege dr med
Klinikk for bildediagnostikk
St Olavs hospital



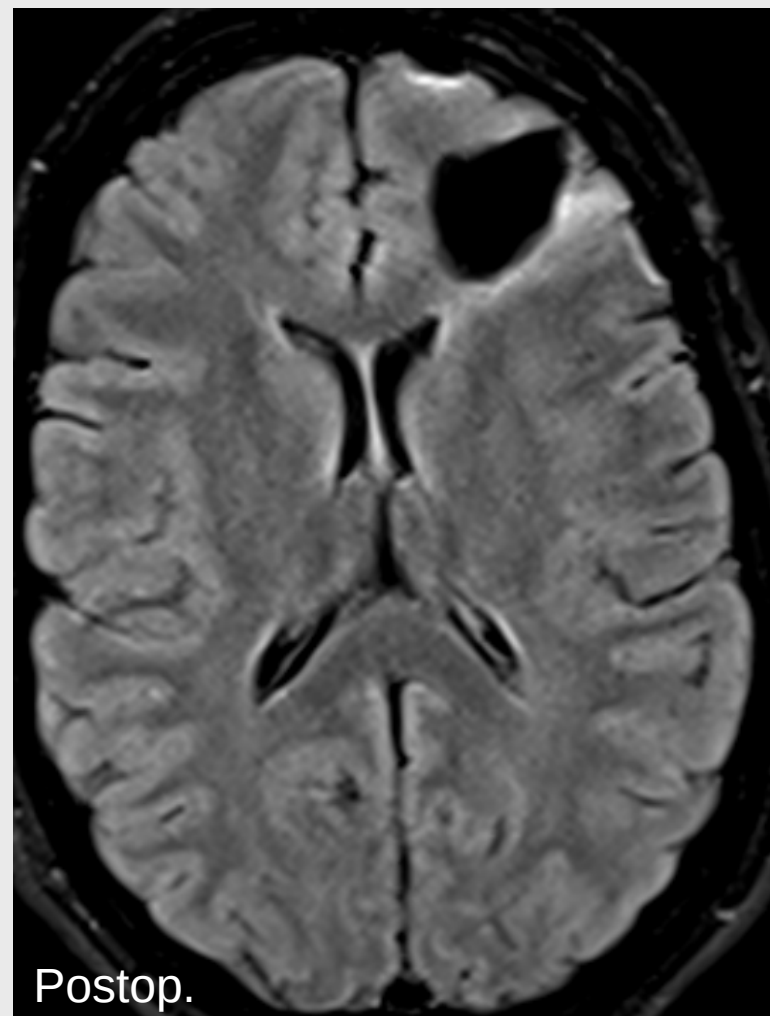
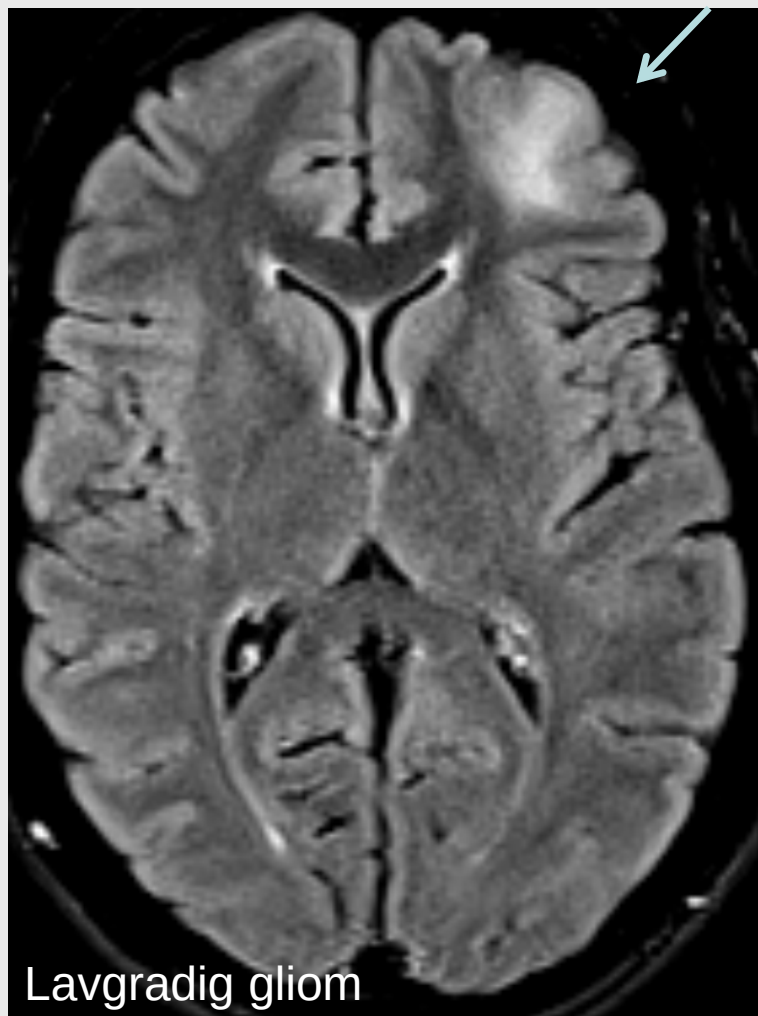
Tumortype



Insidens i Norge:

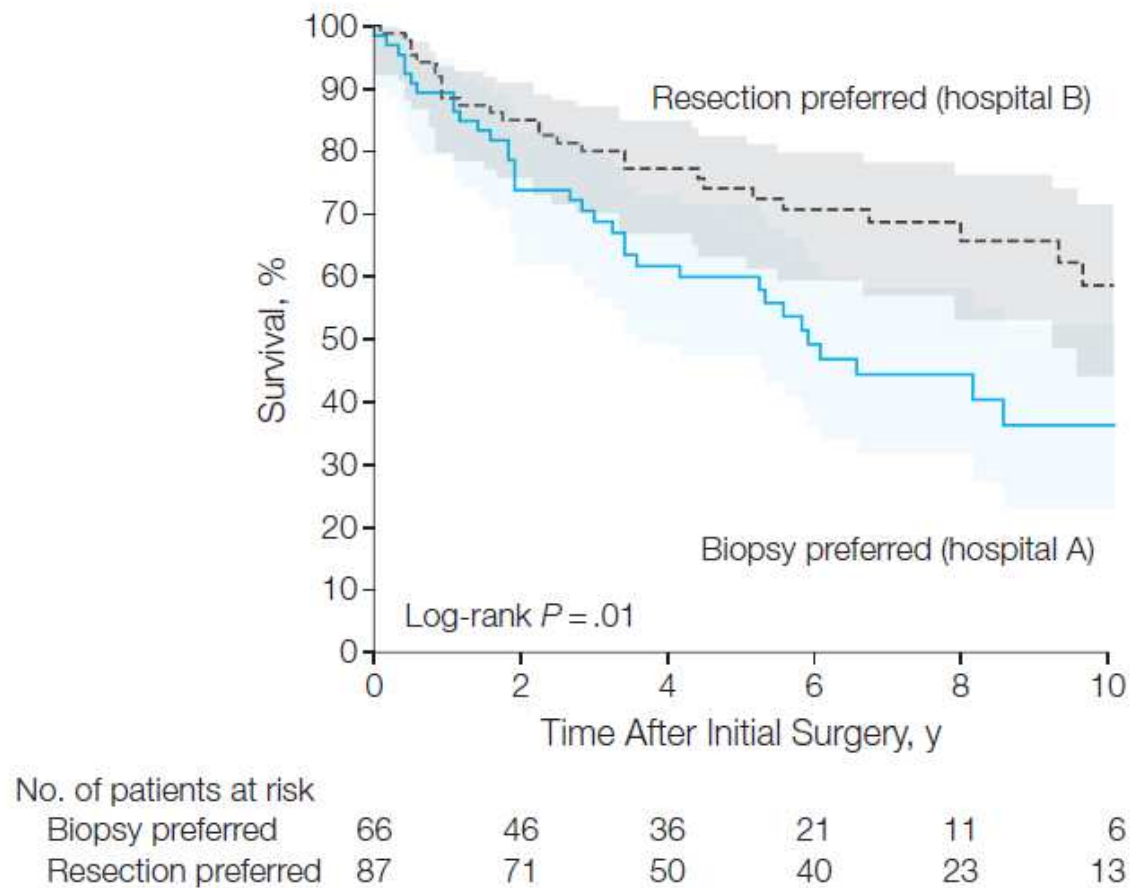
- 1100 primære CNS svulster (alle typer)
- 250 høygradig maligne gliomer (grad III-IV)/år
- 50 lavgradige gliomer (grad II)

Mindre vente og se, også lavgradige svulster bør fjernes

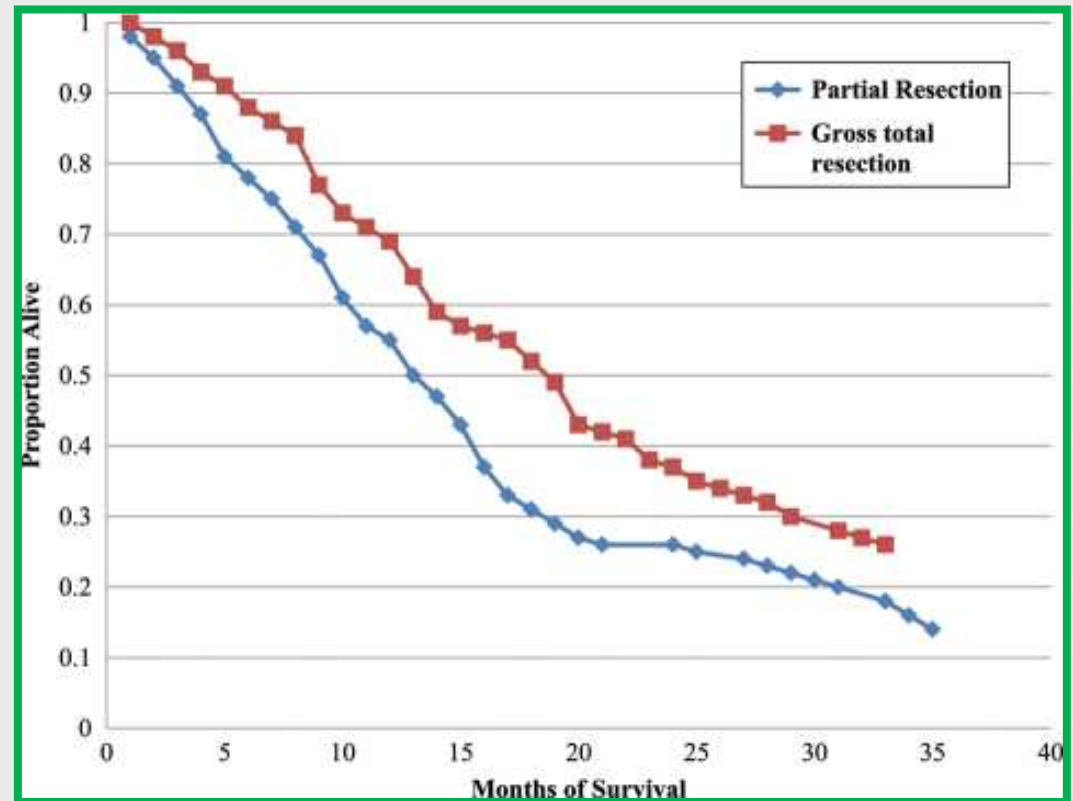
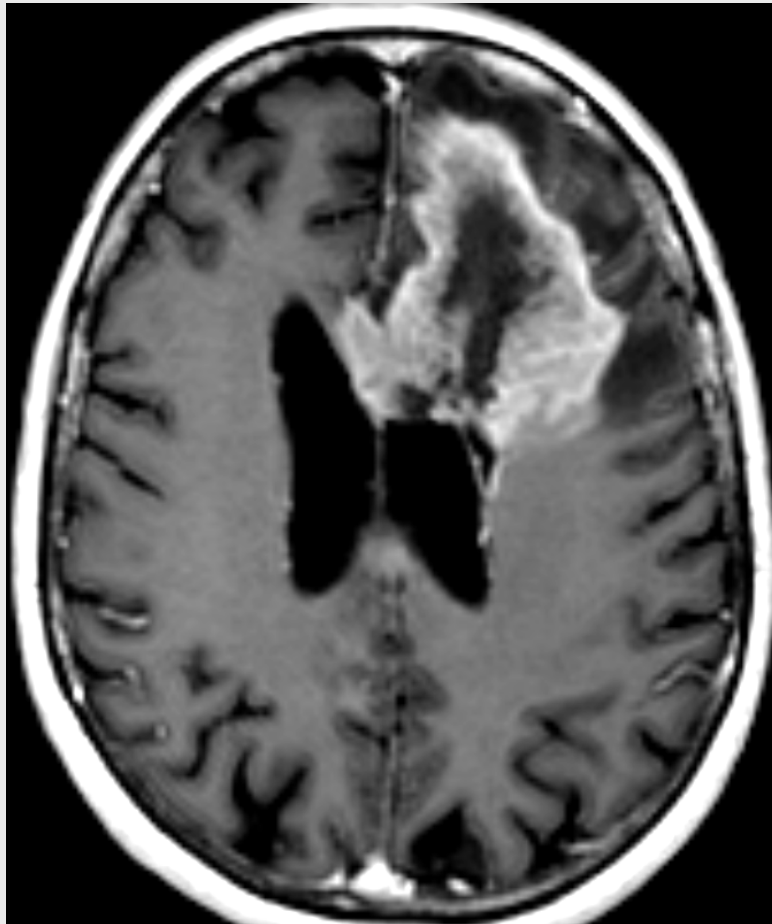


