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Summary

El problema de la gestión hospitalaria de pacientes ectópicos

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El proceso tradicional de hospitalización de pacientes en unidades de Enfermería asignados a determinadas especialidades médicas ha influido en la organización de la asistencia sanitaria, facilitando el trabajo en equipo, la adquisición de conocimientos específicos a nivel médico y los cuidados de enfermería. Además, permite a su vez gestionar de manera adecuada los programas de atención en dispositivos alternativos a la hospitalización convencional, disminuyendo el número de estancias inapropiadas.

La existencia de pacientes periféricos o ectópicos, esto es, aquellos ingresados a cargo de un servicio en camas asignadas a otro, se asocia a efectos asistenciales negativos. Si bien no existe acuerdo en cuanto sus consecuencias en las complicaciones o en la mortalidad, los estudios realizados coinciden en que la asistencia de pacientes ectópicos se asocia a un aumento de las estancias y de los reingresos, como muestra el estudio de Montero et al publicado en este número de Galicia Clínica [1-3].

Es evidente que el paciente ingresado ectópico distorsiona la normal organización de la Unidad de Enfermería receptora del mismo, que tendrá que recordar o aprender planes de cuidados no habituales, nuevas pautas terapéuticas de otros facultativos y que el servicio médico responsable deberá asumir una sobrecarga al atender más pacientes y en unidades de Enfermería no habituales para los mismos. Esta circunstancia se produce con más frecuencia en pacientes de especialidades médicas, especialmente Medicina Interna, debido a que la situación que los genera se asocia habitualmente al concepto de ingreso urgente[1].

La presión a la que está sometido el servicio de urgencias tiene una repercusión directa en la gestión de camas debido a los ingresos que de él derivan. Está sometido a una demanda creciente que favorece en ocasiones largas esperas que generan malestar e incertidumbre en pacientes y familias. Por otra parte, a la necesidad de atender la dinámica inherente al dispositivo asistencial, se suma la dificultad de proporcionar adecuados cuidados médicos y de enfermería a los pacientes en espera de decisión clínica o ingreso. En este sentido, existen diferencias entre los tiempos de espera reales y los percibidos, de tal manera que los percibidos aumentan al estar en camilla respecto a una cama, a la intensidad del dolor o a la autopercepción de una mayor gravedad. También pueden variar en función del horario (mayor por las mañanas) y del tiempo en ser atendido por un médico por primera vez. Por ello, una correcta gestión hospitalaria debe tener en cuenta la responsabilidad del personal sanitario y los derechos del paciente. Es más fácil hacer efectivo el respeto a la intimidad y a la confidencialidad, así como mejorar la sensación percibida por un paciente que debe ser ingresado, en una cama de una unidad de hospitalización[4].

Existen dos modelos en la gestión de camas hospitalarias: el modelo tradicional de asignación a un servicio determinado, que es más rígido y puede crear conflicto ante la necesidad urgente de asignación de una cama, y el flexible con mayor tendencia a generar pacientes ectópicos[5]. Por tanto, la gestión de camas debe provenir de un conocimiento profundo de los circuitos de alta hospitalaria y de ingreso desde urgencias, ya que ambos están lógicamente interconectados. Así, es preciso tener en cuenta una serie de aspectos:

- Conocer número de camas disponibles totales en el centro y asignados a cada especialidad, contemplando las esperas programadas y urgentes. Es obligado disponer de datos históricos y dinámicos actualizados que tengan en cuenta diversas variables: número de urgencias atendidas, número de urgencias ingresadas, estancias, índice de ocupación, índice de rotación y estancia media.
- Conocer en tiempo real la situación de las camas de hospitalización. Este aspecto hace imprescindible la implicación, colaboración y apoyo de todo el personal facultativo durante el proceso completo de hospitalización. Por una parte, es necesario mejorar la eficiencia en la gestión de los recursos asistenciales, como disminuir las

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"Existen dos modelos en la gestión de camas hospitalarias: el modelo tradicional de asignación a un servicio determinado, que es más rígido y puede crear conflicto ante la necesidad urgente de asignación de una cama, y el flexible con mayor tendencia a generar pacientes ectópicos"

estancias inadecuadas en espera de pruebas que se pueden realizar de manera ambulatoria, agilizar la detección precoz de problemas sociales, evaluar adecuadamente la necesidad de transporte sanitario, comunicar las previsiones de altas hospitalaria con tiempo de antelación, etc. Por otra, es obligado disponer de un sistema informático ágil que permita que la información fluya en tiempo real para la toma de decisiones adecuadas.

- Conocer la situación clínica del paciente al que se va o asignar la cama.

Todo este proceso debe estar controlado por una estructura hospitalaria estable en el tiempo y responsable último de la asignación del recurso cama, el servicio de Admisión. En este sentido hay es fundamental la labor de personal de enfermería asignado este servicio, con un papel fundamental en la coordinación con las supervisoras de las distintas unidades de Enfermería y con el servicio de Urgencias, dando prioridad clínica al ingreso y asignándole la cama más apropiada.

La oferta del recurso cama no depende sólo de la cantidad, sino del uso que se haga de ella. Tanto gestores como profesionales sanitarios debemos implicarnos en su gestión, de otra manera será imposible modificar el círculo vicioso que se genera cuando el retraso en el proceso de alta de hospitalización de un paciente repercute directamente en la demora de un ingreso desde el servicio de Urgencias y favorece que éste se realice en una unidad diferente de la apropiada. Es preciso trabajar conjuntamente en el rediseño del proceso para mejorarlo y darle más flexibilidad, sobre todo en los períodos del año en los que sabemos sistemáticamente que se produce un aumento de los ingresos en los servicios médicos (ej. épocas de epidemia gripal). En este sentido, debemos disponer de planes perfectamente trazados y organizados que determinen la necesidad de aumento de recursos tanto materiales como profesionales para no generar ingresos ectópicos, con control exhaustivo de aislamientos e intensificación de medidas diseñadas para la atención en dispositivos alternativos a la hospitalización convencional (Hospitales de Día, Hospitalización a Domicilio).

Por otra parte, es especialmente importante trabajar en una definición clara de todos los flujos ambulatorios de pacientes con programas consensuados entre facultativos de Atención Primaria y las distintas especialidades hospitalarias para todas aquellas patologías que por su potencial gravedad requieran una atención prioritaria y en programas de asistencia compartida dirigida a pacientes crónicos complejos. Todas estas medidas redundarán sin duda no sólo en una mejora de la atención dispensada sino también en disminuir presión de los servicios de Urgencias y en la necesidad de estancias inapropiadas en unidades de hospitalización.

En definitiva, la gestión de las camas hospitalarias y por tanto del problema de los pacientes ectópicos es compleja y tiene múltiples caras e implicaciones. Como la mayoría de los problemas que subyacen en el día a día hospitalario debe contar con la colaboración transversal de gestores y profesionales sanitarios. También en este aspecto debemos formar equipos multidisciplinares centrados en el paciente.

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Resultados asistenciales y económicos en los pacientes "periféricos" de medicina interna

Health care and economic outcomes in "peripherical" patients of internal medicine

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Resumen

Fundamento y objetivo: El sistema tradicional de organización hospitalaria asigna un número de camas consecutivas, en una o varias unidades de hospitalización (plantas), a los diferentes servicios. Esta distribución permite una organización estable y circunscrita espacialmente, pero facilita la aparición de pacientes "periféricos".

Se analizó si los pacientes periféricos de Medicina Interna tienen peores resultados asistenciales y económicos que los ingresados en las camas propias del servicio.

