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Case Report: Radiological Insights in Creutzfeldt - Jakob Disease

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ABSTRACT:

Creutzfeldt –Jakob Disease (CJD) is an exceptional, degenerative, and continually deadly brain disorder achieved by prion proteins that affect abnormal imploding of conventional frontal cortex proteins. This misfolding prompts mind damage and brand name aftereffects, including rapid moderate dementia, myoclonus, visual agitating impacts, and ataxia. Regardless of its special case, CJD presents basic expressive hardships due to its aftereffect get over with other neurodegenerative diseases. This case report looks at a 58-year-old patient with moderate mental deterioration, visual disrupting impacts, and direct changes more than a half year, highlighting the crucial occupation of MRI in diagnosing CJD. Key MRI revelations included diffusion restriction and T2/FLAIR hyperintensities in the separate corpus striatum, forward looking, parietal, and occipital cortices, with commonplace thalami, which immovably maintained the examination of CJD. These radiological encounters feature the meaning of forefront neuroimaging techniques in isolating CJD from various conditions and working with early finding and the leaders.

Keywords: Creutzfeldt –Jakob Disease, CJD, prion disease, rapid dementia, MRI, diffusion restriction, T2 FLAIR, neurodegenerative disorder.

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1. Introduction

Creutzfeldt –Jakob Disease (CJD) is a captivating, degenerative, and never-endingly dangerous psyche disorder achieved by prion proteins that actuate uncommon imploding of common frontal cortex proteins. This misfolding prompts mind hurt and the brand name results of CJD, which integrate rapid moderate dementia, myoclonus, visual aggravations, and ataxia. The disease propels rapidly, much of the time provoking outrageous neurological rot and death in something like a drawn out time of starting. Disregarding its exceptional case, CJD is an essential end to consider due to its tremendous impact on patients and the challenges it presents in differential finding (Johnson and Gibbs, 1998).

The clinical demonstration of CJD much of the time covers with other neurodegenerative diseases, making precise investigation testing. Early incidental effects can integrate mental degradation, lead changes, and visual aggravations, which can without a very remarkable stretch be mistaken for conditions like Alzheimer's disease or frontotemporal dementia. As the disease progresses, patients could encourage outrageous mental impedance, motor brokenness, and ultimately, akinetic mutism. These covering aftereffects require the use of state of the art demonstrative gadgets, including MRI, to isolate CJD from various conditions really (Zerr and Phony, 2002).

Appealing resonation imaging (MRI) accepts a fundamental part in the finding of CJD. Brand name MRI disclosures recollect diffusion restriction and hyperintensities for T2-weighted and FLAIR progressions in unambiguous psyche regions like the caudate center, putamen, and cerebral cortex. These disclosures, when related with clinical history and neurological evaluation, can vehemently suggest a finish of CJD. For this present circumstance report, we present the radiological disclosures of a 58-year-old patient who showed results of moderate mental degradation, visual clouding, and social changes more than a half year. The MRI frontal cortex focus on gave basic pieces of information that provoked the examination of likely CJD (Young et al., 2005).

Clinical Presentation

A 58-year-old patient gave a consistently developing reduction in mental capacity, depicted by immense mental degradation, visual disrupting impacts, and extraordinary social changes all through late months. The patient's family reported extending interruption, inconvenience in performing ordinary activities, and episodes of disorder. Likewise, the patient experienced episodes of darkened vision, which further tangled their ability to independently capacity. These secondary effects overall raised stresses over a possible neurodegenerative condition, instigating the family to search for clinical thought. Given the rapid development and reality of the secondary effects, an exhaustive neurological evaluation was viewed as important to perceive the secret explanation and to sort out a legitimate organization plan.

The patient's lead changes were particularly disrupting, as they included episodes of unsettling influence, bewilderment, and character changes that were disturbing for both the patient and their watchmen. These social secondary effects, got together with the psychological corruption and visual disrupting impacts, proposed a complex neurological disorder. To moreover investigate these secondary effects, a clear neurological evaluation was performed, uncovering deficits in higher mental capacities and anomalies in visual dealing with. Given the clinical show, the clinical gathering decided to go on with appealing resonation imaging (MRI) of the psyche. The MRI was essential in perceiving express neuroanatomical changes that could figure out the patient's secondary effects and separate between various likely examinations, similar to Alzheimer's disease, vascular dementia, or Creutzfeldt –Jakob Disease (CJD).

Radiological Findings**Diffusion Weighted Imaging (DWI)**

MRI mind concentrate on uncovered diffusion restriction and T2/FLAIR hyperintensity in a few locales:

- Bilateral corpus striatum
- Bilateral frontal cortex
- Bilateral parietal cortex
- Bilateral occipital cortex

Strikingly, the thalami seemed ordinary, which is a distinctive component with regards to diagnosing likely CJD.