Mindre vente og se, også lavgradige svulster bør fjernes

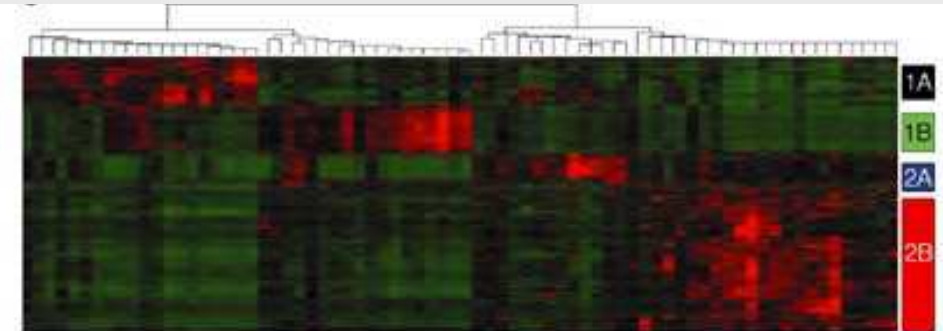
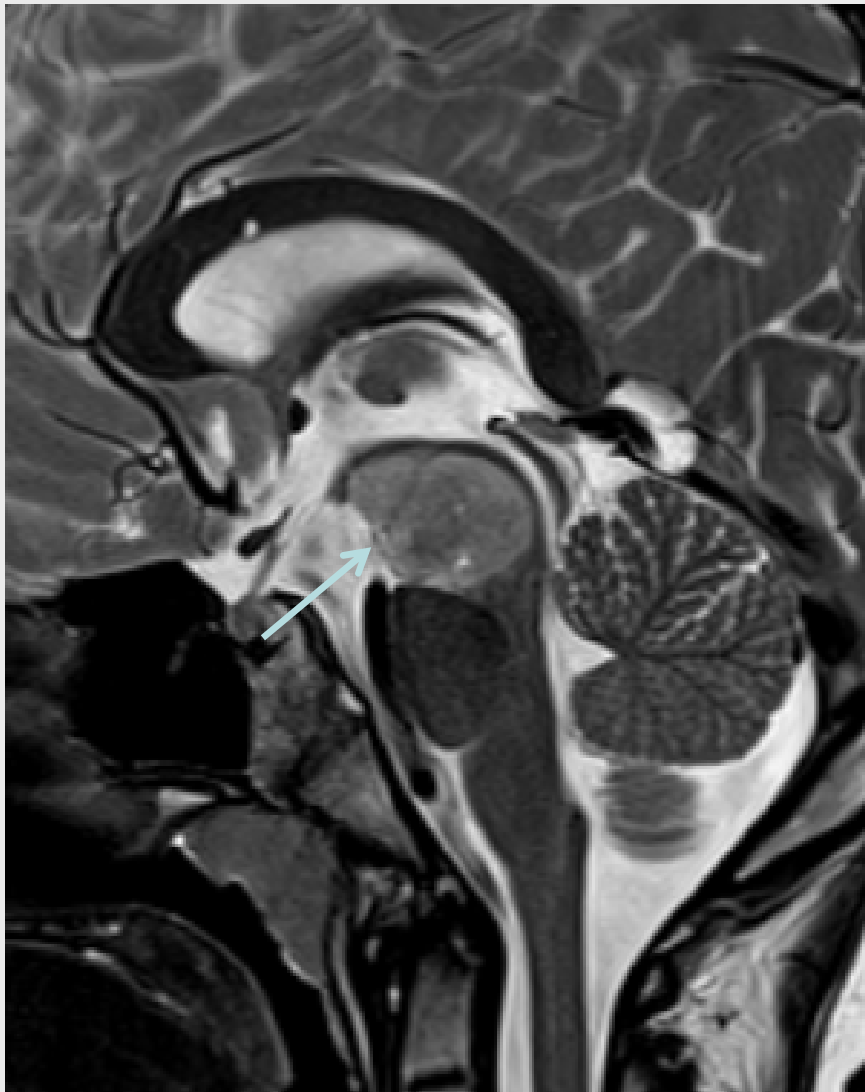
Survival Analysis Comparing Favored Surgical Strategies for Low-Grade Gliomas



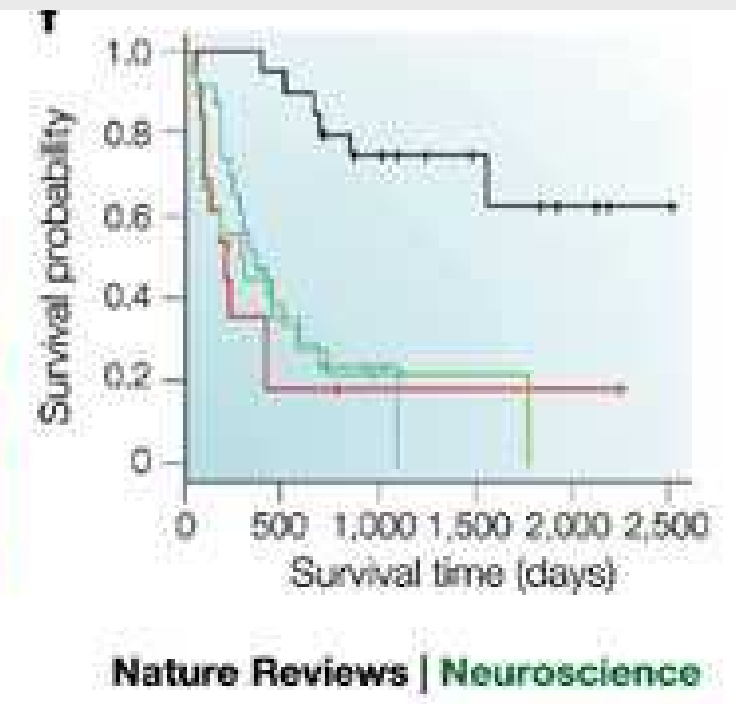
Høygradig maligne svulster: fjern så mye som mulig



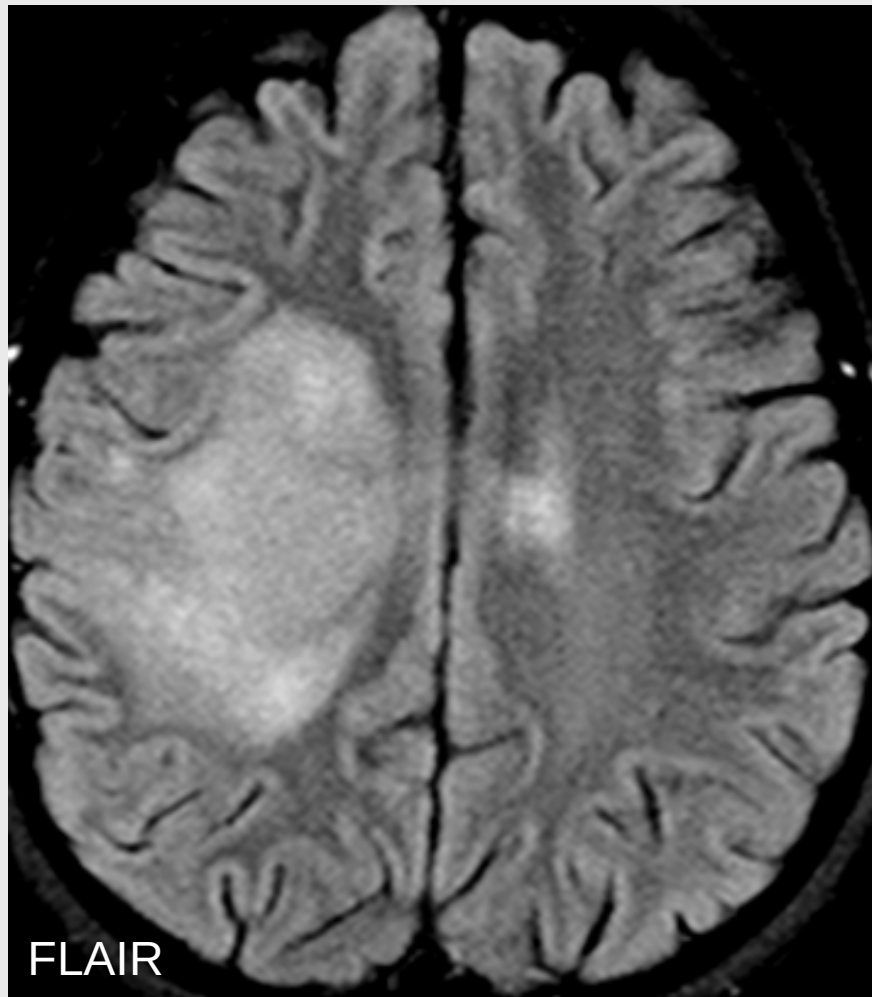
Med få unntak vil tumortype bestemmes med histologi og immunhistokjemi



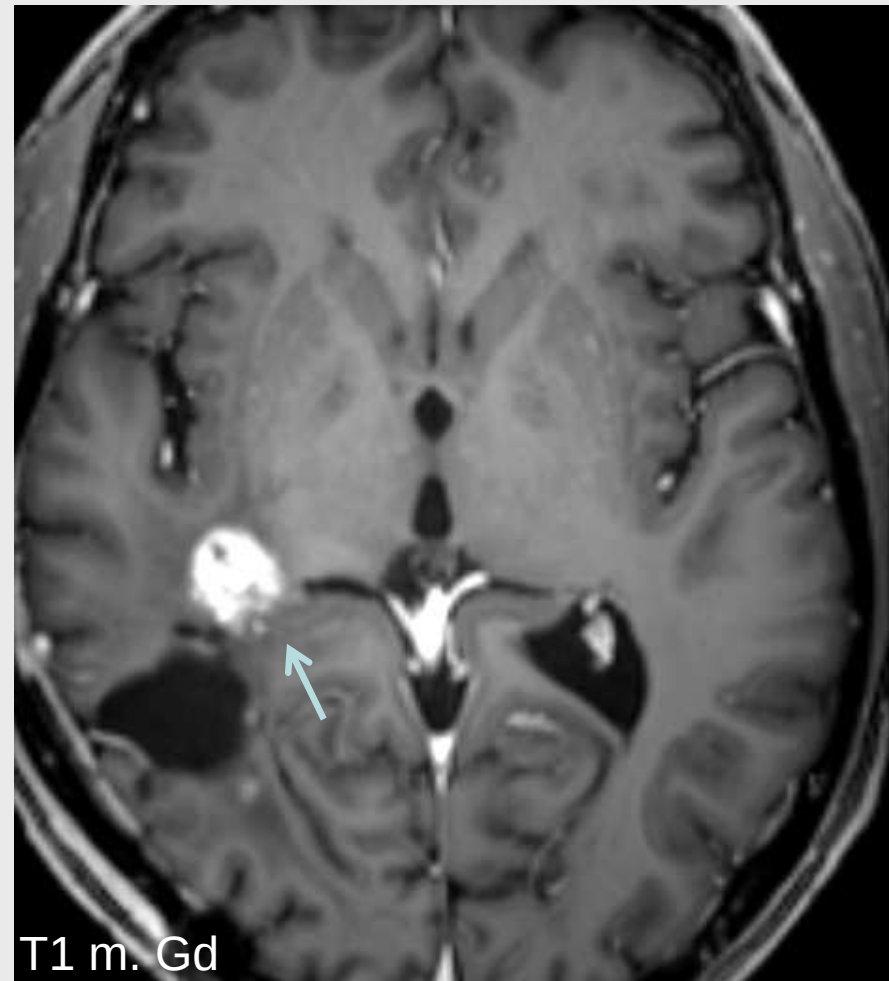
Genekspresjon i ulike hjernesvulster



De to mest sentrale spørsmål:



Er det tumor?