Material y método: Estudio observacional retrospectivo que incluyó a los pacientes ingresados del 1 de enero al 15 de marzo y dados de alta antes del 1 de abril de 2015. Se compararon la estancia y mortalidad hospitalaria y los reingresos de los pacientes hospitalizados en las camas del servicio frente a los de los periféricos. Para el ajuste estadístico se utilizaron la edad, sexo, número de diagnósticos al alta y el índice de comorbilidad de Charlson.

Resultados: Participaron 1045 pacientes, el 27,8% (IC 95% 25,1 a 30,6) periféricos. La mortalidad en ambos grupos no difirió significativamente (OR 0,8 [IC 95% 0,5 a 1,3]), pero sí los reingresos (OR 1,6 [IC 95% 1 a 2,5]; p=0,037) y la estancia media (1 día [IC 95% 0,1 a 1,8]; p=0,034). Esto supuso un incremento del 51,2% en los reingresos y del 12,2% en la estancia. El exceso en la estancia originó un aumento del gasto en 157.703,3€.

Conclusiones: Los pacientes periféricos de Medicina Interna tienen unos resultados asistenciales peores que los hospitalizados en las plantas propias del servicio, con un incremento asociado del gasto.

Palabras clave: Medicina Interna. Pacientes ingresados. Estancia hospitalaria. Reingreso hospitalario

Abstract

Background and objective: Hospital management system assigns consecutive beds, in one or more inpatient units, to different medical or surgical services. This distribution allows a stable and spatially circumscribed organization, but facilitates the presence of "peripheral" patients.

It was studied whether peripheral Internal Medicine inpatients had worse health care and economic outcomes than those admitted to their own beds.

Materials and methods: Retrospective observational study involving patients admitted from January 1 to March 15 and discharged before 1 April 2015. Hospital readmissions, length of stay and in-hospital mortality of inpatients of Internal Medicine Service wards were compared against the peripheral ones. Used for statistical adjustement: age, sex, number of diagnoses at discharge and Charlson comorbidity index.

Results: The study included 1045 patients, 27.8% (95% CI 25.1 to 30.6) peripherals. Mortality in both groups did not differ significantly (OR 0.8 [95% CI 0.5 to 1.3]), but they did readmissions (OR 1.6 [95% CI 1 to 2.5]; p = 0.037) and length of stay (1 [95% CI 0.1 to 1.8]; p = 0.034). This represented an increase of 51.2% in readmissions and 12.2% in length of stay. Excess of hospitalization days has led to an increase in spending € 157,703.3.

Conclusions: Peripheral Internal Medicine patients have worse outcomes than inpatient care in their own beds, with an increase in associated costs.

Key words: Internal Medicine. Inpatients. Length of stay. Hospital readmission.

Introducción

En el sistema tradicional de organización hospitalaria se asigna un número determinado de camas consecutivas, en una o varias unidades de hospitalización (plantas), a los diferentes servicios. Esta distribución permite una organización estable y circunscrita espacialmente, y facilita el trabajo en equipo, en especial entre el personal médico y el de enfermería. Su principal desventaja es que haya camas sin utilizar o, por el contrario, que su número sea insuficiente para hospitalizar a todos los enfermos asignados a ese servicio en un momento dado. Esta última posibilidad aboca a la aparición de pacientes "periféricos" o "ectópicos", que son aquellos a cargo de un servicio pero ingresados en camas asignadas a otro distinto. La aparición de estos pacientes periféricos distorsiona la dinámica del hospital y, en especial, el funcionamiento del servicio a cargo del enfermo y el del "invadido". Asimismo, puede conllevar riesgos para los propios pacientes.

En general, los gestores no suelen tomar medidas para evitar o paliar en lo posible la presencia de este tipo de pacientes y controlar sus efectos. Es probable que esto se deba a que, en la gestión de las camas hospitalarias, existe una tendencia a la desaparición de la asignación de camas a cada servicio y la formación de unidades de hospitalización polivalentes[1], cuyo principal objetivo es el ahorro de costes. En los sistemas de salud actuales la productividad se ha incrementado a expensas de factores de seguridad[2]. Esto contrasta con otros campos de alto riesgo en los que la productividad es limitada por su adherencia a medidas de seguridad, las cuales tienen la máxima prioridad[2].

Los servicios de Medicina Interna sufren con mucha frecuencia

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períodos de sobrecarga asistencial que originan un importante número de pacientes periféricos. Esta situación, habitual en nuestros hospitales, podría conllevar peores resultados asistenciales y riesgos para dichos enfermos, así como un incremento en los costes, al estar ubicados fuera de su "entorno natural", el servicio de Medicina Interna. Sin embargo, a pesar de su importancia para clínicos y gestores, no hemos encontrado trabajos que analicen estos problemas.

Nuestro objetivo fue estudiar si los pacientes periféricos de Medicina Interna tienen peores resultados asistenciales y económicos que los ingresados en las camas propias del servicio.

Material y métodos

El hospital donde se ha realizado el estudio está dotado con 450 camas para atender a una población casi exclusivamente urbana de 250.000 habitantes. Imparte docencia de pre y postgrado y está acreditado para la formación de residentes médicos y quirúrgicos. El servicio de Medicina Interna tiene asignadas 115 camas, repartidas entre tres secciones que ocupan tres unidades de hospitalización completas. En períodos de sobrecarga asistencial, los enfermos ingresan periféricos en diferentes plantas del hospital según la disponibilidad de camas, sin ningún otro criterio de selección adicional, siendo repartidos entre las tres secciones del servicio en función de los ingresos que ha tenido cada una de ellas cada día. Los facultativos de cada sección, junto con sus residentes, atienden a los periféricos además de a sus pacientes asignados habitualmente en la sección. No hay facultativos específicos para los periféricos, ni circuito diferente para las altas ni ninguna otra actividad.

El estudio, observacional retrospectivo, ha incluido a todos los pacientes con fecha de ingreso en Medicina Interna desde el 1 de enero hasta el 15 de marzo de 2015 y dados de alta antes del 1 de abril de 2015. Se ha realizado comparando la estancia y mortalidad hospitalarias y el número de reingresos de los pacientes hospitalizados en las camas propias de Medicina Interna con los de los hospitalizados periféricos. El reingreso fue definido como el nuevo ingreso por cualquier motivo y en cualquier servicio en el plazo máximo de 15 días desde el alta previa; no se diferenció entre ingreso urgente y programado porque este último tipo supone menos del 2% de los ingresos de pacientes de Medicina Interna. Para el ajuste estadístico se recogieron las siguientes variables: Edad, sexo, número de diagnósticos al alta y el índice de comorbilidad de Charlson (ICh), el cual está validado para su uso con bases de datos administrativas[3], y con los pesos actualizados[4]. Además, en los pacientes periféricos se registró si el ingreso fue en camas de especialidades médicas o quirúrgicas, o en una planta abierta expresamente para este cometido (planta *ex profeso*), con personal de nueva contratación, por la presión asistencial, y que admitió ingresos desde el 13 de enero hasta el 18 de febrero. Con la excepción de esta última planta, el resto de las plantas del hospital no sufrieron ningún tipo de variación en su dotación de personal y medios. Por último, se registró la distribución por meses de los ingresos.

Los datos fueron obtenidos del Conjunto Mínimo Básico de Datos (CMBD) del hospital, el cual admite hasta 13 diagnósticos codificados de acuerdo con la CIE-9-MC. Según los servicios administrativos del centro, el coste medio del día de estancia en Medicina Interna durante el año 2014 fue de 385,3 €. Para el cálculo de costes, se multiplicó este coste por el número de estancias.

