FACULTAD DE MEDICINA
UNIVERSIDAD DE BARCELONA

EL DETERIORO COGNITIVO EN LOS ENFERMOS ALCOHOLICOS

Trabajo presentado para la obtención del grado de doctor

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Enero 1989

apreciado asociación con la presencia de antecedentes de deliriums o blackouts. Las contusiones craneales, las antecedentes de alteración perinatal, de la primera infancia o las enfermedades físicas de la edad adulta incluida la valoración bioquímica del estado hepático no han demostrado ser factores determinantes del defecto neuropsicológico.

La repercusión del consumo abusivo de alcohól es notablemente diferente de unos pacientes a otros. En algunos el deterioro es muy marcado, pero en otros sus rendimientos se sitúan en la zona de la normalidad. El análisis de clusters ha permtido definir empíricamente tres grupos bien diferenciados. El primero (28.4%) se caracteriza por una intensa afectación de las funciones abstractivas e integrativas con preservación de los rendimientos mnésicos, el segundo (30.4%) sobre un trasfondo de deterioro general ligero destacan los problemas de memoria, mientras que el tercero (41.2%) mantiene unos rendimientos intelectuales situados dentro de la normalidad.

El análisis longitudinal de un grupo de 27 pacientes que persistieron en la abstinencia a lo largo de un año permite apreciar una mejoría generalizada de sus logros en las pruebas de la betería neuropsicológica, pero su magnitud no alcanza la significación estadística. Es importante destacar que este grupo presentaba ya inicialmente un perfil medio más conservado, lo que sugiere que la ausencia de deterioro es un indicador de buen pronóstico para la persistencia en el

tratamiento. Además, si el rendimiento no se halla inicialmente afectado tampoco es esperable que se produzcan incrementos posteriores.

Después de un año de abstinencia los perfiles neuropsicológicos de los enfermos etílicos abstinentes no se diferencia significativamente de los controles normales. Pero ello no significa que en este periodo se haya normalizado el rendimiento en todos los sujetos, sino que a la mejoría se suma un efecto de selección por el que los pacientes deteriorados tienen más probabilidades de recaer en el consumo y, por tanto, ser excluidos del grupo abstinente

4.2 INTEGRACION DE LOS RESULTADOS EN UN MODELO TEORICO

Los resultados de los diferentes análisis hasta ahora realizados señalan la presencia de un patrón de deterioro en los enfermos etílicos con una abstinencia comprendida entre uno y dos meses cuyas características son coincidentes con las descritas en la bibliografía internacional. Desde una prespectiva de rendimientos absolutos los déficits son más acusados en las tareas más complejas y que, por consiguiente, requieren una mayor integración de funciones, especialmente cuando el material es no verbal. No obstante, como ya hemos señalado, ello no implica un efecto específico del alcohol, sino que manifiesta la especial vulnerabilidad de estas funciones a los efectos deteriorantes cualquiera que sea su causa. Cuando se controla la susceptibilidad de las tareas a los efectos de las lesiones cerebrales generalizadas estas diferencias se desvanecen. En contraposición a lo que ocurre en otros trastornos que cursan con afectación de los rendimientos, el patrón de deterioro etílico es similar al que se produce en la involución fisiológica debida a la edad.

Pero la consideración de los pacientes alcohólicos en conjunto es engañosa, ya que entre ellos existe una notable

variabilidad. Al igual que en los trabajos realizados por otros autores hemos hallado un porcentaje importante (que supera el 40%) de pacientes sin signos de deterioro intelectual, mientras que los restantes quedan subdivididos en partes casi iguales en dos grupos. El primero presenta una afectación muy selectiva de los rendimientos integrativos más complejos, teniendo preservada tanto la memoria como la rapidez motriz, el segundo se caracteriza por déficits más generalizados que abarcan todas las funciones pero que inciden más sobre la memoria.