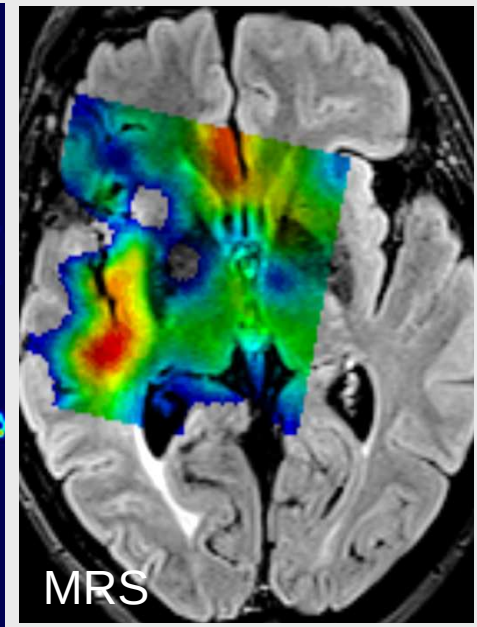
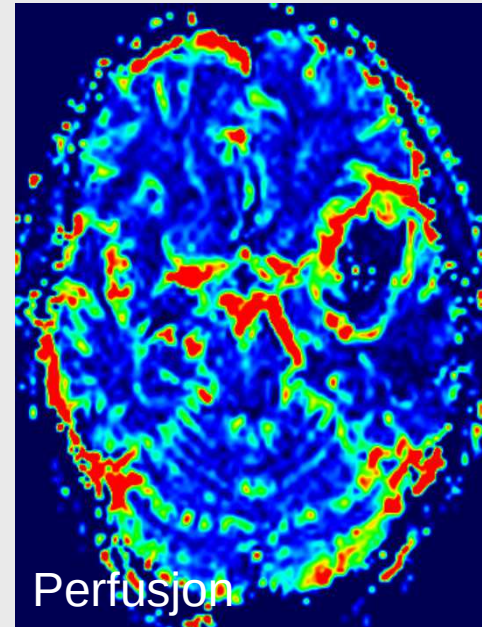
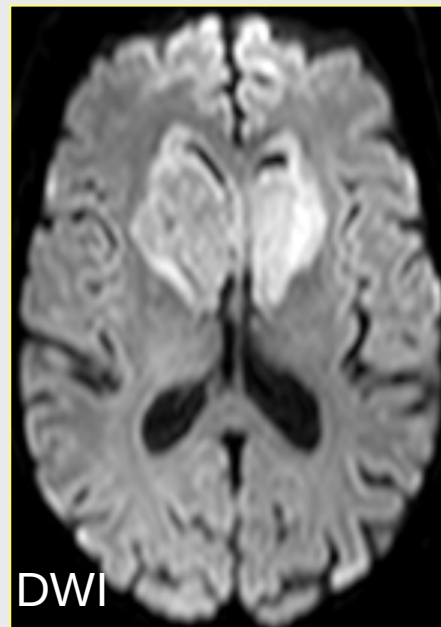
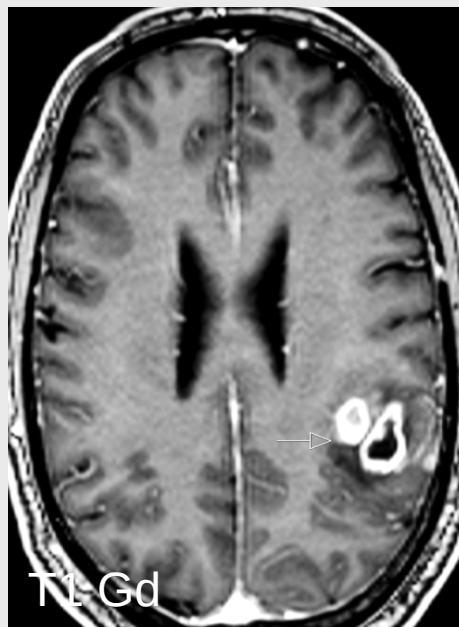
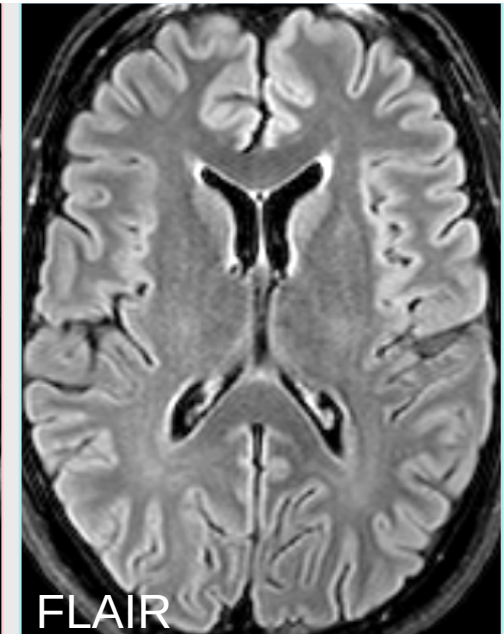
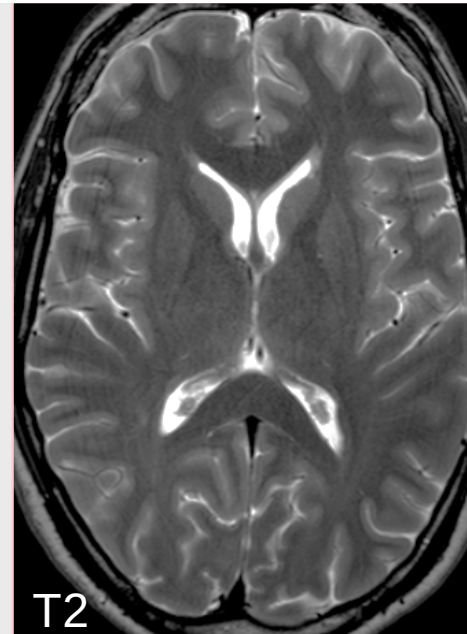
Er det tumorresidiv?

Er det tumor, eller... ??

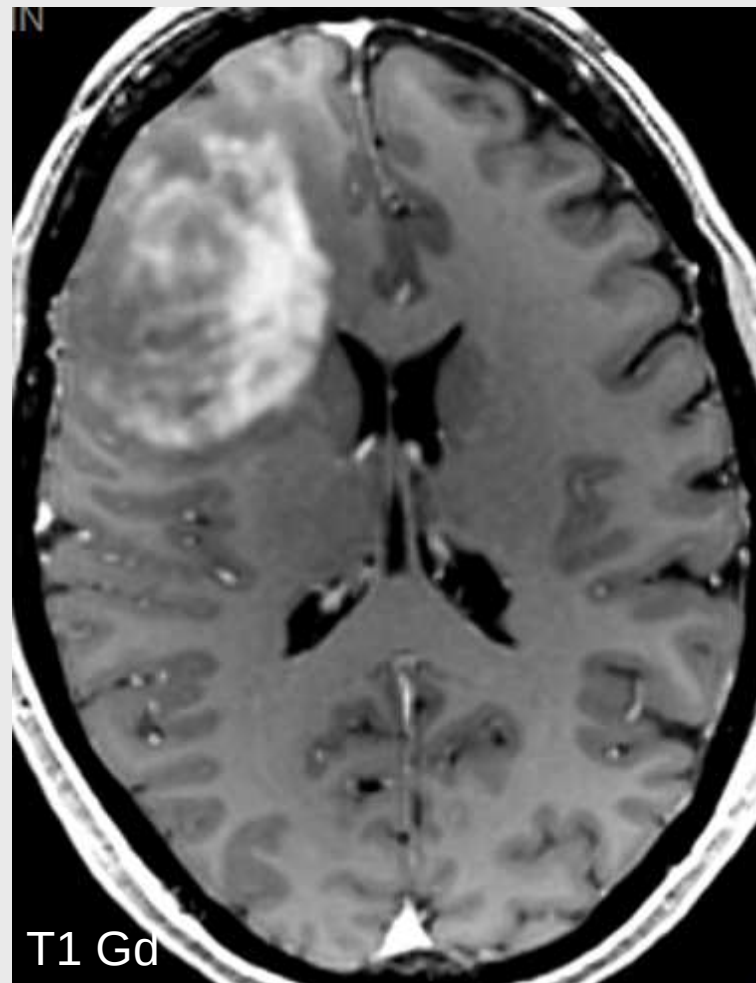
- Demyeliniserende sykdommer (tumefaktiv MS, ADEM)
- Infeksjoner (abscess, encefalitt)
- Subakutte infarkter

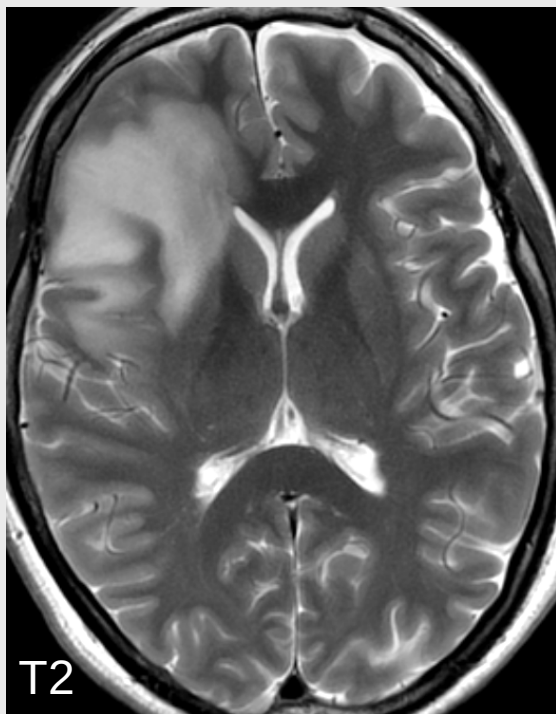


- Klinikk (alder, immunsuppresjon, kreftsykdom, utenlandsopphold)
- Blodprøver, CSF
- CT collum/thorax/abdomen



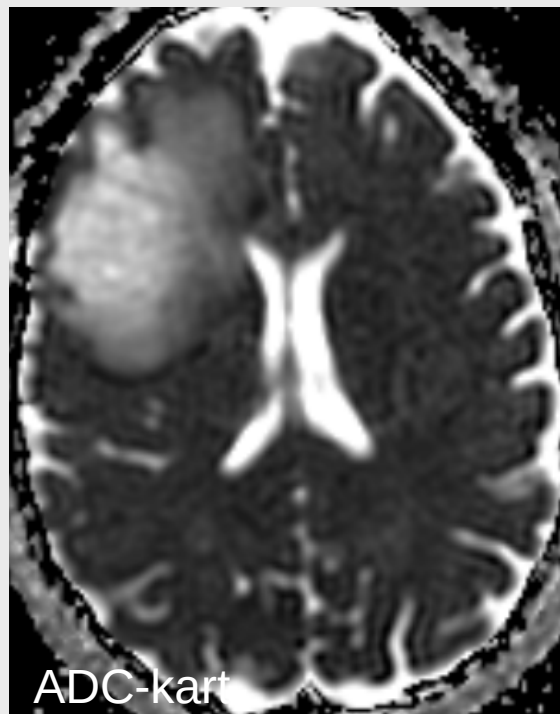
Hodepine og personlighetsforandringer over uker