Análisis estadístico

Las variables cuantitativas fueron descritas con la media y su intervalo de confianza del 95% (IC 95%), y se comprobó su distribución normal mediante la prueba de Kolmogorov-Smirnov; las cualitativas mediante el porcentaje y su IC 95%. Por el tipo de trabajo y la naturaleza de los resultados se consideró suficiente una precisión de ±5 centésimas, por lo que fueron redondeados a un decimal. Las diferencias de medias se analizaron con la *t* de Student o la U de Mann-Whitney según fuera pertinente, las variables categóricas mediante la obtención de la *Odds Ratio* (OR). La estancia fue ajustada con un modelo de regresión lineal múltiple que incluyó todas las variables de ajuste estadístico. Las variables las introducimos en el modelo mediante el método de regresión por pasos. Los criterios de inclusión y exclusión de las variables en el modelo los fijamos en $p < 0,05$ para la inclusión y $p > 0,10$ para la exclusión. Los modelos finales los seleccionamos utilizando el criterio del cuadrado del coeficiente de correlación múltiple ajustado. El estudio multivariante de la mortalidad y de los reingresos fue llevado a cabo mediante regresión logística, introduciendo todas las variables de ajuste estadístico con el método de inclusión por pasos. Los criterios de inclusión y exclusión de las variables en los modelos los fijamos en $p < 0,05$ para la inclusión y $p > 0,10$ para la exclusión. La calibración de los modelos la valoramos por medio de la prueba de bondad de ajuste de Hosmer-Lemeshow. Para el análisis de tendencias el orden de los grupos ha sido Medicina Interna, plantas médicas, plantas quirúrgicas, planta *ex profeso*. Las tendencias en la estancia entre las diferentes plantas de hospitalización fueron estudiadas mediante el análisis de la varianza con contrastes polinómicos, y para la mortalidad y los reingresos con la prueba de tendencia lineal de Mantel-Haenszel. El nivel de significación estadística se estableció en $p<0,05$. Todos los cálculos fueron realizados con el paquete estadístico SPSS 15.0 (SPSS Inc. Chicago, Illinois, EEUU).

Resultados

El estudio incluyó un total de 1045 pacientes, de los que el 98,3% (IC 95% 97,5 - 99,1) fueron ingresos urgentes. El 72,2% (IC 95% 69,4 - 74,9) fueron hospitalizados en las camas de Medicina Interna y el 27,8% (IC 95% 25,1 - 30,6) periféricos: 4,3% (IC 95% 3,1 - 5,6) en plantas médicas, 15,5% (IC 95% 13,3 - 17,7) en quirúrgicas y 8% (IC 95% 6,4 - 9,7) en la planta abierta *ex profeso* (tablas 1 y 2). No se apreciaron diferencias en los ingresos periféricos entre el fin de semana y el resto de los días de la semana. La distribución por meses y unidades de hospitalización se muestra en la figura 1. Aunque aparecen algunas diferencias entre los diversos grupos en las variables de ajuste, no alcanzan significación estadística en ningún caso. Una vez realizado el ajuste con las variables de ajuste estadístico, la mortalidad de los enfermos ingresados en las camas de Medicina Interna y la de los periféricos no difiere significativamente (OR 0,8 [IC 95% 0,5 - 1,3]; no significativo), pero sí lo hacen los reingresos (OR 1,6 [IC 95% 1 - 2,5]; p=0,037) y la estancia media (diferencia 1 día [IC 95% 0,1 - 1,8]; p=0,034). Esto su-

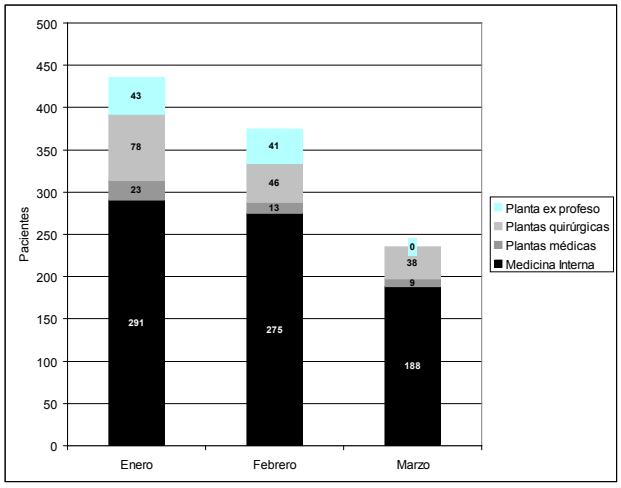
Tabla 1. Resultados de los grupos Medicina Interna y Periféricos

	Total	Medicina Interna	Periféricos	Dif/OR
N	1045	754	291	
Edad; años (IC 95%)	79 (78,2 - 79,8)	79 (78 - 79,9)	78,9 (77,3 - 80,5)	-0,1 (-1,9 – 1,8) n.s.
Mujer; % (IC 95%)	52,1 (49 - 55,1)	52 (48,4 - 55,6)	49,2 (46,1 - 53,5)	OR 1 (0,8 – 1,3) n.s.
ND (IC 95%)	11,1 (10,9 - 11,2)	11,1 (10,9 - 11,3)	11,1 (10,8 - 11,4)	0 (-0,3 – 0,4) n.s.
ICh (IC 95%)	5,6 (5,4-a 5,8)	5,7 (5,4 - 5,9)	5,5 (5,2 - 5,8)	-0,2 (-0,5 – 0,2) n.s.
Estancia; días (IC 95%)	9,9 (9,5 - 10,3)	9,7 (9,2 - 10,1)	10,6 (9,7 - 11,4)	0,9 (-0,02 – 1,8) n.s. (p=0,055)
Exitus; % (IC 95%)	12,4 (10,4 - 14,4)	13 (10,6 - 15,4)	11 (7,4 - 14,6)	OR 0,8 (0,5 – 1,3) n.s.
Reingresos % (IC 95%)	9,4 (7,6 - 11,1)	8,2 (6,3 - 10,2)	12,4 (8,6 - 16,2)	OR 1,6 (1 – 2,4) p=0,041

ND: Número de diagnósticos al alta. ICh: Índice de comorbilidad de Charlson. IC 95%: Intervalo de confianza del 95%. Dif: Diferencia. OR: Odds Ratio. n.s.: No significativo.

pone un incremento del 51,2% en los reingresos y del 12,2% en la estancia. La media de pacientes periféricos ingresados cada día del estudio fue 35,6, lo que supone un 31% más sobre la capacidad del servicio.

Fig. 1. Distribución de pacientes por meses y plantas



No se ha observado ninguna tendencia ni en la mortalidad ni en los reingresos, pero sí una tendencia lineal ascendente en la estancia (F=5,021; p=0,025).

Cada uno de los 291 pacientes periféricos tuvo un día de estancia más con respecto a los ingresados en Medicina Interna, originando 291 estancias extra. Asimismo, el grupo de periféricos ocasionó un 4,2% más de reingresos que el de Medicina Interna, por lo que los 291 pacientes periféricos originaron 12,2 reingresos más que si hubieran estado ingresados en Medicina Interna.

Utilizando como referencia la estancia media de Medicina Interna (9,7 días), esos 12,2 reingresos implican otras 118,3 estancias. El exceso total de estancias acumulado por estos pacientes fue de 409,3. Aplicando el coste de día de estancia, los pacientes periféricos de Medicina Interna, en el periodo de estudio, implicaron un incremento del gasto en 157.703,3€.