De forma general puede afirmarse que los rendimientos neuropsicológicos se hallan poco relacionados con variables externas. Efectivamente, no se ha encontrado una relación clara con posibles factores etiológicos derivados de la historia del consumo etílico ni con posibles variables intermedias de riesgo médico sugestivas de afectación orgánica. Ello obliga aceptar la existencia de otras causas no controladas en el conjunto de estos estudios y que, en último extremo y a falta de más información, pueden atribuirse a factores de vulnerabilidad individual.

A pesar de que los alcohólicos que persisten abstinentes y en tratamiento durante un año obtienen unos rendimientos neuropsicológicos similares a los sujetos controles, no puede hablarse de una reversión del deterioro. Efectivamente, en el seguimiento longitudinal la mejoría de estos enfermos no

alcanzó niveles estadísticamente significativos. Por tanto, una vez han aparecido los déficits neuropsicológicos pueden considerarse permanentes, por lo menos durante el período de un año que hemos estudiado.

Dado que nuestra muestra se diferencia sustancialmente de las descritas en la mayoría de los estudios procedentes del área anglosajona al presentar una historia de consumo etílico abusivo mucho más prolongada, la persistencia de déficit puede ser atribuida a un tiempo muy prolongado de exposición a los efectos del tóxico que dificultaría su reversibilidad.

La delimitación de dos subgrupos de pacientes deteriorados sugiere la existencia de un doble mecanismo de afectación del alcohol sobre las funciones intelectuales. Una parte de estos enfermos presentarían un déficit ubicable en las estructuras corticales y se manifestaría por una pérdida de rendimiento en las tareas más complejas que requieren mayor capacidad de integración. Su expresión clínica máxima correspondería a la demencia cortical alcohólica.

El segundo subgrupo de alcohólicos con deterioro presentaría un cuadro similar, aunque atenuado, del síndrome de Korsakoff. La afectación de las estructuras cerebrales diencefálicas explicaría los déficits mnésicos y la alteración de las conexiones neocorticales (posiblemente la interrupción de los aferencias colinérgicas que parten del

núcleo basal de Meynert tal como sugiere Lishman [1986]), la coexistencia de afectación de las funciones complejas.

En este sentido la discusión sobre cuál es el modelo neuropsicológico explicativo de los déficits intelectuales de los pacientes alcohólicos debería replantearse tomando en consideración la heterogeneidad de rendimientos. La hipótesis de una afectación generalizada cortico-subcortical (Parsons, 1987, Parsons y Leber, 1981) sería en realidad la consecuencia de la sumación de dos grupos de patología.

El primero correspondería al modelo de afectación cortical difusa (Cermak, 1987) o de las áreas corticales y sus conexiones (Tarter y cols. 1984, 1985). Esta disfunción justificaría las similitudes con las consecuencias de la involución fisiológica secundaria a la edad: el discutido "envejecimento prematuro" (Blusewicz y cols., 1977; Ryan, 1980; Ryan y Butters, 1980).

Por el contrario, en el segundo grupo de pacientes la teoría del continuum de destrucción específica de las estructuras centrodiencefálicas propias del síndrome de Korsakoff (Butters y Granholm, 1987; Ryan y cols, 1980; Ryback, 1971) explicaría una prevalencia de los déficits mnésicos junto a un deterioro generalizado no tan acusado.

La aceptación de estas dos vías de afectación, basada en la incuestionable heterogeneidad de los rendimientos

intelectuales en los enfermos alcohólicos, permite la integración de la mayoría de las concepciones teóricas alternativas propuestas hasta la actualidad. Los resultados diferentes obtenidos en los estudios empíricos que las sustentan podrían ser debidos a la distinta proporción de enfermos de cada uno de estos grupos en las poblaciones asistidas en los distintos centros donde se han realizado las investigaciones.

Esta última conjetura, quizás excesivamente aventurada, halla su justificación en las distintas historias toxicológicas y características sociodemográficas de los enfermos estudiados por cada autor. En base a los conocimientos actuales puede atribuirse la afectación cortical a las consecuencias directas del alcohol sobre el encéfalo, mientras que las lesiones axiales se producirían preferentemente por deficiéncias nutricionales o malabsorción.