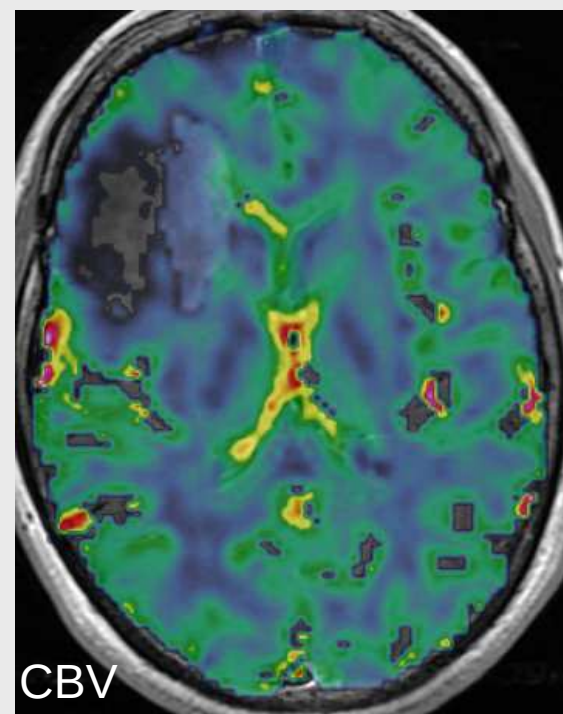
T2

Manglende masseffekt



ADC-kart

Høy diffusjon

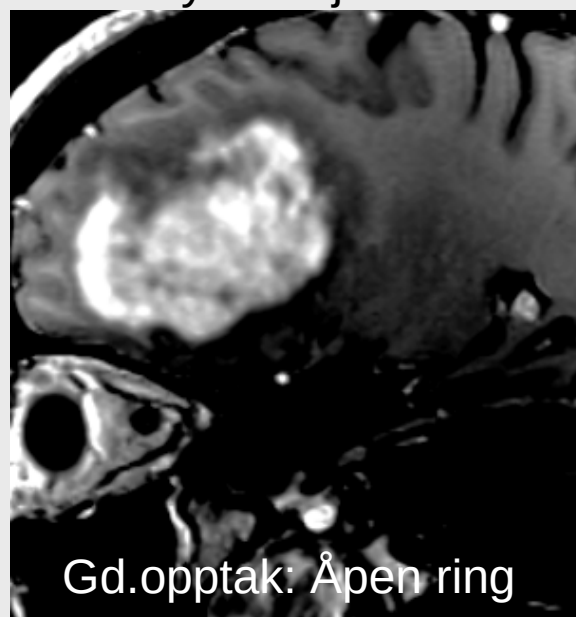


CBV

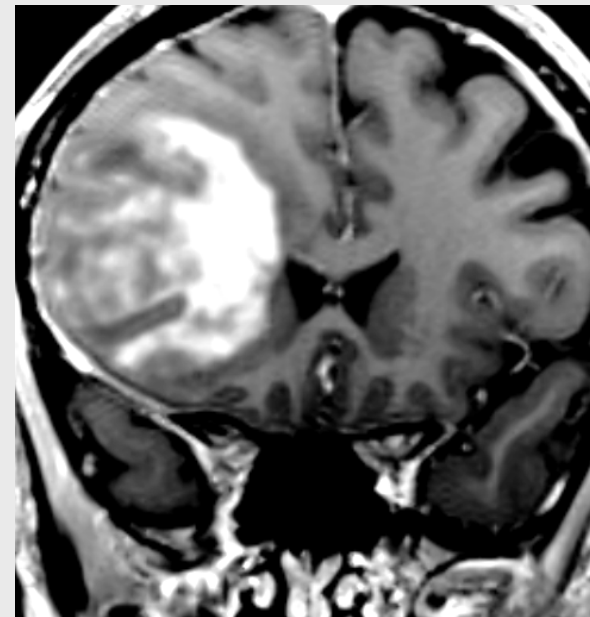
Lav CBV



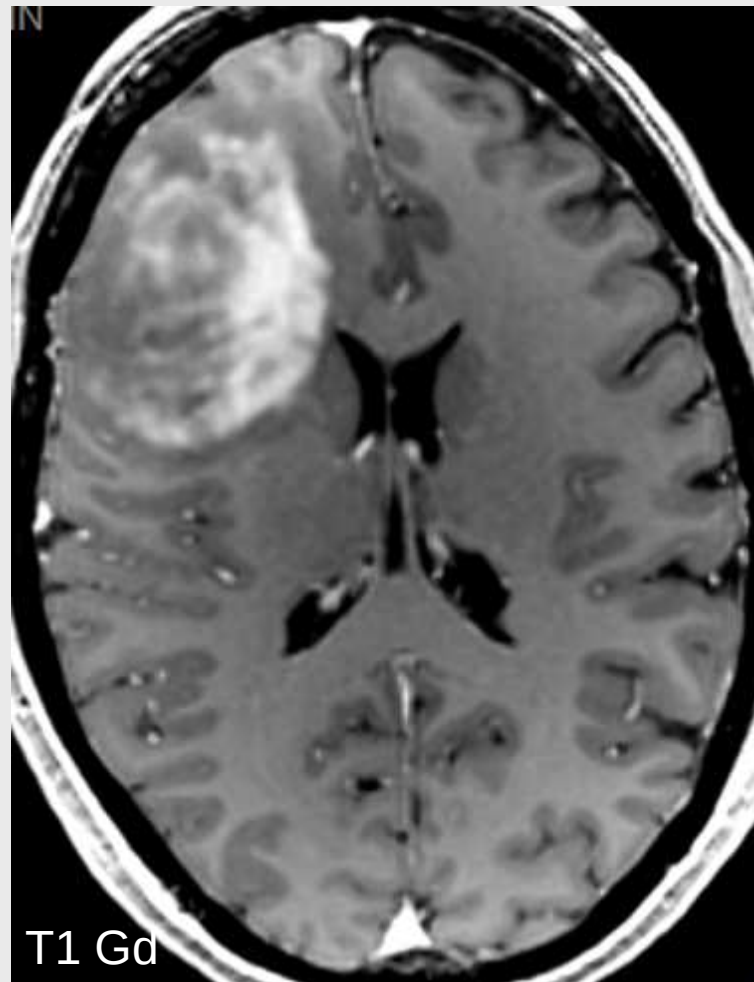
Høy cholin
 Lav NAA



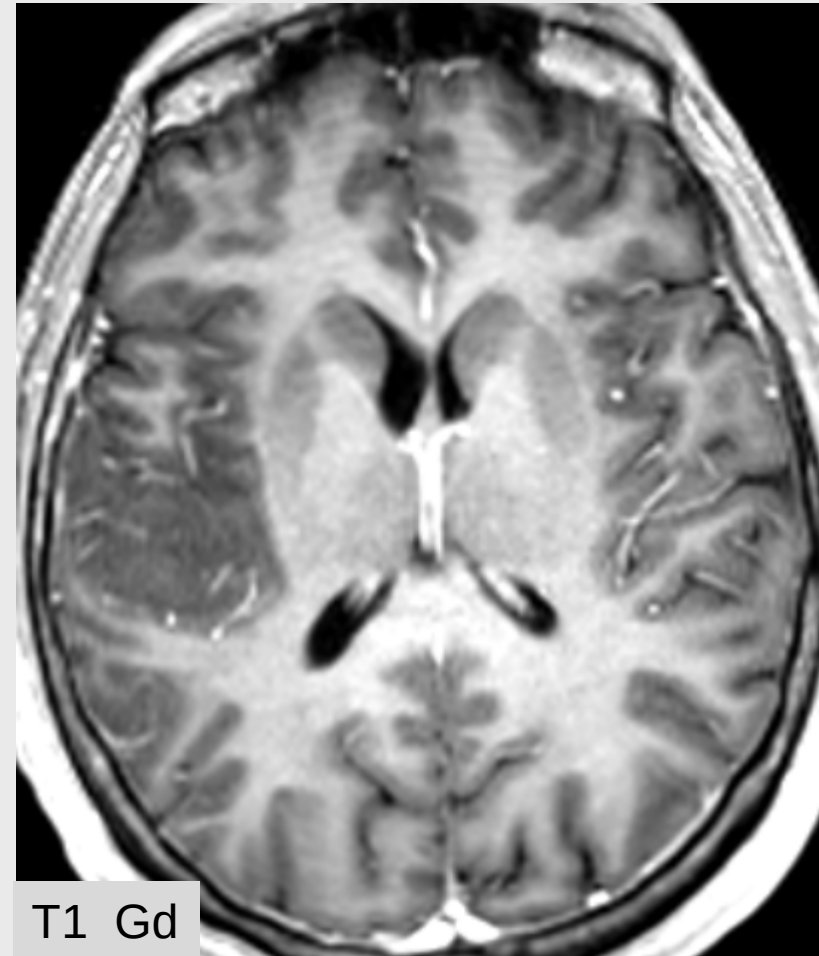
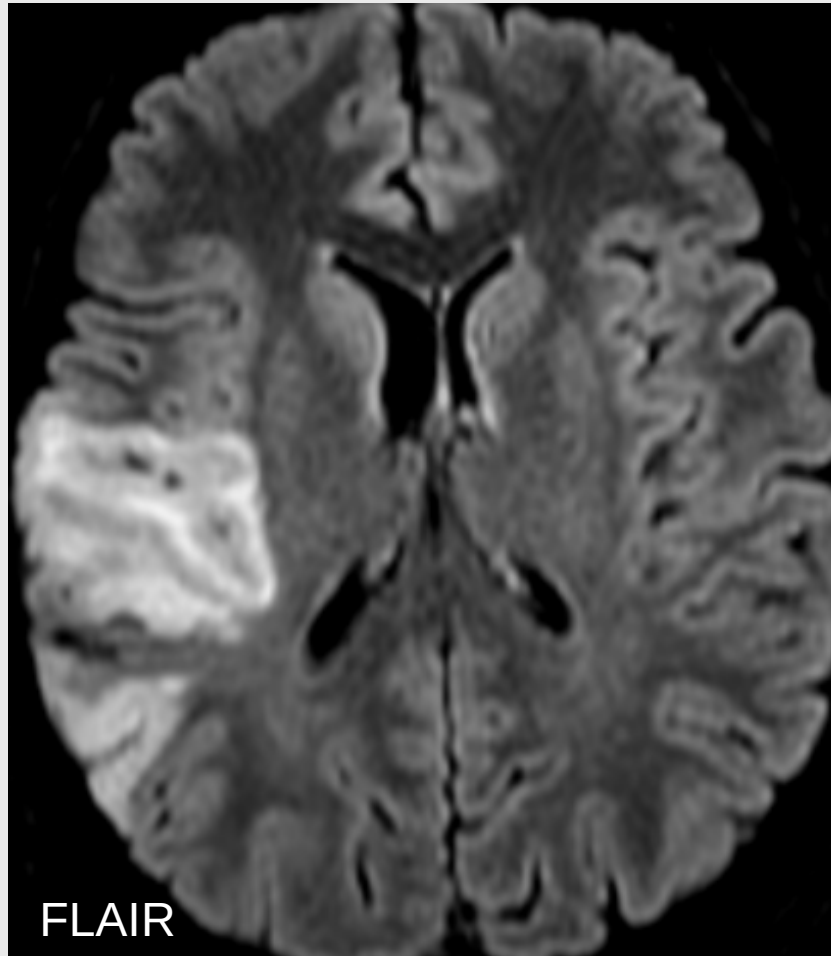
Gd.opptak: Åpen ring

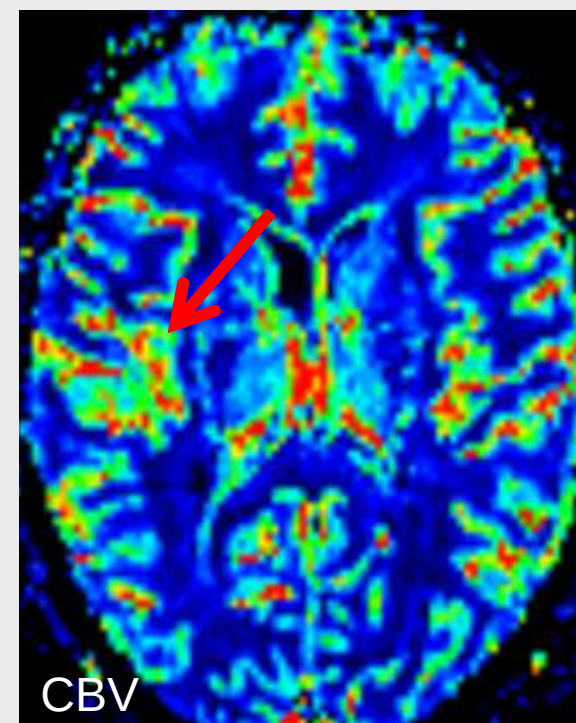
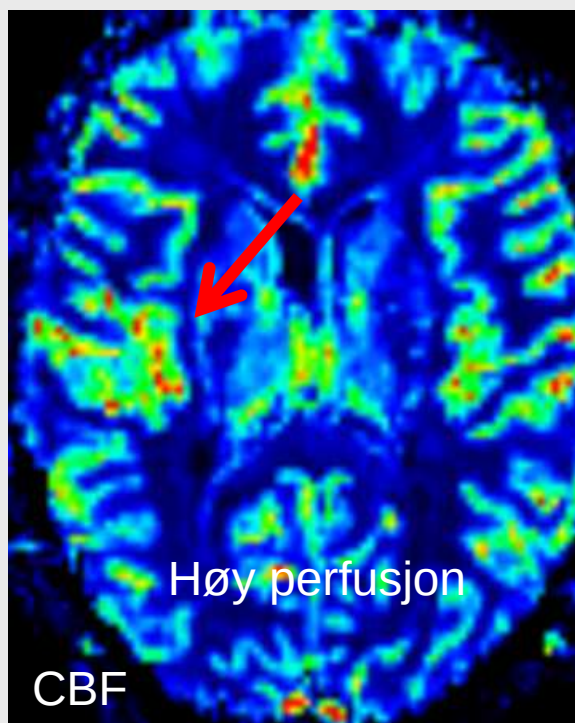
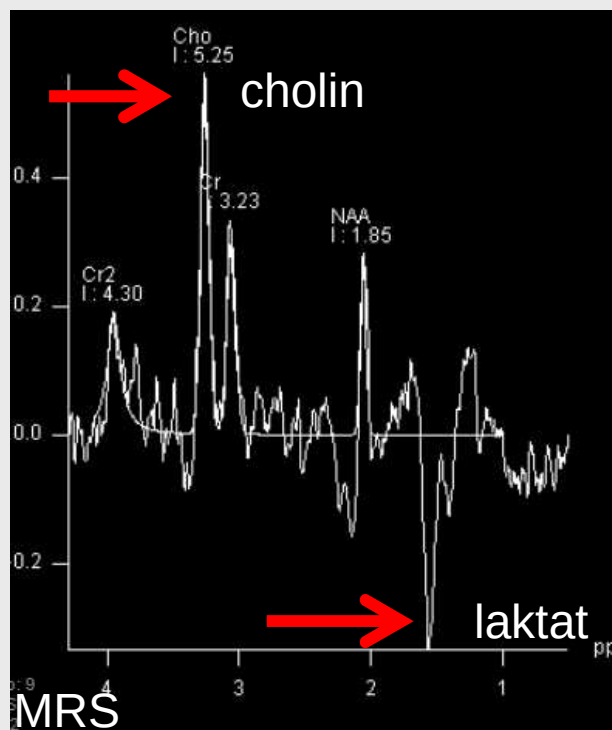
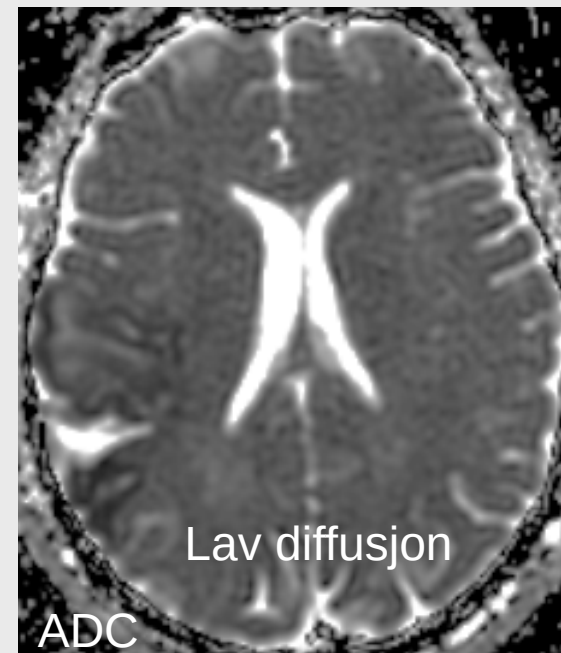
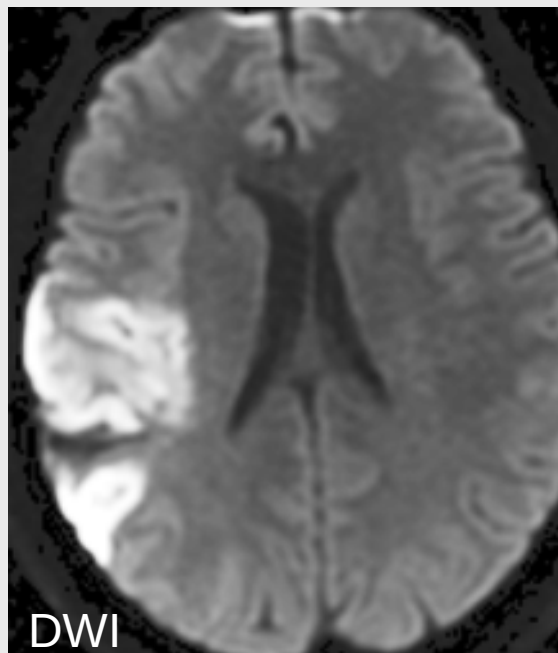
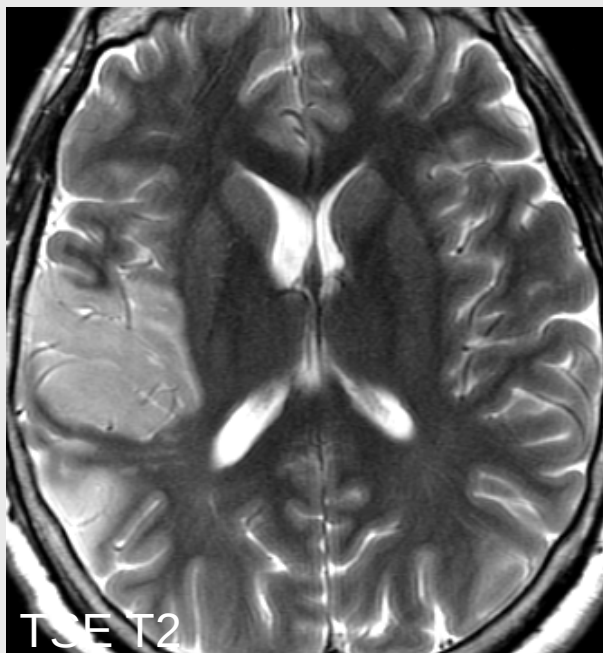