Discusión

Nuestros hallazgos muestran que los pacientes periféricos de Medicina Interna tienen unos resultados asistenciales peores que los hospitalizados en las plantas propias del servicio, con un incremento asociado del gasto. Como el ingreso como periférico sólo depende de la disponibilidad de camas, el estudio podría considerarse muy próximo a uno aleatorizado. De hecho, ni la edad, el sexo ni la comorbilidad difieren significativamente entre los diferentes grupos, ésta última estudiada con el ICh y el número total de diagnósticos al alta que, conjuntamente, permiten valorar la complejidad y la comorbilidad médica[5]. No hubo traslados desde periféricos a las plantas de Medicina Interna. Debemos tener muy presente que la complejidad de un paciente se debe a una relación intrincada entre diversos factores, como son la enfermedad, el tratamiento, la familia o el comportamiento general de todos los estamentos e individuos que intervienen en su cuidado, más que a la coexistencia de varias enfermedades[6]. Por lo tanto, la complejidad podría estar influida por el hecho de ingresar en plantas con organizaciones diferentes o no habituadas a este tipo de pacientes y afectar a los resultados obtenidos.

Una forma indirecta de valorar la dificultad de manejo de los pacientes es mediante el análisis de la estancia, la cual además predice bien el coste asistencial[7,8] y el riesgo de padecer sucesos adversos: 12% para 1-2 días y 46% para más de 12 días[9]. Nuestros resultados muestran que los pacientes periféricos parecen presentar una mayor dificultad de manejo que quedaría reflejada en su mayor estancia. La tendencia observada entre los diferentes grupos nos indica que el problema parece radicar en las diferentes plantas y no en los médicos, ya que son los mismos para todas ellas. Según se van alejando las diversas plantas en sus características con respecto a la hospitalización de Medicina Interna, aumentan las dificultades, siendo las mayores en la planta de nueva creación, la cual precisa un rodaje para alcanzar la efectividad del resto de ellas. Este último hallazgo demuestra que las medidas precipitadas pueden ser contraproducentes.

El grado de organización y el desarrollo de las rutinas apropiadas de cualquier actividad, incluida la asistencial, es fundamental para obtener el fin deseado. Por ello, según nos alejamos de dichos parámetros los resultados empeoran. Así, se ha observado que los pacientes ingresados en Medicina Interna durante el fin de semana tienen mayor mortalidad[10], se obtienen peores resultados en los pacientes operados los viernes[11], existe una mayor dificultad de manejo en los pacientes ingresados en

Tabla 2. Distribución y resultados de los pacientes periféricos

	Pl. médicas	Pl. quirúrgicas	Planta ex profeso
N	45	162	84
Edad; años (IC 95%)	80,3 (76 - 84,5)	79 (76,9 - 81,1)	78,1 (75,1 - 81,1)
Mujer; % (IC 95%)	48,9 (40,2 - 58,8)	48,8 (41,1 - 56,5)	50 (39,3 - 60,7)
ND (IC 95%)	11 (10,2 - 11,7)	11,3 (10,9 - 11,6)	10,9 (10,3 - 11,5)
ICh (IC 95%)	5,4 (4,7 - 6,2)	5,9 (5,4 - 6,3)	4,9 (4,5 - 5,4)
Estancia; días (IC 95%)	9,8 (7,4 - 12,1)	10,4 (9,4 - 11,4)	11,3 (9,4 - 13,2)
Exitus; % IC 95%)	11,1 (1,9 - 20,3)	11,1 (6,3 - 16)	10,7 (4,2 - 17,3)
Reingresos % (IC 95%)	11,1 (1,9 - 20,3)	13 (7,8 - 18,1)	11,9 (5 - 18,8)

ND: Número de diagnósticos al alta. ICh: Índice de comorbilidad de Charlson. IC 95%: Intervalo de confianza del 95%.

servicios quirúrgicos que no son operados[12], o que la mezcla de pacientes de diferentes especialidades quirúrgicas en las mismas habitaciones aumenta las infecciones de herida quirúrgica por gérmenes habituales de la otra especialidad y, además, las infecciones en general[13].

Cabría esperar que a mayor dificultad mayor mortalidad, sin embargo no es así. Esto podría explicarse por una mayor implicación, a pesar de las dificultades, del personal médico y de enfermería en los pacientes más graves. También habría que tener en cuenta el efecto de la guardia médica en reducir la mortalidad, como ya se ha observado en el área quirúrgica[14].

El reingreso hospitalario se considera un resultado adverso y un marcador de calidad asistencial[15]. Su precocidad, habitualmente antes de 30 días, está relacionada con la asistencia recibida durante el ingreso previo[16]. Por este motivo, hemos intentado asegurar más esta relación acortando a 15 días la definición de reingreso. Las razones por las que reingresan pacientes son múltiples y variadas, entre ellas su mayor complejidad[17], la cual ya hemos comentado que también está relacionada con la organización y capacidades de las diferentes plantas.

Nuestros hallazgos muestran que el hecho de ingresar periférico acarrea un incremento importante de los costes económicos, a los que habría que añadir los posiblemente originados en todos aquellos servicios que se ven interferidos en su dinámica de trabajo por la presencia de los periféricos de Medicina Interna. Este hecho nos hace pensar que no está tan claro que la política de unidades de hospitalización polivalentes sea rentable económicamente.

Este trabajo tiene algunas limitaciones. Una de ellas es su diseño observacional retrospectivo. Asimismo, la obtención de datos a partir de bases administrativas podría hacer que éstos no fueran del todo fiables, si bien estas bases parecen tener una buena concordancia con los registros clínicos[18]. El hecho de estar realizado en un único hospital podría hacer que los resultados y conclusiones de este estudio pudieran no ser extrapolables a otros centros. Un inconveniente adicional es que no hemos encontrado bibliografía con la que poder comparar nuestros resultados.

Como conclusión podemos decir que la existencia de pacientes periféricos del servicio de Medicina Interna no solo empeora sus resultados asistenciales y económicos, sino que, además, podría afectar negativamente a otros servicios. Por ello, se deben tomar medidas, no improvisadas, que eviten o, al menos, palien esta situación. Los argumentos y resultados expuestos en este trabajo también pueden ser de utilidad a la hora de establecer unidades de hospitalización polivalentes.

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Tuberous sclerosis complex: an opportunistic diagnosis

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Abstract

Tuberous sclerosis complex (TSC) is a rare genetic multisystem disease that involves brain, skin, kidneys, eyes and lungs. It typically presents during childhood with facial adenomas, seizures and mental retardation and its diagnosis is mainly clinical. We report a case of 68-year-old man that sought medical care due to a transient ischemic stroke. The past clinical history and physical observation showed symptoms and signs of TSC. An extend imagiological study allowed for a complete assessment of organ involvement. A delayed diagnosis places adults at increased risk for morbidity and mortality and therefore, clinicians must be familiar with the full spectrum of TSC-associated diagnostic features.

Keywords: Angiofibromas. Tubers.

Introduction

Tuberous sclerosis complex (TSC) is an neurocutaneous disorder affecting cell development and maturation and is characterized by pleomorphic features that can affect every organ system. The classic TSC diagnostic triad of seizures, intellectual disability and facial angiofibromas (Vogt triad) occurs in less than one--third of patients with TSC. Though its treatment is only symptomatic, an early diagnosis is crucial to prevent long term organ system damage[1].