A la luz de estos resultados creemos que posteriores trabajos deberían tomar en consideración la existencia de estos subgupos y buscar independientmente en cada uno de ellos los posibles factores etiológicos relacionados y la recuperación tras períodos prolongados de abstinencia. Si las estructuras y los mecanismos etiológicos responsables son diferentes es previsible que el restablecimiento funcional siga cursos distintos que queden enmascarados al efectuar análisis con todos los casos agrupados.

Lamentablemente, esta pespectiva de trabajo no ha podido ser cubierta con la muestra actual, ya que la partición da lugar a grupos excesivamente pequeños.

4.3 CONCLUSIONES FINALES

En base a los resultados del presente estudio se desprenden las siguientes conclusiones:

1) La batería neuropsicológica diseñada ha demostrado ser válida para la diferenciación entre sujetos normales y con deterioro leve. Su administración requiere 50 minutos, tiempo razonable para su empleo en situaciones clínicas.

2) Los rendimientos neuropsicológicos de los pacientes alcohólicos recientemente abstinentes reflejan un deterioro generalizado, más acusado en las tareas complejas y que requieren mayor capacidad de abstracción, integración de funciones y planificación de la respuesta.

3) La disminución de la capacidad intelectiva no se justifica únicamente por los efectos de los interdictores o las interferencias psicopatológicas.

4) En nuestra muestra, en que el tiempo de consumo abusivo es similar entre hombres y mujeres, la intensidad y las características del deterioro son idénticas en ambos sexos.

5) La edad de los pacientes incrementa los niveles de deterioro intelectual en una cuantía similar a lo que sucede en la población general. El déficit de rendimientos se aprecia ya en el grupo de menor edad.

6) El patrón de deterioro no es comparable al que se produce en síndromes lesionales neurológicos difusos ni en pacientes depresivos remitidos. A diferencia de estos grupos, los alcohólicos abstinentes presentan dificultades más marcadas en las tareas abstractivas e integrativas y menores en las de memorización o rapidez motriz.

7) Los resultados de este estudio no justifican una presunta lateralización de la disfunción ni especificidad de afectación de las estructuras fronto-límbico-diencefálicas.

8) No se ha hallado relación entre la magnitud del deterioro y la historia de consumo etílico, presencia de deliriums o blackouts, enfermedades médicas premórbidas o concomitantes ni alteraciones bioquímicas hepáticas.

9) Entre el 40 y el 50 por ciento de los pacientes, según se adopte un modelo categorial o continuo respectivamente, no presentan indicios de afectación de las funciones intelectuales. En los restantes aparecen dos perfiles diferenciados, uno caracterizado por déficits limitados a la esfera abstractiva e integrativa y el otro por deterioro generalizado con mayor afectación de la memoria.

10) El conjunto de estos datos sugiere la existencia de un grupo de pacientes con una susceptibilidad específica a los efectos deteriorantes del alcohol, sin que se haya logrado determinar ninguna variable explicativa de dicha susceptibilidad.

11) Este subgrupo parece hallarse dividido entre los que desarrollan una expresión mínima de síndrome de Korsakoff y otro de la demencia cortical etílica

12) La ausencia de deterioro al iniciarse el tratamieto es un índice de buen pronóstico de mantenimiento de la abstinencia a lo largo del primer año.

13) Los pacientes que se mantienen abstinentes al cabo de un año presentan rendimientos neuropsicológicos no diferenciables de los sujetos control. En parte ello es debido a que los sujetoss inicialmente no deteriorados tienen una mayor probabilidad de permanecer abstemios.

14) La discusión de los resultados obtenidos sugiere que posteriores investigaciones deberían tomar en consideración la heterogeneidad de la afectación neuropsicológica y desarrollar análisis independientes en cada uno de los tipos definidos, ya que las estructuras anatómicas y los mecanismos etiopatogénicos pueden ser diferentes en cada uno de ellos confundiéndose los efectos al agruparlos.

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