D: Tumefaktiv MS / ADEM (histologi)

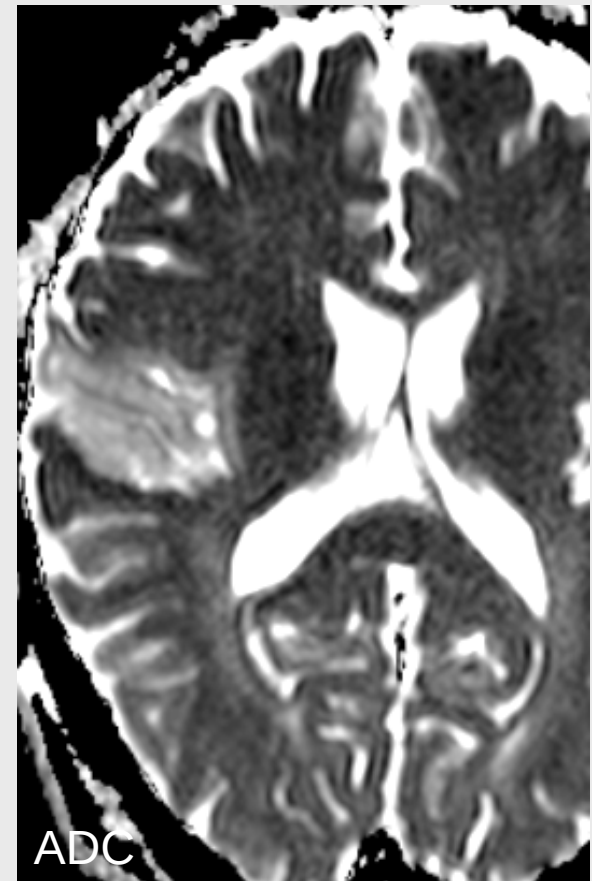
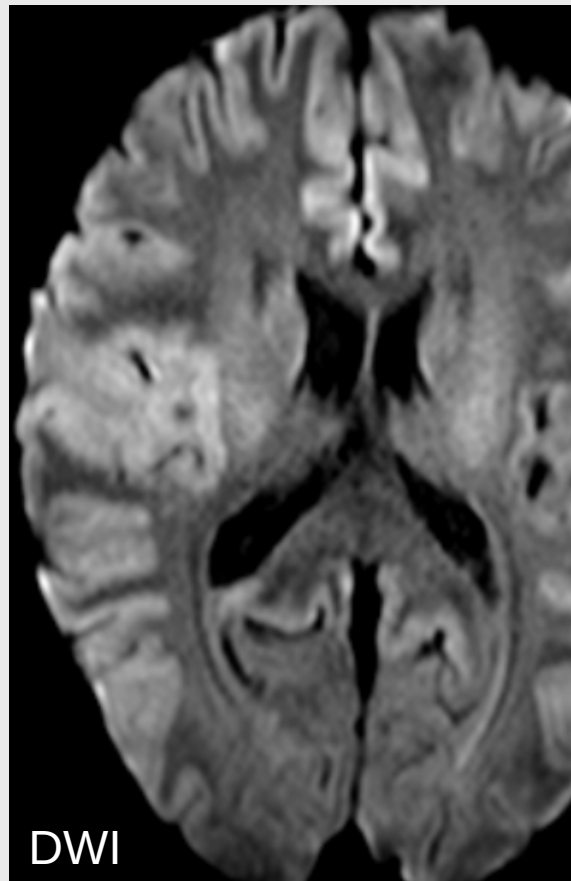
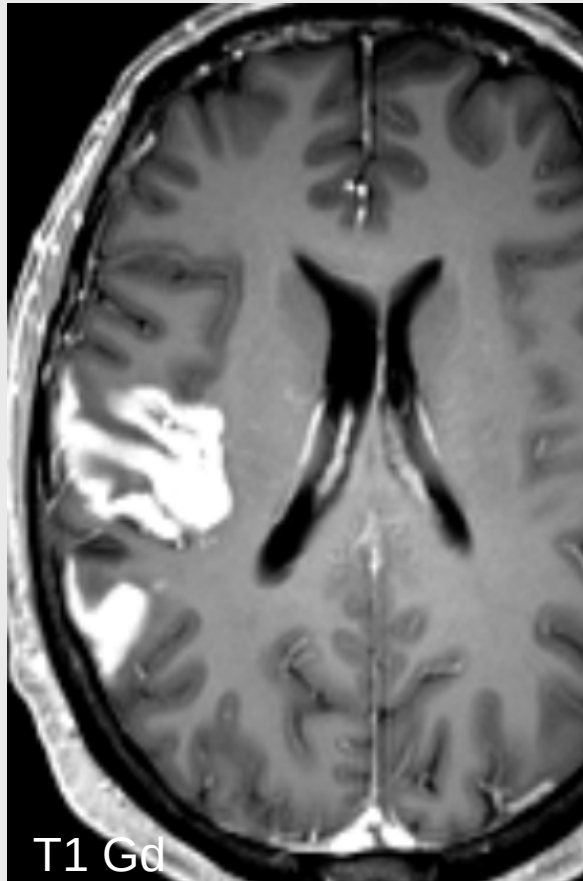


Hodepine og venstresidige parestesier de siste dagene



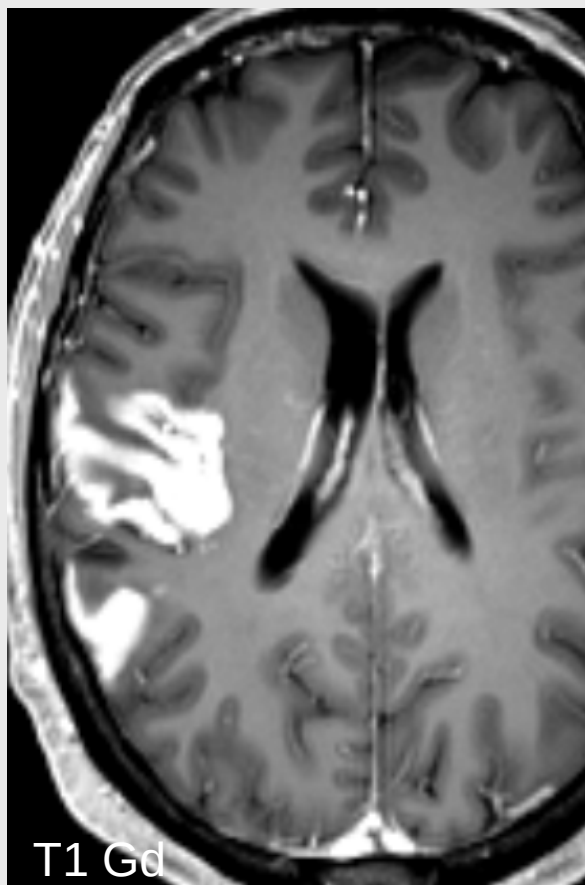


1 uke senere

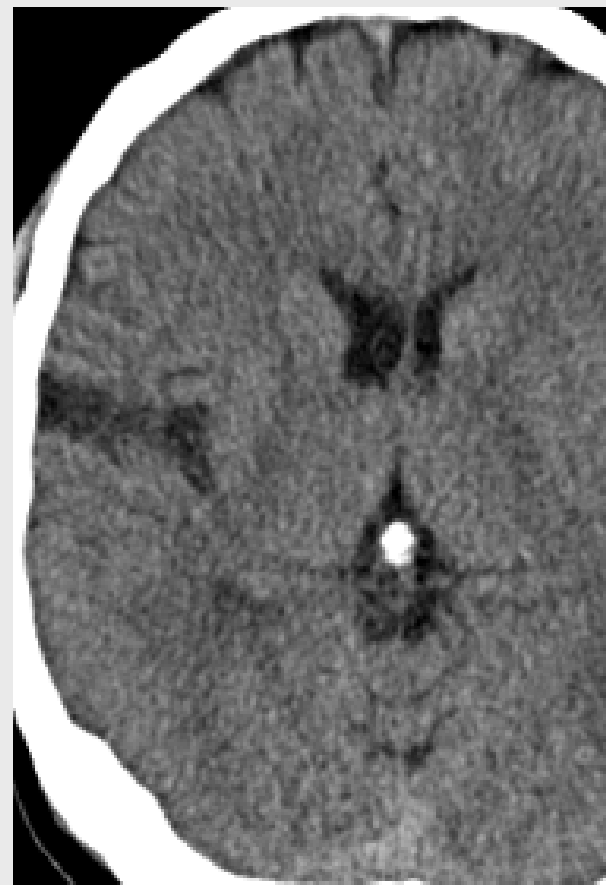
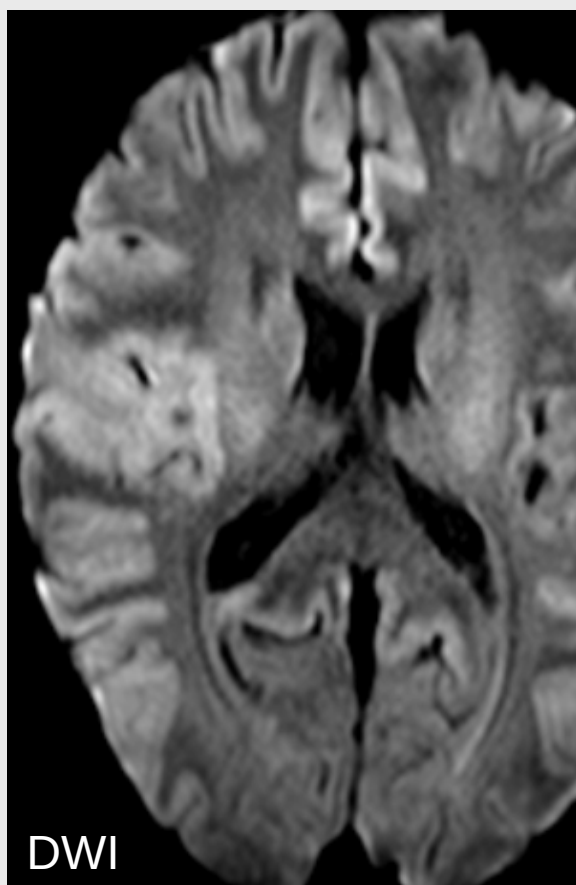


- Gd opptak i gyri
- Variabel DWI avhengig av alder
- Luksusperfusjon
- Raske endringer over tid