Case report

A 67--year--old man presented in the emergency room with a transient ischemic stroke ABCD2 score 4. No other neurological or systemic symptoms were present. On observation, he had multiple pebble--like pink tubers on his facial mask below the lower rim of the eyes and extending to the cheeks and composure of his mouth, as well as just below his lower lip and above his chin (Figure 1). On his lower back, he had a tuberous growth with a smooth surface and dimensions of approximately 10 mm of diameter and 2 to 3 mm in height (Figure 2). His finger and toe nail beds were normal. His past medical history included mental retardation with learning disability that made him drop out of school at 11 years old. As a single man without offspring, he lived with his sister after a psychological evaluation, at age 40, showed him unfit to work due to his childish behavior. His father had had epilepsy but there was no other relevant family history. Haematological and biochemical profile had no alterations. Computed tomography (CT) scan of the brain revealed subependymal and supratentorial nodules. Magnetic resonance imaging showed bilateral fronto--parieto-occipital tubers (Figure 3). His electroencephalogram was normal. Thoracic, abdominal and pelvic CT scans were normal. An ophtalmologic evaluation revealed 2 retinal hamartomas on his right eye. No areas of retinal hypopigmentation were present. A cardiac resonance showed a rhabdomyoma in the left ventricle without compromise of its function. He was dismissed with the diagnosis of Tuberous Sclerosis Complex with brain, eyes and cardiac involvement. The patient maintains annual surveillance in our hospital.

Fig. 1. Facial angiofibromas

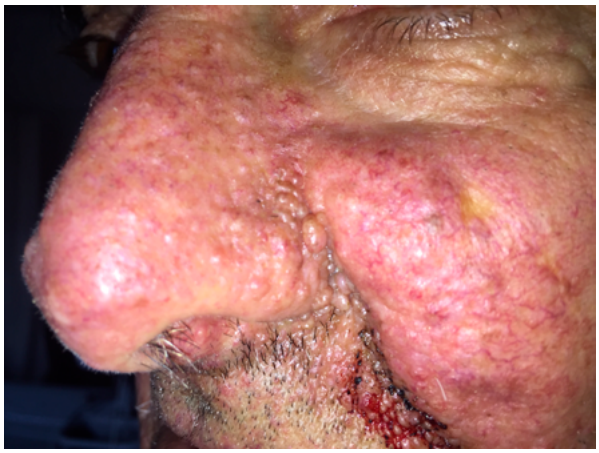

Fig. 2. Tuberous growth in the lumbar región

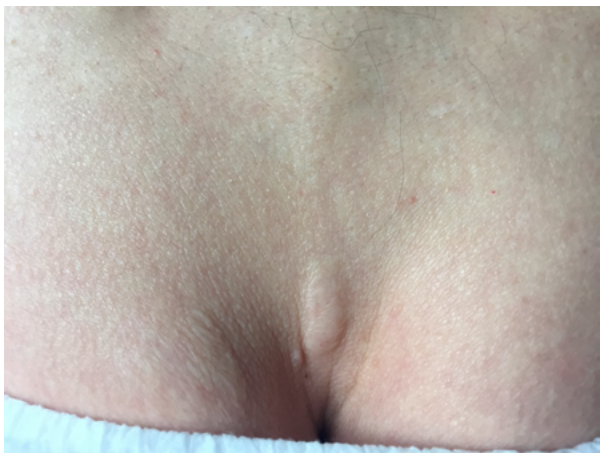

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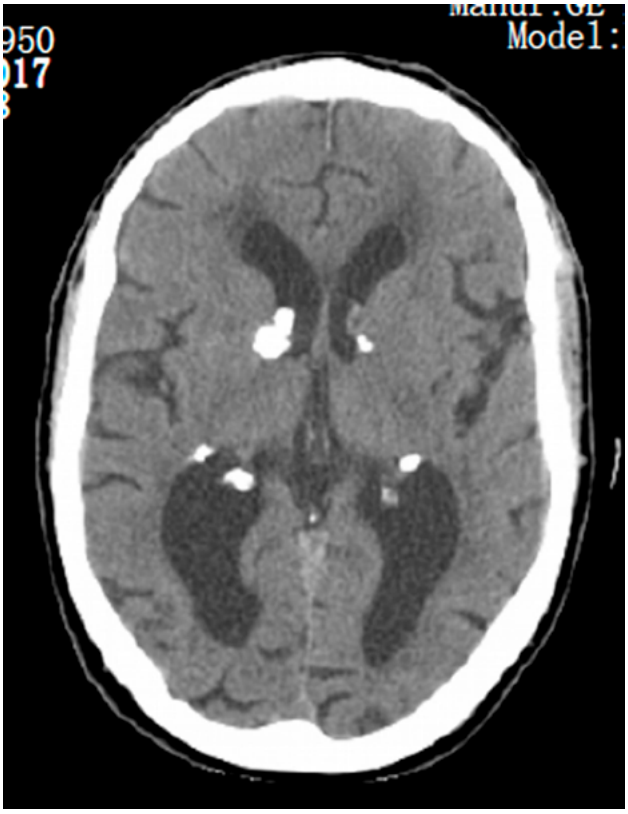
Fig. 3. Tomography scan of the brain with subordinate and supratentorial nodules



Discussion

Tuberous sclerosis complex (TSC) or Bourneville's disease was first described by Desiree-Magloire Bourneville in 1880[2]. It is a rare genetic disorder of autosomal dominant inheritance with an incidence of approximately 1 in 5000 to 10,000 live births[3].

It has been determined that mutations in two genes are responsible for the development of tuberous sclerosis: TSC1 gene, called the hamartin gene, is located on chromosome 9, and the TSC2 gene, called the tuberin gene, is located on chromosome 16. This hamartin--tuberin complex inhibits mTOR (mammalian target of rapamycin), a serine/threonine protein kinase that regulates cell growth and differentiation. When either of these tumor suppressor genes contains a defect, tumors in the form of normal organ tissues (hamartomas) can result[4].

As an autosomal dominant disease, one--third of the cases derive from an altered TSC1 or TSC2 gene inheritance. A patient with TSC has a 50% chance of having a child affected by the disease, and the risk of a healthy couple, who had a child with TSC, to have another child with the disease is 2%. Medical evaluation of healthy family members, who are planning to have children, should be performed by a dermatological examination, renal ultrasonography and cranial CT scan. Genetic evaluation by polymerase chain reaction, DNA sequencing and TSC1 and TSC2 dosage is not routinely indicated but helps when the clinical and radiological diagnosis is uncertain, especially in pre--natal cases[4].

TSC is highly variable in its age of onset and severity, even within the same family. Its phenotypic expression involves many organ systems, including multiple benign hamartomas of the

Table 1. Diagnostic criteria for tuberous sclerosis complex. Definite diagnosis: 2 major features or one major feature with ≥2 minor features. Possible diagnosis: either 1 major feature or ≥2 minor features. *A combination of the two major clinical features (lymphangioleiomyomatosis and angiomyolipomas) without other features doesn't meet criteria for definite diagnosis

Genetic diagnostic criteria	Clinical diagnostic criteria	
Identification of either TSC1 or TSC2 pathogenic mutation in DNA from normal tissue	**Major Criteria**	**Minor Criteria**
	Cortical displasias (includes tubers and cerebral white matter radial migration lines)	Dental enamel pits (>3)
	Subependymal nodules	Intraoral fibromas (≥2)
	Angiofibromas (≥3) or fibrous cephalic plaque	Retinal achromatic patch
	Ungual fibroma (≥2)	*Confetti* skin lesions
	Hypomelanotic macules (≥3, at least 5 mm in diameter)	Nonrenal hamartomas
	Shagreen patch	Multiple renal cysts
	Multiple retinal hamartomas	
	Cardiac rhabdomyoma	
	Angiomyolipomas (≥2)*	
	Lymphangioleiomyomatosis*	

brain, eyes, heart, lung, liver, kidney, and skin that lead to organ dysfunction as the normal parenchyma is replaced by a variety of cell types[5].

The diagnosis of TSC is made clinically. Diagnostic criteria for TSC is as given in the table 1. Definite diagnosis is made when two major or one major plus two minor features are present. Genetic testing is not required to make a diagnosis in patients who fulfill criteria for definite TSC, but it is helpful for family studies[6].