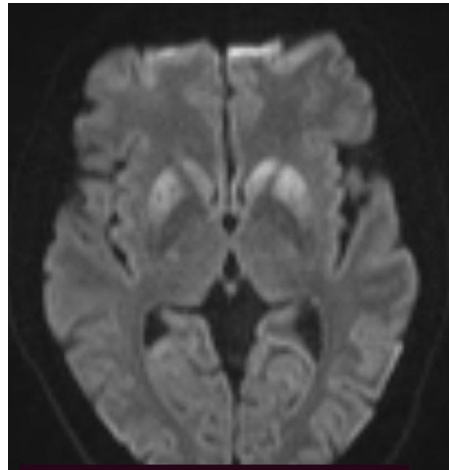


Figure 1: Diffusion Weighted Imaging (DWI) Sequence of Bilateral Corpus Striatum

The Diffusion Weighted Imaging (DWI) succession portrayed in Figure 1 uncovers huge diffusion restriction in the two-sided corpus striatum. This radiological finding is especially characteristic of Creutzfeldt –Jakob Disease (CJD) and supports the clinical doubt of this neurodegenerative disorder. In DWI, diffusion restriction shows up as hyperintense (splendid) locales, implying regions where water atom development is obstructed, frequently because of neurotic changes in the mind tissue. With regards to CJD, such diffusion restrictions are ordinarily seen in the basal ganglia, including the caudate core and putamen, which contain the corpus striatum. The diffusion restriction in the respective corpus striatum, as found in the picture, is a trademark element of CJD. This example of limited diffusion isn't regularly seen in other neurodegenerative diseases, making it an essential demonstrative standard. The presence of these hyperintense signals lines up with recently reported instances of CJD, where comparative imaging discoveries have been corresponded with the disease's rapid movement and serious clinical appearances. The radiological appearance in DWI gives a harmless technique to help the determination, directing further clinical administration and affirming the thought neurodegenerative pathology. In synopsis, the DWI grouping in Figure 1 really features the reciprocal diffusion restriction in the corpus striatum, a critical radiological marker that, joined with clinical discoveries, unequivocally recommends a conclusion of Creutzfeldt –Jakob Disease. This imaging methodology subsequently assumes a crucial part in separating CJD from other neurological circumstances, working with right on time and exact determination.

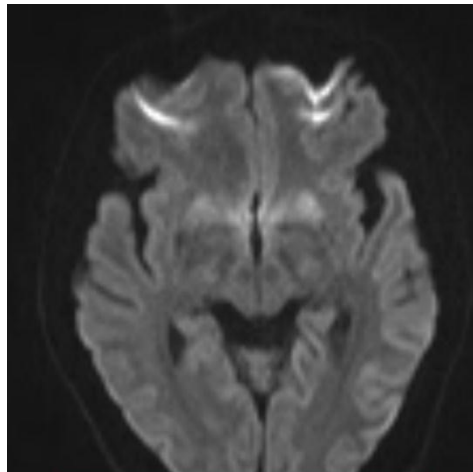


Figure 2: Diffusion Weighted Imaging (DWI) Sequence of Bilateral Frontal Cortex

Figure 2 presents a Diffusion Weighted Imaging (DWI) grouping displaying critical diffusion restriction in the reciprocal cerebrum. This finding further proves the determination of Creutzfeldt –Jakob Disease (CJD), as it lines up with the trademark radiological examples found in this uncommon and lethal neurodegenerative disorder. The presence of hyperintense signals in the cerebrum, demonstrating confined diffusion, mirrors the obsessive changes normally connected with CJD. This diffusion restriction is because of the aggregation of strangely collapsed prion proteins, which upset ordinary cell capability and cause neuronal demise. The inclusion of the cerebrum is especially imperative as it corresponds with the patient's clinical show of moderate mental deterioration, cognitive decline, and social changes. These side effects are frequently connected to cerebrum brokenness, which is obvious in the MRI discoveries. The two-sided nature of the diffusion restriction saw in the cerebrum underscores the far and wide and forceful nature of CJD. Dissimilar to other neurodegenerative circumstances that might give more limited or one-sided changes, CJD frequently shows respective and symmetric association on MRI, helping with its separation from different analyses. The imaging discoveries in Figure 2, when thought about close by clinical side effects, give undeniable proof supporting the determination of CJD. The utilization of DWI is especially important in this unique situation, as it can distinguish early changes in cerebrum tissue that probably won't be apparent on other MRI arrangements. This makes DWI a basic device in the early finding of CJD, empowering convenient mediation and the executives of the disease. The predictable appearance of diffusion restriction across various mind districts, including both the corpus striatum and the cerebrum, features the utility of MRI in following the movement and degree of CJD pathology. Subsequently, the DWI grouping in Figure 2 is an essential part in the far reaching radiological evaluation of this patient's condition.