1 uke senere

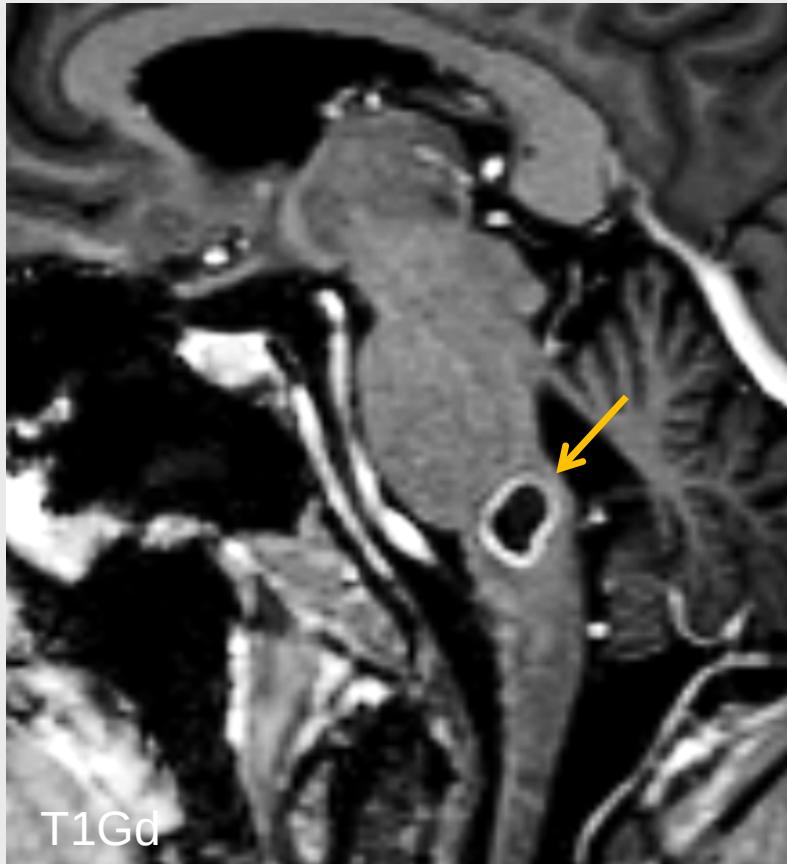


D: Subakutt infarkt



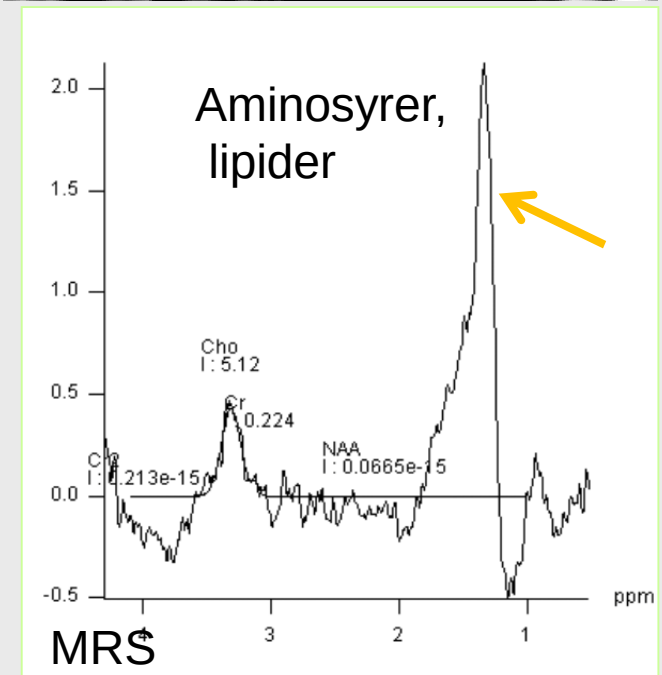
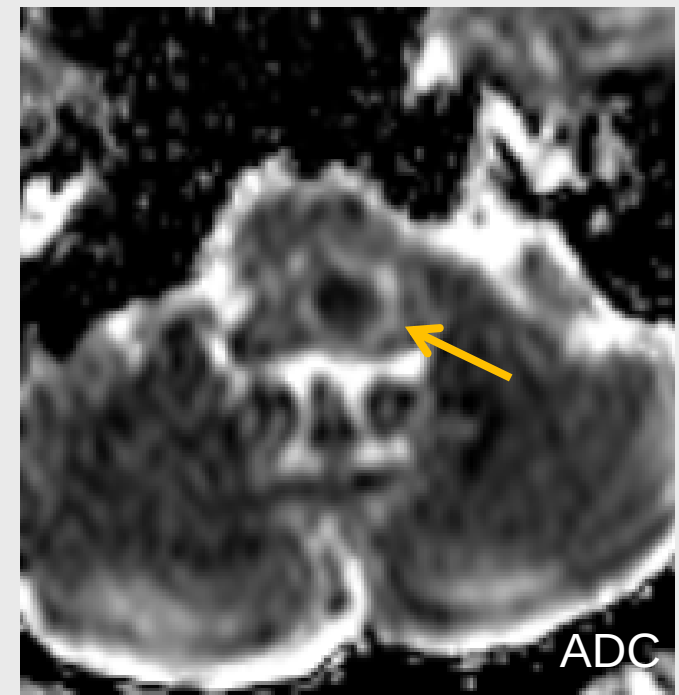
CT 1 mnd. senere

Venstresidig hemiparese.

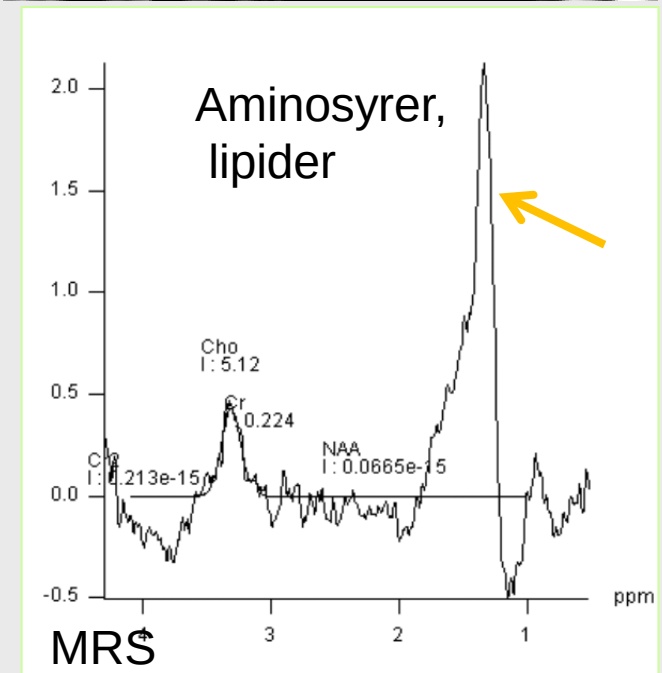
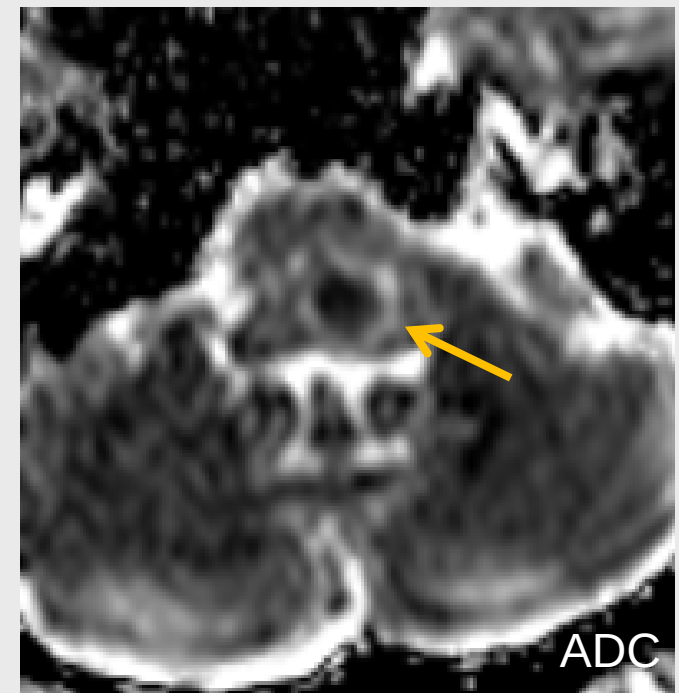


Abscess eller cystisk/nekrotisk tumor ?

- Ringformet, velavgrenset Gd opptak
- Lav diffusjon (ADC)
- Flere ringe på T2
- Aminosyrer og lipider

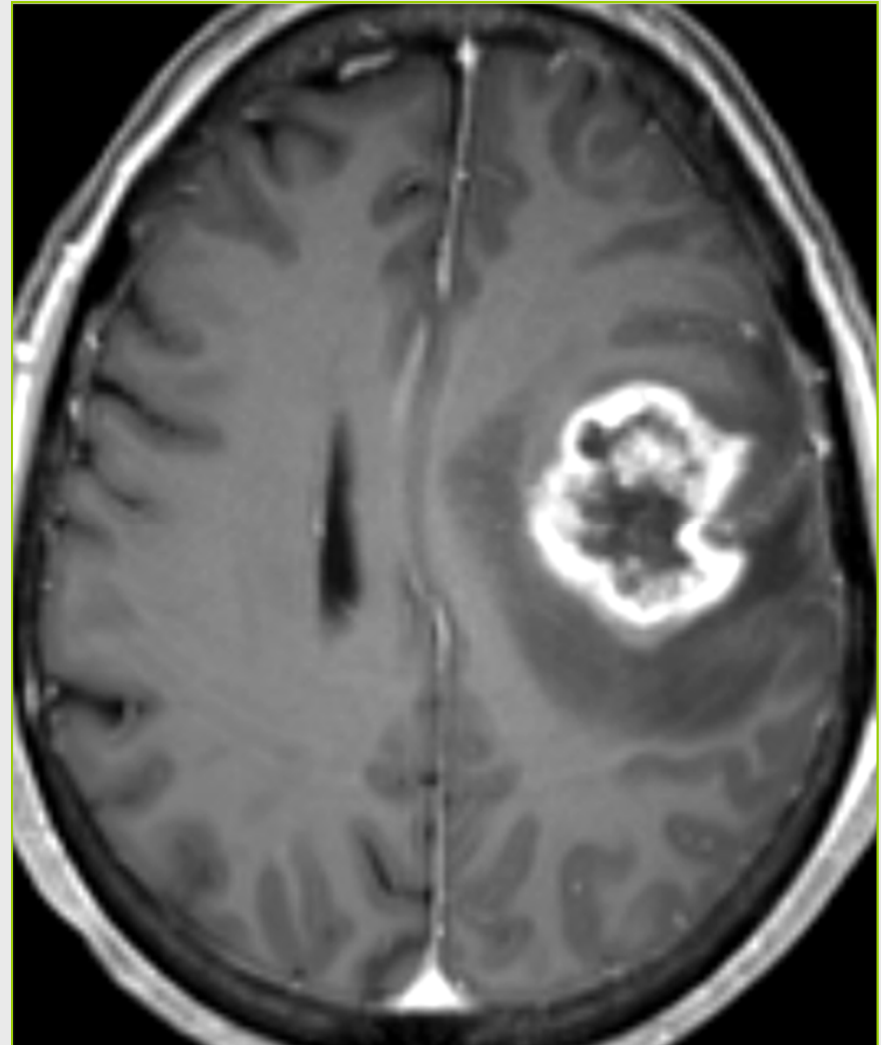


- Ringformet, velavgrenset Gd opptak
- Lav diffusjon (ADC)
- Flere ringe på T2
- Aminosyrer og lipider
- D: Abscess (nocardia)



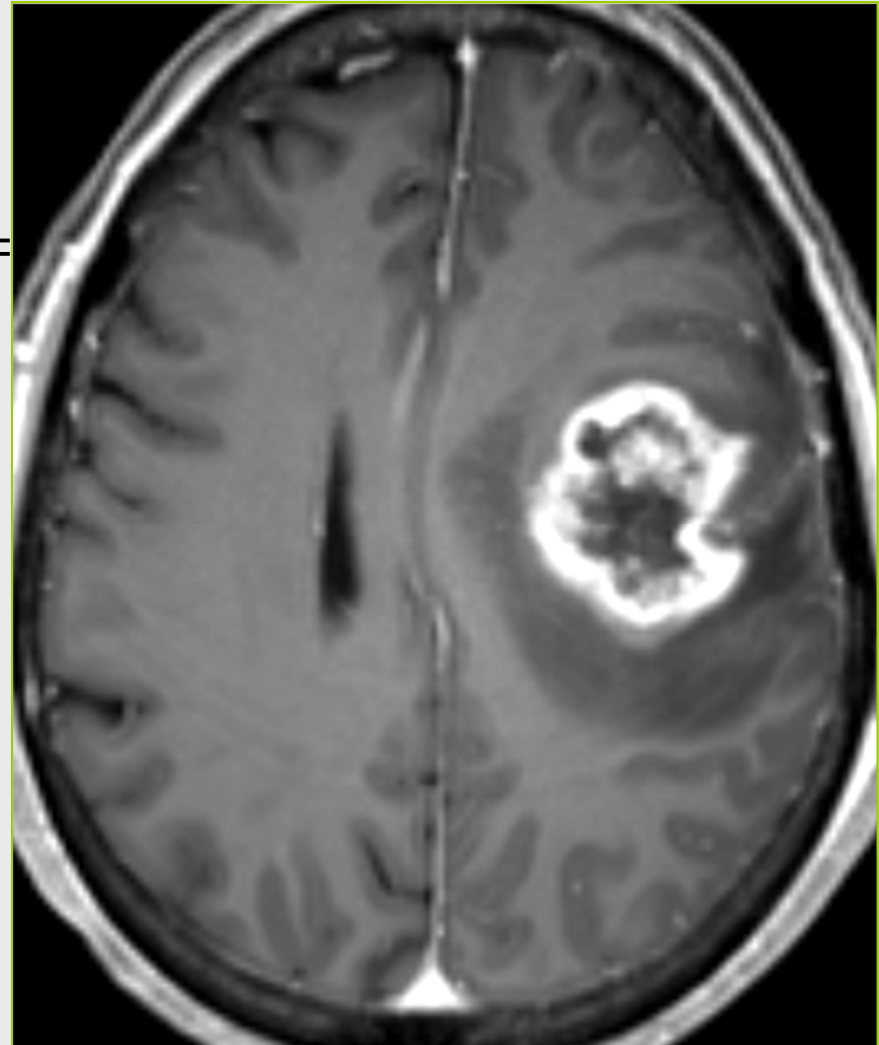
Er det tumorresidiv?