Nearly all patients with TSC have one or more of the skin lesions characteristic of the disorder (hypopigmented macules; angiofibromas; Shagreen patches). A distinctive brown fibrous plaque on the forehead may be the most readily recognized feature. International guidelines for TSC recommend performing a detailed skin examination at the time of diagnosis and annually thereafter. Although, there is no significant risk of malignant transformation, good sun protection, closer surveillance and intervention (laser therapy, dermabrasion) is recommended for skin lesions that rapidly change in size or number, and for those that cause pain, bleeding, functional impairment or social problems[7].

The major neurological manifestations of TSC are seizures, autism, developmental delay and behavioral and psychiatric disorders. They usually associate with brain lesions including glioneuronal hamartomas (also called tubers), periventricular giant cell astrocytomas and abnormalities of cerebral white matter. Seizures are the most common and difficult aspect of management in TSC. The International Tuberous Sclerosis Complex Consensus Group recommends vigabatrin as first--line therapy for infantile spasms, and oxcarbazepine or carbamazepine for simple partial or complex partial. Approximately 63% of patients with TSC and epilepsy develop medically intractable epilepsy and the treatment options for these patients include a ketogenic diet; vagus nerve stimulation; epilepsy surgery; and everolimus, a mTOR inhibitor. For brain tumors there are two main treatments: medical therapy with mTOR inhibitor or surgical resection. For the neuropsychiatric disorders, measures include early intervention and individual education programs, social support and psychiatric evaluation and treatment[8].

Ophtalmologic involvement is described as the presence of more than one retinal hamartoma that have similar histologic features to the tubers located in the brain. Retinal achromatic patches can also occur[5].

Renal lesions such as angiomyolipomas, cysts, lymphangiomas and renal cell carcinoma can occur and their prevalence increases with age. Nephron--saving surgery or partial nephrectomy can be performed when renal lesions are symptomatic. Total nephrectomy is reserved for cases with suspected malignancy and non--functioning kidneys with uncontrolled hypertension[5].

Some adults with TSC develop pulmonary disease that is indistinguishable from the diffuse interstitial fibrosis known as lymphangioleiomyomatosis (LAM). This condition represents a cystic lung disease that can result in significant limitation in pulmonary function. The most common presenting features of LAM are dyspnea and pneumothorax. The treatment of LAM associated with tuberous sclerosis is identical to that of sporadic LAM. General measures include smoking cessation, bronchodilators, supplemental oxygen and pulmonary rehabilitation. Sirolimus and everolimus are first and second--line treatment, respectively, for patients with moderate--to--severe disease. For patients with advanced LAM or for those refractory to mTOR inhibitors, lung transplantation is a therapeutic option[9].

Cardiac rhabdomyomas usually do not cause serious medical problems but, if symptomatic, surgical resection maybe necessary[10].

In conclusion, tuberous sclerosis complex is highly variable and the majority of the cases are diagnosed during childhood. This report shows a delayed and opportunistic diagnosis, but as disease manifestations continue to develop over the lifetime of an affected individual, it is of the utmost importance the implementation of an appropriate medical surveillance and treatment[6].

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Aneurisma micótico de la arteria glútea superior, complicación de una endocarditis bacteriana

Mycotic aneurism of the superior gluteal artery, complication of an endocarditis

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Resumen

El aneurisma micótico de la arteria glútea superior es excepcional. Tras una revisión de las publicaciones de aneurismas de esta arteria tan solo se han encontrado documentados seis casos. Presentamos el caso de un varón de 65 años con un aneurisma micótico en la arteria glútea superior presentada en el seno de una endocarditis bacteriana.

Palabras clave: Aneurisma. Micótico. Endocarditis.

Abstract

Mycotic aneurysm of the superior gluteal artery is exceptional, there are only six cases reported. This is the case of a 65-year-old man with a mycotic aneurysm, the first symtom of a bacterial endocarditis.

Keywords: Aneurysm. Mycotic. Endocarditis.

Caso clínico

Varón de 65 años con deterioro cognitivo leve de origen etílico e institucionalizado recientemente, sin otros antecedentes de interés. Cuatro días antes del ingreso presenta inflamación en el glúteo izquierdo. Se realiza una analítica donde destaca una hemoglobina 4,5 mg/dl normocítica-normocrómica, ingresando para estudio. Tras 24 horas de hospitalización presenta fiebre, hemiparesia derecha y dolor abdominal. Se extraen hemocultivos, se inicia antibioterapia empírica con amoxicilina-clavulánico y se solicita prueba de imagen donde muestra ACVA isquémico occipital izquierdo y un gran hematoma glúteo izquierdo de 20x3x6 cm con sangrado activo a expensas de la arteria glútea superior izquierda. Se realiza de forma urgente cateterización supraselectiva y embolización de la rama, lográndose el cese del sangrado (imagen 1). En los hemocultivos crece *Streptococcus gallolyticus spp. gallolyticus* .Por el cuadro clínico, los hallazgos radiológicos y el resultado microbiológico se plantea el diagnóstico de endocarditis, que se confirma mediante ecocardiograma. La evolución posterior del paciente fue favorable, no presentando nuevos eventos embólicos o resangrado del aneurisma. Se desestimó la intervención quirúrgica de la válvula por la situación sociofamiliar y el deterioro cognitivo basal.

Imagen 1. La arteriografía selectiva muestra el aneurisma de la arteria glútea superior (flecha)