Apparent Diffusion Coefficient (ADC) Mapping

The ADC planning uncovered relating low qualities in districts showing diffusion restriction, especially in the respective corpus striatum.

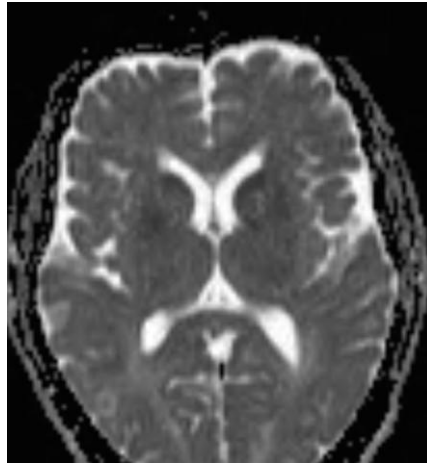


Figure 3: Apparent Diffusion Coefficient (ADC) Sequence of Bilateral Corpus Striatum

Figure 3 represents the Clear Diffusion Coefficient (ADC) grouping, which uncovers low qualities in the two-sided corpus striatum. This finding is predictable with the diffusion restriction saw in the DWI groupings, further proving the analysis of Creutzfeldt –Jakob Disease (CJD). In MRI studies, ADC maps are gotten from DWI information and give quantitative proportions of water diffusion inside tissue. Low ADC esteems normally demonstrate areas of confined diffusion, frequently because of cell expanding or cytotoxic edema related with prion-related neurodegeneration in CJD. The low ADC values found in the two-sided corpus striatum feature the degree of prion-actuated harm in these areas. The corpus striatum, which incorporates the caudate core and putamen, is essential for engine control and mental capabilities. Harm to these areas can appear as the rapid mental deterioration, memory disability, and engine irregularities oftentimes saw in CJD patients. The ADC succession consequently fills in as a significant supplement to DWI, giving a more itemized and quantitative evaluation of the diffusion irregularities. These imaging discoveries are especially significant for clinicians in affirming the analysis of CJD. The connection between's low ADC values and diffusion restriction upholds the presence of far and wide neuronal harm, a sign of this prion disease. Also, the respective association seen on the ADC map lines up with the trademark even example of mind pathology in CJD, recognizing it from different circumstances that might give more topsy-turvy discoveries.

T2/Flair Imaging

The T2/FLAIR groupings showed hyperintensities in the respective caudate core and putamen, which are characteristic of neurodegenerative changes related with CJD.

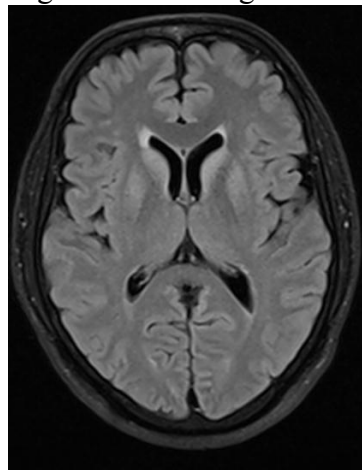


Figure 4: MRI T2 FLAIR Sequence of Bilateral Caudate Nucleus and Putamen

Figure 4 presents the MRI T2 FLAIR (Liquid Lessened Reversal Recuperation) arrangement, uncovering hyperintensities in the respective caudate core and putamen. These hyperintense signals are demonstrative of obsessive changes in these locales, usually connected with Creutzfeldt –Jakob Disease (CJD). The T2 FLAIR grouping is especially helpful in featuring anomalies in mind tissue by stifling the cerebrospinal liquid sign, accordingly improving the perceivability of sores and other obsessive changes. The hyperintensities saw in the caudate core and putamen propose the presence of spongiform changes and gliosis, which are trademark highlights of CJD. These progressions result from the amassing of misfolded prion proteins, prompting neuronal misfortune and spongiform degeneration. The reciprocal and symmetric example of these hyperintensities lines up with the common radiological show of CJD, further supporting the determination. The contribution of the caudate core and putamen is critical, as these designs are fundamental to engine and mental capabilities. Obsessive changes here can show clinically as rapid mental degradation, conduct changes, and engine side effects, which were all seen in the patient. The T2 FLAIR grouping in this manner gives basic physical and obsessive experiences that connect with the clinical show. The MRI T2 FLAIR grouping portrayed in Figure 4 upgrades the symptomatic exactness for CJD by exhibiting trademark hyperintensities in the reciprocal caudate core and putamen. These discoveries, related to diffusion restrictions and low ADC values saw in past imaging groupings, give a far reaching radiological profile that upholds the determination of CJD. The utilization of T2 FLAIR imaging is fundamental in identifying unpretentious changes in cerebrum tissue, working with ahead of schedule and exact determination of this rapidly moderate and lethal neurodegenerative disease.