- Økende kontrastopptak og ødem etter kirurgi på malign hjernetumor = residiv



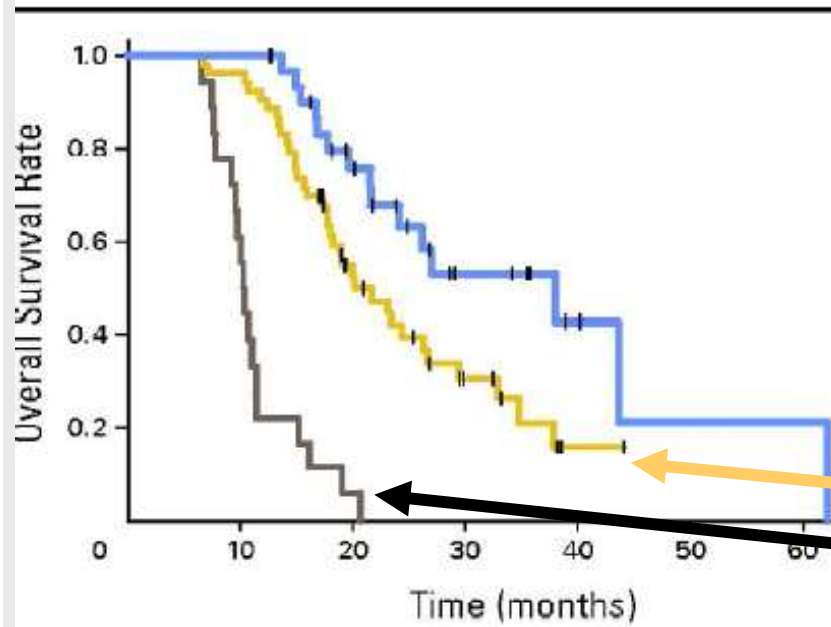
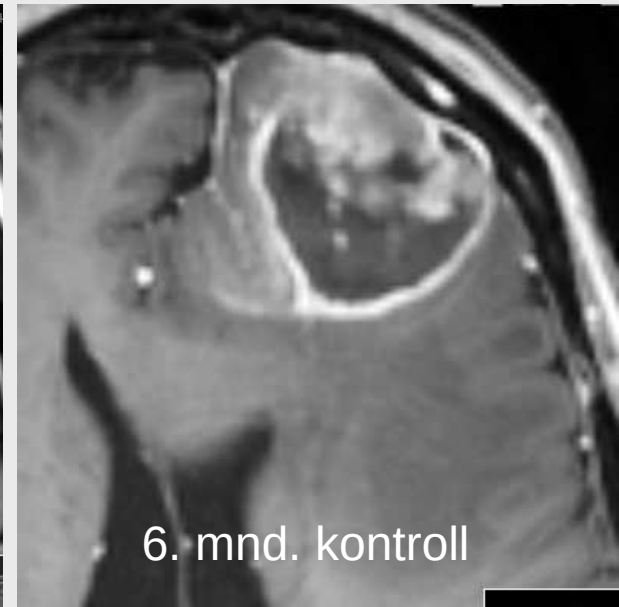
Er det tumorresidiv?

- Økende kontrastopptak og ødem etter kirurgi på malign hjernetumor = residiv
- Postoperativ behandling av maligne gliomer er kombinasjon av kjemo- (Temodal) og stråleterapi
- Ca. 30% får forandringer i karveggene som gir kontrastopptak og ødem = pseudoprogresjon
- Pseudoprogresjon oppstår oftest i løpet av de første 3 mnd etter avsluttet radiokjemoterapi



Hvorfor er det viktig?

- Pseudoprogresjon er behandlingseffekt
- Prognose ulik
- Behandling ulik
- Standard MR teknikker (FLAIR, T1 Gd) gir ikke svaret



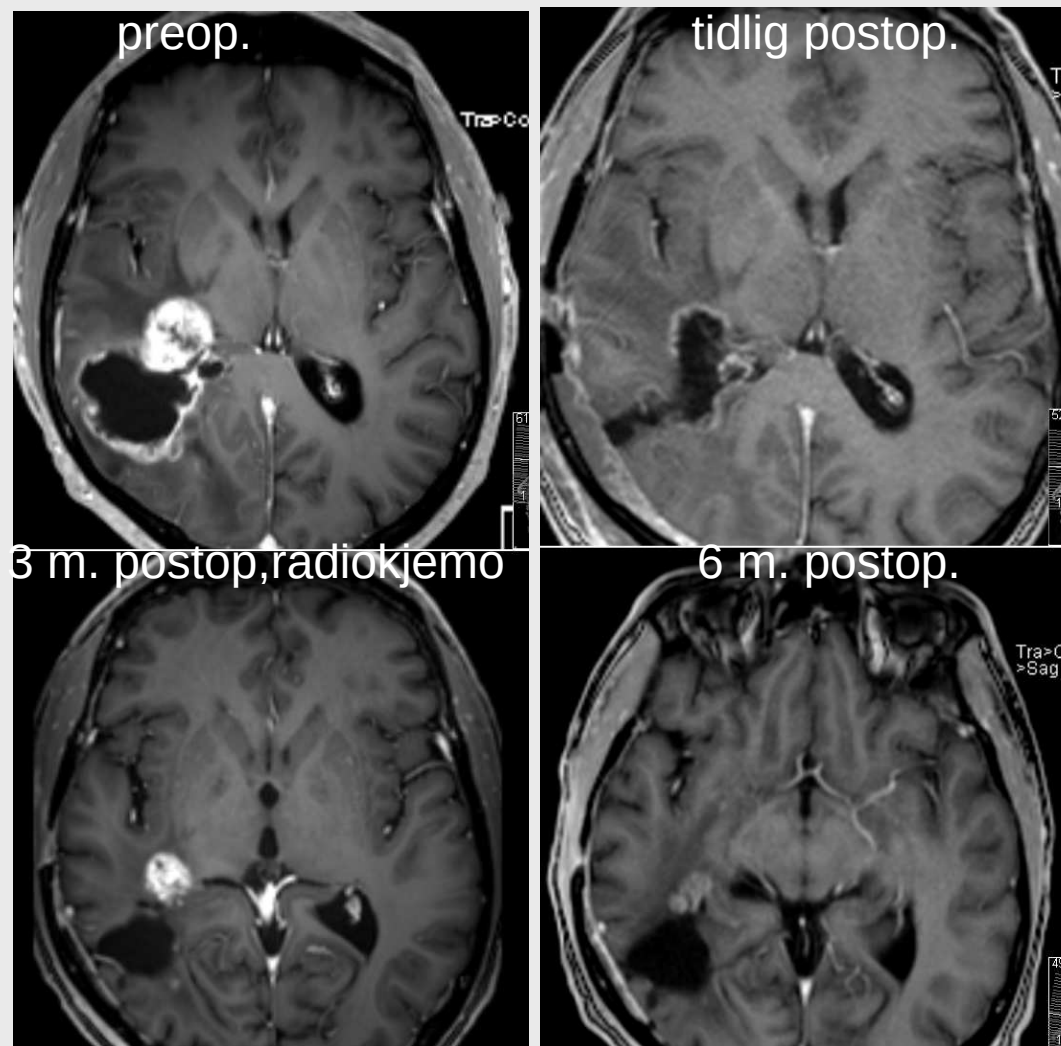
pseudoprogresjon

median overlevelse

reell tidlig progresjon

Hvordan differensiere mellom reell progresjon og pseudoprogresjon?

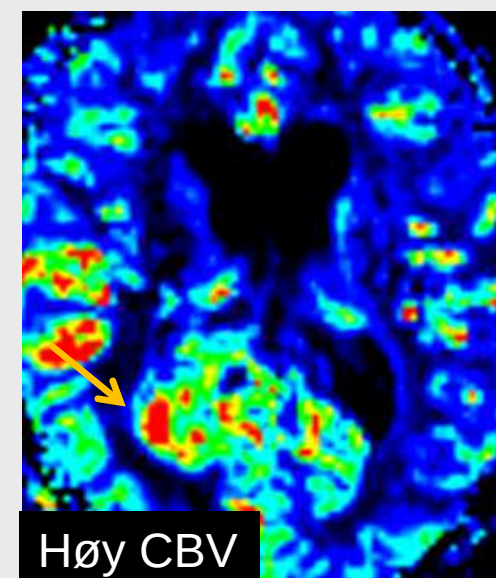
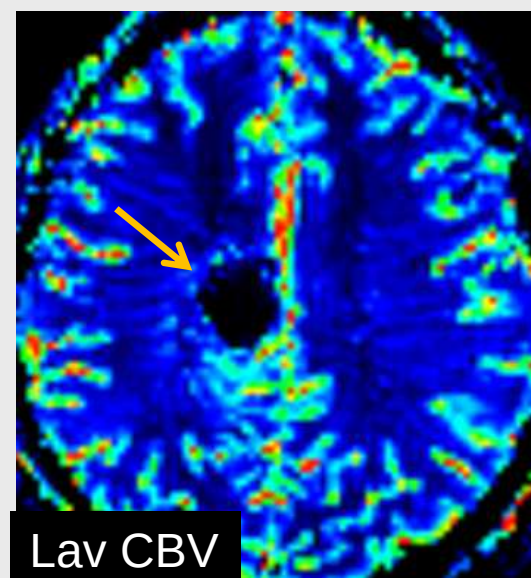
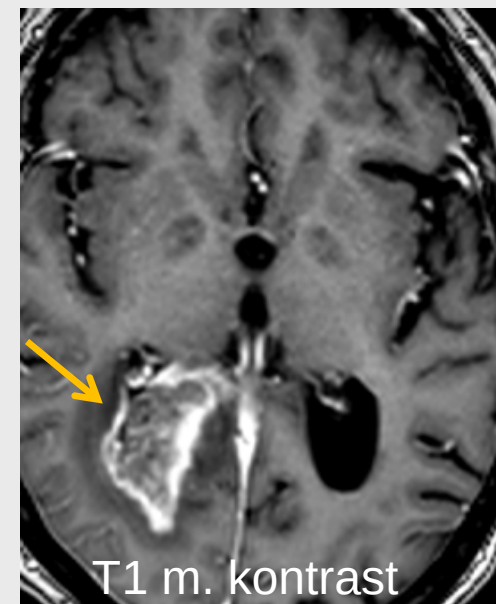
Tidsfaktoren



- Pseudoprogresjon typisk 1-3 mnd etter avsluttet radiokjemoterapi
- Reell progresjon vokser, pseudo- stabiliseres eller skrumper

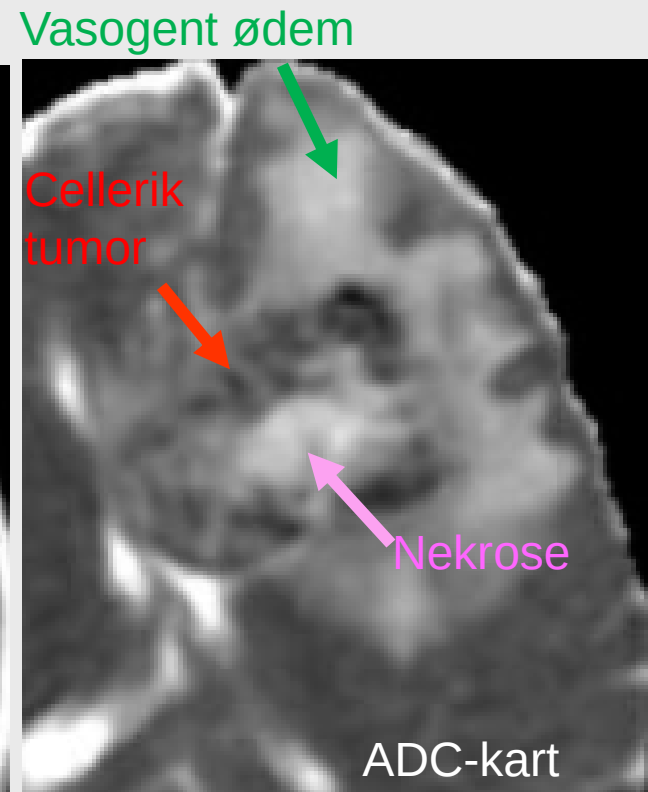
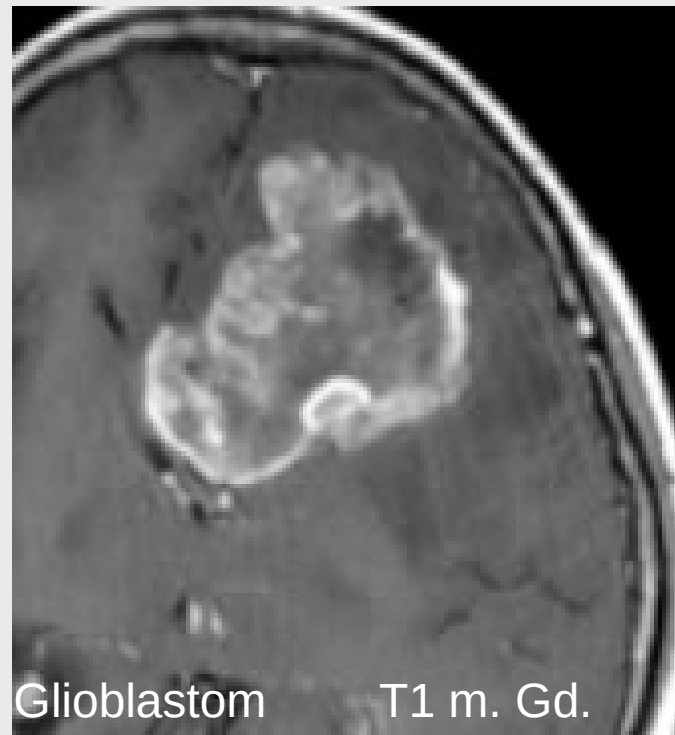
MR perfusjon