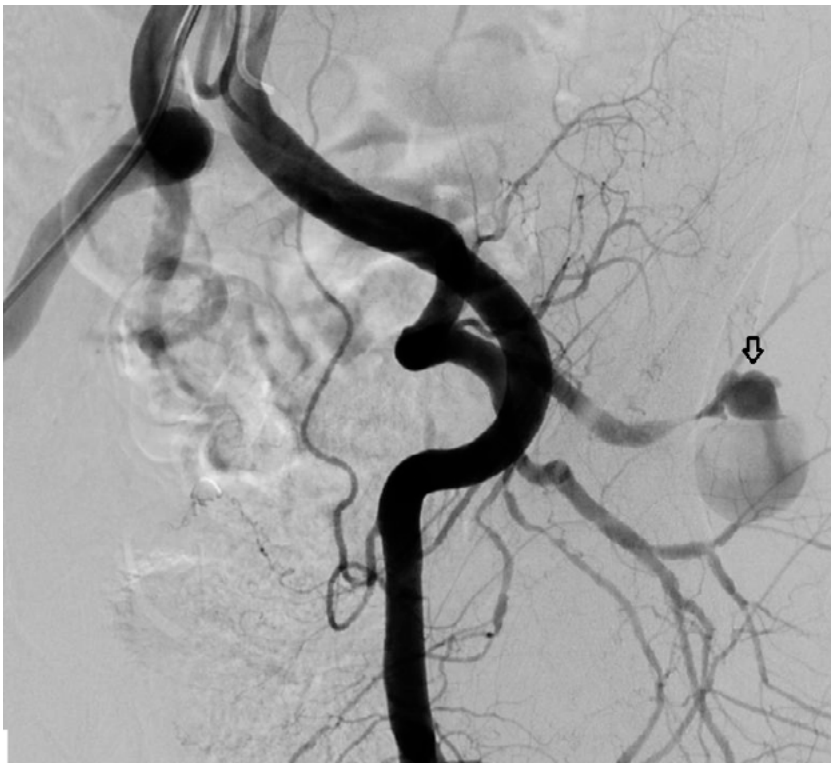

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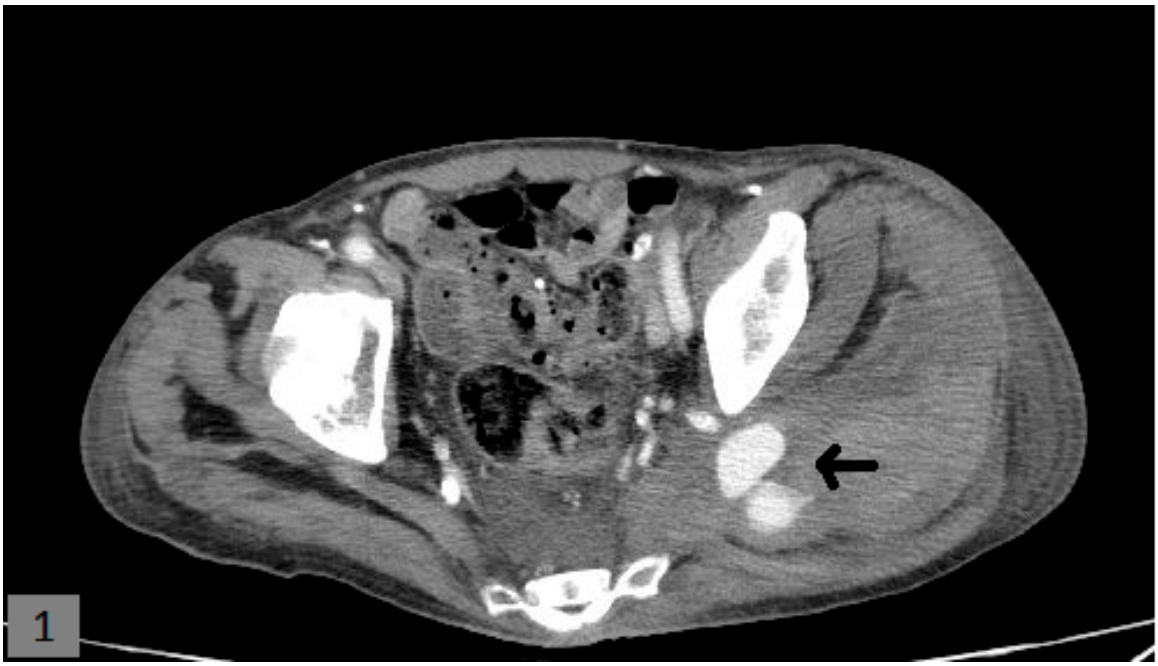
Imagen 2. TAC mostrando la situacion del aneurisma



Discusión

El aneurisma micótico como complicación de una endocarditis ocurre en un 2-4% de las endocarditis, aunque es probable que esta cifra este infraestimada[1]. La mayoría son a nivel intracraneal, y en localizaciones extracraneales las más frecuentes son las arterias viscerales o de la región proximal de miembros inferiores. La localización más frecuente es en bifurcaciones, y por su origen embolígeno suelen ser múltiples[2]. Los pseudoaneurismas de cualquier origen de la arteria glútea superior suponen menos del 1% y la mayoría son resultado de un traumatismo pélvico o heridas inciso-contusas. Causas menos frecuentes son la infección, poliarteritis nodosa, arterioesclerosis y degeneración mucoide intimomedial[3]. El primer caso de aneurisma micótico de la arteria glútea superior se describió en 1992[4]. Desde entonces sólo se han documentado 6 casos. Tienen una alta tasa de mortalidad debido a la tendencia a la rotura por presentar paredes delgadas y friables. No se ha identificado un predictor de ruptura, aunque parece no guardar relación con el tamaño; a diferencia de los aneurismas no infecciosos[1]. La prueba definitiva para su diagnóstico es la angiografía, si bien otras pruebas de imagen como TC o RMN han mostrado buena sensibilidad y especificidad[1] (imagen 2). Su manejo depende de su tamaño y localización. Los de localización intracraneal se tratan de forma conservadora mediante antibióticos y controles, salvo complicaciones que requieran tratamiento endovascular[1-2]. En los de localización extracraneal, clásicamente manejados mediante cirugía abierta, en los últimos años aumenta la evidencia de que un abordaje endovascular puede ser la primera línea en el tratamiento[5].

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4th time is a charm: Peritoneal tuberculosis and how difficult is to reach it

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Abstract

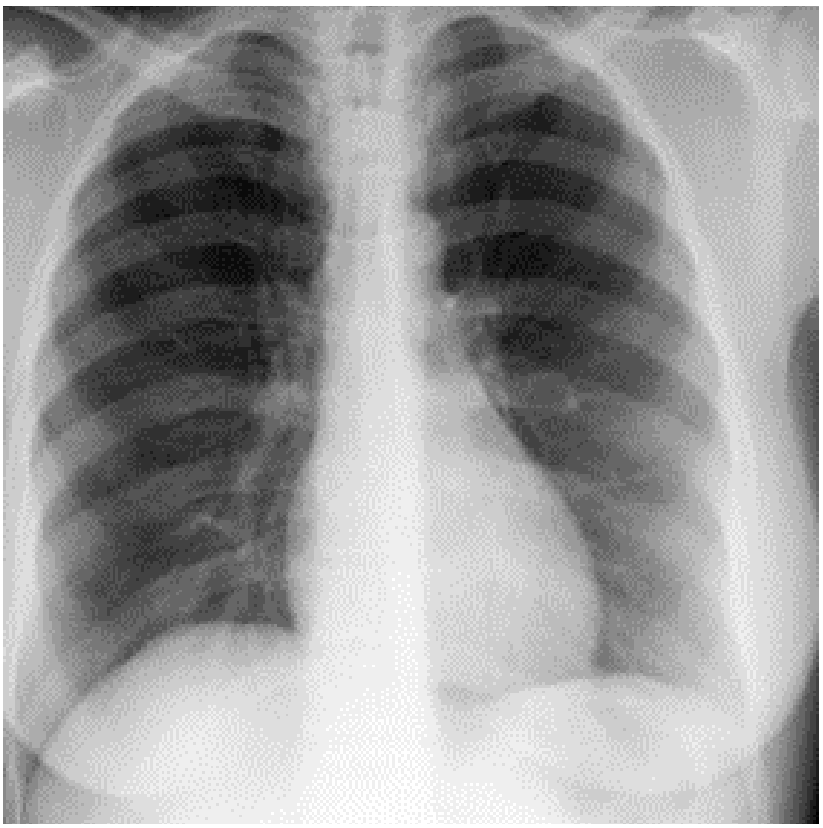
Tuberculosis has an increased incidence in the last few years due to the phenomenon of migration and remains a public health problem in certain regions of the world. The abdominal form is the sixth leading cause of extrapulmonary TB, after lymphatic, genitourinary, osteoarticular, miliary and meningeal. Usually, is more prevalent in <40years old females and due to its nonspecific clinical manifestations remains a clinical challenge for most physicians. We present the case of 42--year--old female with peritoneal tuberculosis (PTB). The patient suffered from abdominal pain and increasing abdominal perimeter 13 days prior to admission. She went to the emergency department four times in 10 days and was discharged with multiple diagnosis. The diagnosis was established on the basis of clinical features, findings from an abdominal tomography scan and histopathological analysis of peritoneal biopsy. The aim of this review is to expose the struggle experience diagnosing such a challenging disease.

Key words: Peritoneal Tuberculosis, Tuberculosis, Abdominal pain, Ascitis, Exploratory Laparotomy.