2. Discussion

Pathophysiology of CJD

Creutzfeldt –Jakob Disease (CJD) is a prion disease depicted by the misfolding of prion proteins, which in this way prompt peculiar imploding of ordinary cell proteins inside the brain. This fanatical connection prompts basic frontal cortex hurt, showing up as the brand name results of CJD, including rapid moderate dementia, myoclonus, and ataxia. The prion proteins assemble, molding aggregates that steamed customary cell ability and lead to neuronal passing. This steadily developing neurodegeneration is clear through expansive spongiform changes, gliosis, and neuronal mishap saw in affected mind tissues. The disease exists in sporadic, procured, and acquired structures, with the unpredictable variety being the most widely recognized. Unpredictable CJD addresses generally 85% of cases and usually gives rapid start and development of secondary effects, regularly completing in serious neurological rot and end in something like a period of starting (Johnson and Gibbs, 1998).

Definite Imaging

Appealing Resonation Imaging (MRI) is an indispensable gadget in the finding of CJD, offering fundamental encounters into the essential and valuable abnormalities in the psyche. Brand name MRI revelations in CJD recall diffusion restriction and hyperintensities for T2-weighted and FLAIR (Fluid Tightened Inversion Recovery) plans in unambiguous brain regions like the caudate center, putamen, and cerebral cortex. These imaging features are expressive of the spongiform changes and gliosis related with prion diseases. The presence of diffusion restriction, appearing as hyperintense districts on DWI (Diffusion Weighted Imaging) plans, reflects areas of cytotoxic edema and neuronal disaster. Famously, the thalami as often as possible appear to be regular in CJD, remembering it from other neurodegenerative disorders where thalamic affiliation is more recognizable. This instance of imaging revelations, when compared with the patient's clinical history and neurological evaluation, decidedly proposes an

assurance of CJD. Undeniable level MRI systems, including ADC (Apparent Diffusion Coefficient) arranging, give quantitative assessments that help the diffusion restriction saw on DWI, further working on decisive accuracy (Zerr and Extortion, 2002).

Differential End

The differential end for rapidly moderate dementia wraps an extent of neurodegenerative and prion diseases. Alzheimer's disease, depicted by moderate mental crumbling and memory shortcoming, is a commonplace differential assurance anyway routinely misss the mark on unquestionable MRI disclosures tracked down in CJD. Frontotemporal dementia gives early friendly and character changes yet doesn't show the diffusion restriction and hyperintensities in the basal ganglia found in CJD. Vascular dementia, coming about due to cerebrovascular disease, may show infarcts or white matter changes on MRI, which are unquestionable from the models tracked down in CJD. Lewy body dementia, related with visual dreams and parkinsonism, may have MRI disclosures of reduced substantia nigra volume anyway not the all over cortical and basal ganglia changes typical for CJD. Other prion diseases, similar to variety CJD, can give covering secondary effects yet every now and again incorporate different areas of the frontal cortex, similar to the thalamus. The unquestionable MRI disclosures for this present circumstance, particularly the diffusion restriction in the separate corpus striatum and hyperintensities in the caudate center and putamen, got together with the clinical history of rapid mental corruption, visual aggravations, and social changes, solidly suggest a finding of CJD (Energetic et al., 2005).

3. Conclusion

This case features the basic significance of attractive reverberation imaging (MRI) in the early and precise conclusion of Creutzfeldt –Jakob Disease (CJD). The trademark radiological discoveries, like diffusion restriction in the two-sided corpus striatum and hyperintensities in the respective caudate core and putamen, are crucial for recognizing CJD from other neurodegenerative disorders. These MRI highlights, when connected with the clinical history and neurological assessment, give a hearty structure to diagnosing this deadly prion disease. Early analysis is fundamental for suitable patient administration, advising, and thought of restorative mediations, notwithstanding the ongoing absence of remedial medicines for CJD. The extensive radiological evaluation, as shown for this situation, builds up the utility of cutting edge neuroimaging procedures in distinguishing and understanding prion diseases, subsequently working with ideal and exact clinical direction.

4. References

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