- CBV høy i tumor, lav i nekrose
- CBV  40% i pseudoprogresjon
- CBV  12% i reell progresjon

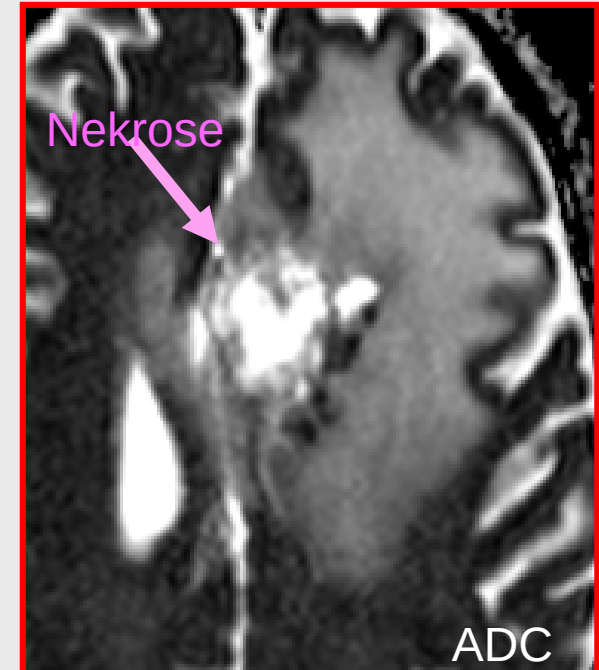
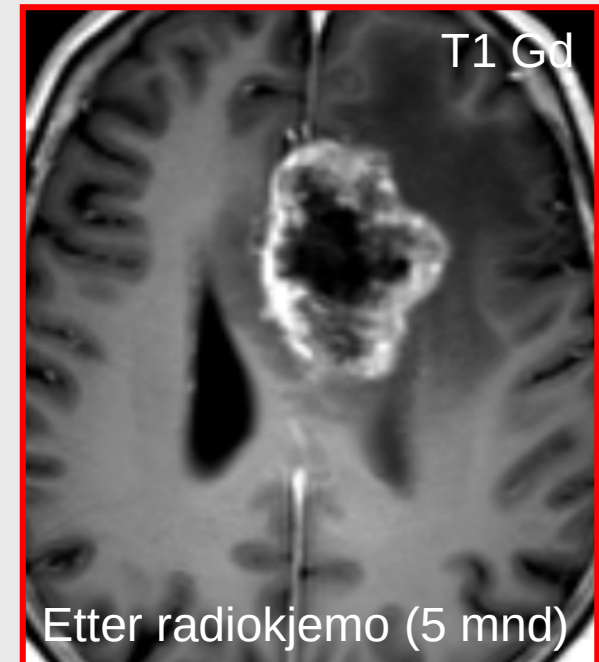
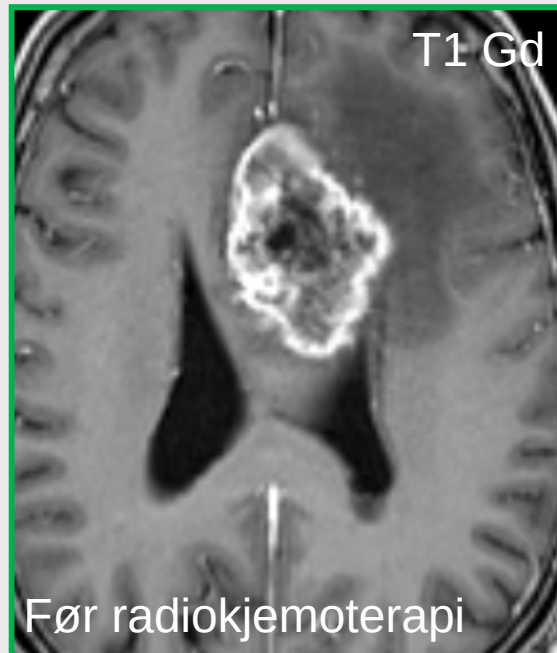


Diffusjon

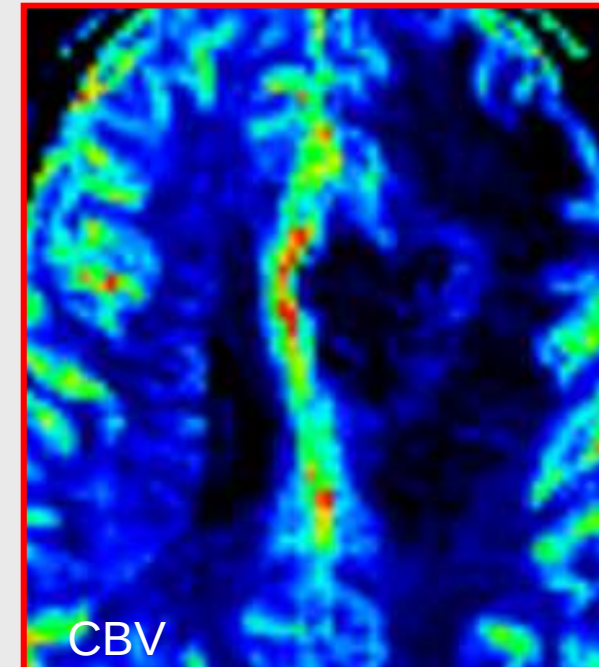
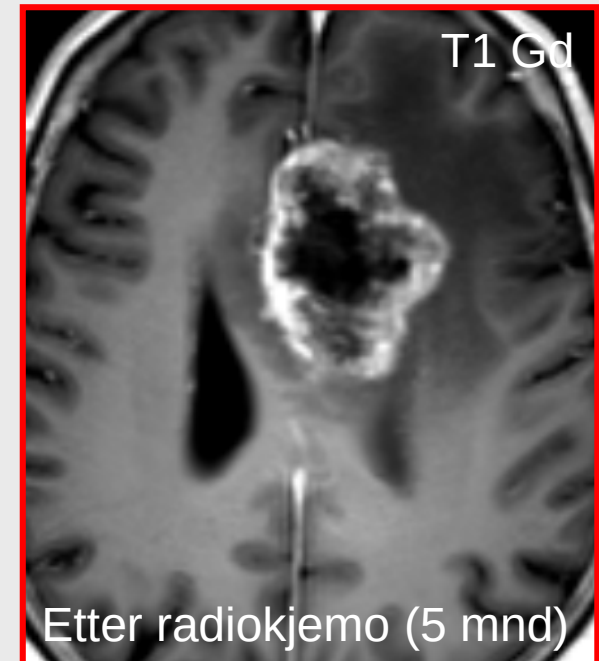
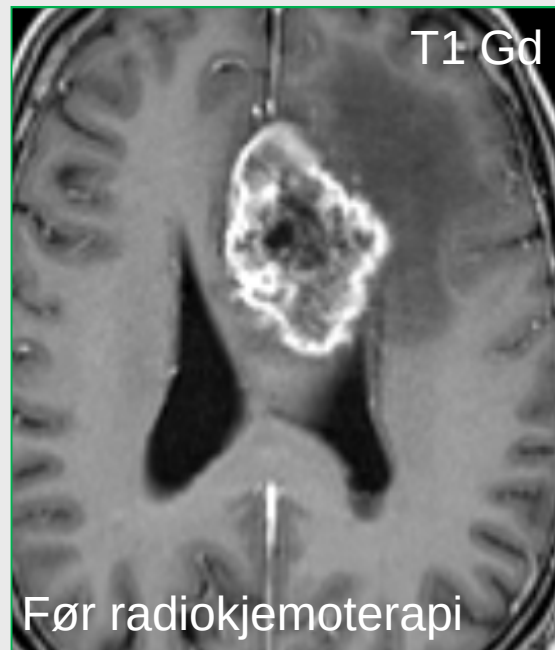
- Områder med høy cellularitet har lav diffusjon
- Peritumorødem (vasogent ødem) har høy diffusjon
- Nekrose har ofte høy diffusjon (avhengig av viskositet)



- Pseudoprogresjon har ofte høyere diffusjon enn reell progresjon pga lav celletetthet og nekrose



- Pseudoprogresjon har ofte høyere diffusjon enn reell progresjon pga lav celletetthet og nekrose



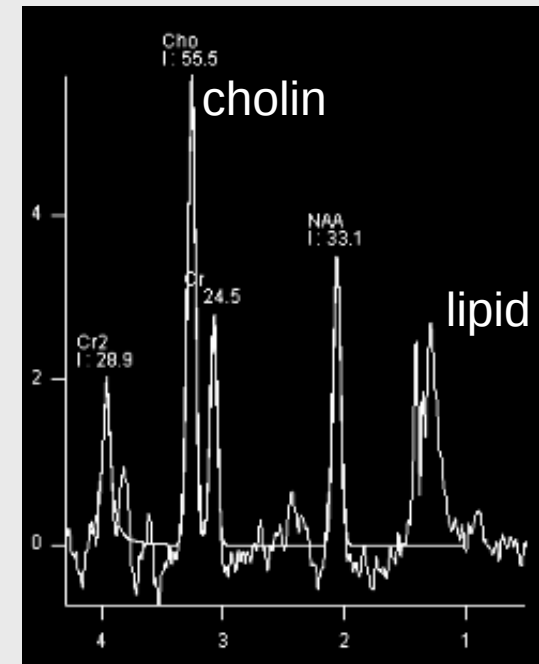
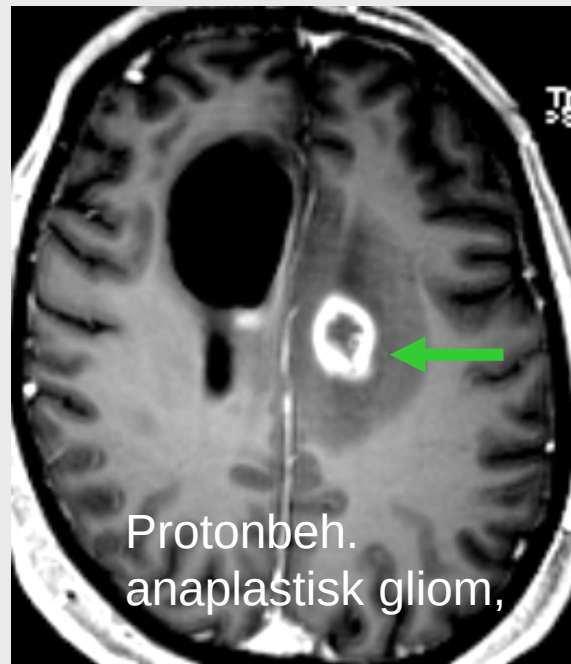
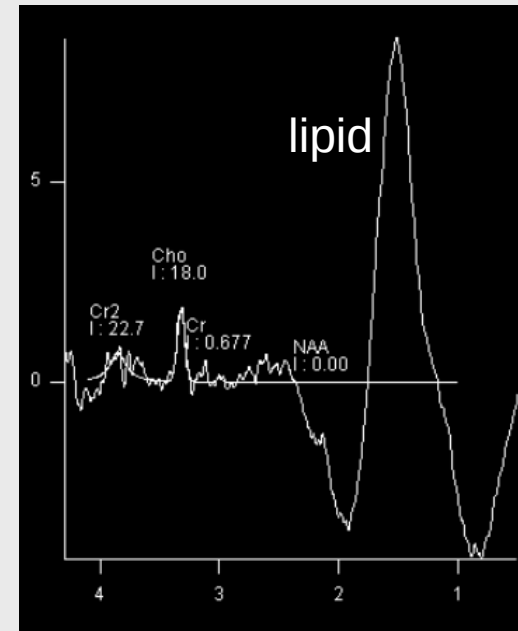
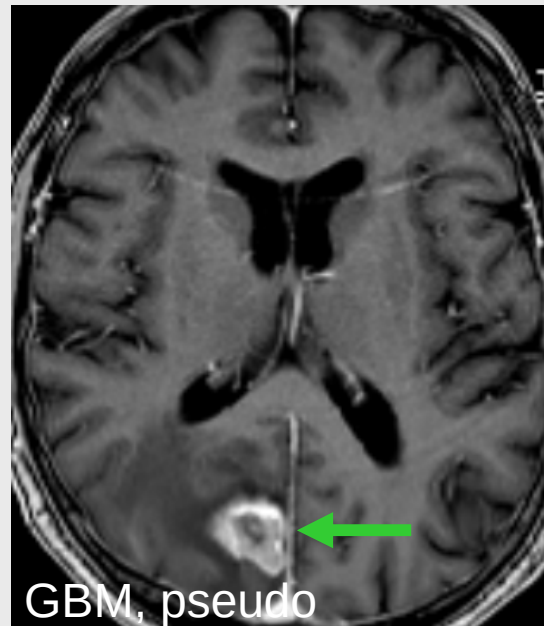
MR spektroskopi

I teorien:

- Tumor gir høy cholintopp
- Nekrose gir høy lipid og laktat

I virkeligheten:

Reell progresjon er en blanding av nekrose, inflammasjon og tumor som vanskelig lar seg skille fra pseudoprogresjon.



Oppsummering

- Mer aggressiv tumorkirurgi - histopatologisk diagnose hos de fleste
- Mindre behov for at radiologen skal gi en spesifikk tumordiagnose
- Det viktigste er **tumor** (kirurgi) eller **ikke-tumor** (medik.terapi /ekspektans)
- Det andre diagnostiske hovedproblemet er **tumorresidiv eller pseudoprogresjon.**
- Bruk flere MR teknikker, ikke stol for mye på en enkelt sekvens.