Introduction

Peritoneal tuberculosis is one of the forms of abdominal tuberculosis, and can be located in any site of the gastrointestinal and genitourinary tract, solid organs, abdominal aorta and linf nodes. About 30% of cases of extrapulmonary tuberculosis and 20% of all causes of ascites occur in developing countries. This form of tuberculosis is rarely associated with the active form of pulmonary disease, sometimes presenting with nonspecific symptoms, and for this reason has been described by some authors as "the great mimic". Peritoneal involvement is often accompanied by abdominal pain (80--95%) often associated with fever(40--90%), weight loss (40-90%)and anorexia (30%), and this combination may occur for many months. Among the known risk factors are patients diagnosed with cirrhosis, human immunodeficiency virus infection, diabetes mellitus, malignancies, immunosuppressive patients, and patients undergoing dialysis. In most cases, the infection occurs through the reactivation of a

Fig. 1. Chest X Ray revealed no infiltrations



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latent focus located in the peritoneum (through hematogenous dissemination of a pulmonary focus), hematogenous through an active pulmonary focus or miliary tuberculosis, with less invasion of the cavity peritoneal disease through intestinal infections or genitourinary tuberculosis. Peritoneal TB has three types: the wet type with ascites, the encysted type with abdominal swelling and the fibrotic type with abdominal masses.

Case Report

A 42--year--old female, with no history of disease, was admitted to the emergency department with a 13days history of abdominal pain and low fever. After four episodes of medical consultation, the patient was admitted for etiological study. She was feverish, with complaints of malaise and anorexia with weight loss of 7Kg. The patient´s abdomen was blown, soft with normal bowel sounds and ascites. The initial laboratory studies showed low hemoglobin (10,7g/dL), normal white blood cells (4,987/ µl) increased erythrocyte sedimentation rate (51mm/sec), protein C reaction (39mg/dL) and CA--125 (346 IU/mL). Hepatic and renal function were normal. HIV and hepatotrophic viruses were negative. A tuberculin skin test was negative.

Chest X--Ray did not present any infiltrations (fig 1). A pelvic--abdominal computed tomography (fig 2 and 3) showed ascites of moderate volume and a pelvic left adnexial tubular lesion with serpiginous morphology with hypothesis of peritoneal tuberculosis with tubular involvement. A diagnostic paracentesis was performed and revealed exsudate findings. The patient underwent exploratory laparoscopic with peritoneal biopsy which confirmed peritoneal tuberculosis. The patient received fourfold antituberculous treatment with isonizid, rifampicin, pyrazinamide and ethambutol and was discharged from the hospital to continue treatment at home for 6 months. A regular follow--up was performed.

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Still's Disease – Unlikely Diagnosis on the Old Age

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Abstract

Adult Still's Disease is a rare systemic inflammatory disease. It is characteristically identified in the young adult, with few cases described in older patients. The aetiology and pathogenesis remain unknown. The absence of uniformly accepted diagnostic criteria may contribute to underdiagnosis.

We present a case of 74 year old patient with fever, polyarthralgia, odynophagia, salmon evanescent rash, elevated inflammatory parameters and serum ferritin. Excluded other diagnostics (infections, neoplasia and other immune diseases), the diagnosis of Adult Still's Disease was assumed. The clinical and analytical improvements with immunosuppressive therapy, and long follow-up without symptomatology, give strength to the diagnosis.

We present this case by the rarity of this pathology in this age and to emphasize the need to consider this entity whenever we are studying an fever of unknown origin with this clinical triad (daily spiking high fevers, evanescent rash, arthritis) and biologic triad (hyperferritinaemia, hyperleucocytosis with neutrophilia and abnormal liver function test).

Keywords: Adult-Onset Still's disease, fever of unknown origin, arthralgia, ferritin.

Introduction

Adult Still Disease (ASD) is a systemic inflammatory disease characterized by the clinical triad - daily febrile peaks, arthritis, and salmon colour evanescent rash; and by the biological triad - leukocytosis greater than 10 000 leukocytes with a predominance of neutrophils, hyperferritinemia (with decreased glycosylated ferritin) and changes in liver function tests[1]. Less common manifestations include odynophagia, serositis, myalgias, lymphadenopathy and hepatosplenomegaly.

Its etiology and pathogenesis remain unknown, but some factors have been described: genetic predisposition (association with HLA-B17, HLA-B18, HLA-B35 and HLA-DR2), innate immune system activation (elevated levels of IL1, IL6, IL18, TNFα and interferon γ) and environmental factors[2].

A variety of infectious triggers have been suggested, including viral agents (Rubella, Measles, Echovirus 7, Epstein-barr, Cytomegalovirus, Parvovirus B19, Adenovirus, Herpesvirus 6, Influenza, Parainfluenza, Hepatitis B virus and Hepatitis C virus) and intracellular bacteria (*Chlamydia Pneumoniae, Mycoplasma Pneumoniae, Borrelia burgdorferri, Brucella abortus, Yersinia enteroclitica*)[3-5].It has an equal distribution between genders, with a bimodal age peak: 16 to 25 years and 35 to 45 years. There are few reported cases in patients over 70 years old [6-7].

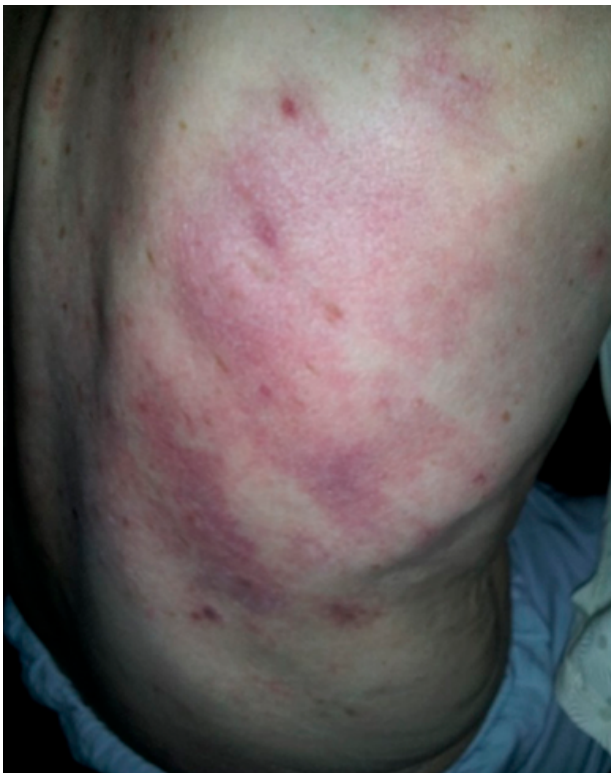
Clinical case

We report a case of 74 years old male, caucasoid. His medical problems included hypertension, benign prostatic hypertrophy, varicose ulcers of the lower limbs, and joint pain. He was treated with antihypertensive and occasionally anti-inflammatory drugs.

He was admitted for daily febrile peaks (39ºC), predominantly evening, with 3 weeks duration, odynophagia, myalgias, asthenia and weight loss (approximately 5 kilograms). He also had additive symmetric inflammatory polyarthralgia, with chronic and persistent evolution, involving dorsolumbar, large and small joints (shoulders, wrists, interphalangeal joints).

Patient examination revealed a decrease pulmonary sounds at left pulmonary base, with increase vocal vibrations in the same location; left axillary adenopathy with 1.5 centimeters of greater diameter (elastic consistency, not adhering to deep planes, painless). He presented an evanescent salmon colour macular rash affecting the trunk and limbs, slightly pruritic, with Koebner phenomenon (predominant involvement of the areas of greatest friction) that was intensified during the fever peaks and during the night (figure 1). He had no signs of arthritis or articular deformities on the skeletal muscle examination. There were no changes on the oropharynx observation.

Fig. 1. Salmon colour evanescent macular rash more evident in the posterior trunk (zone of greater friction)



The complementary study showed leukocytosis (34.1 x 10^3 / µL) with a predominance of polymorphonuclear cells (> 90%), elevated C-reactive protein (188 mg / L) and sedimentation rate (76 mm / 1h),

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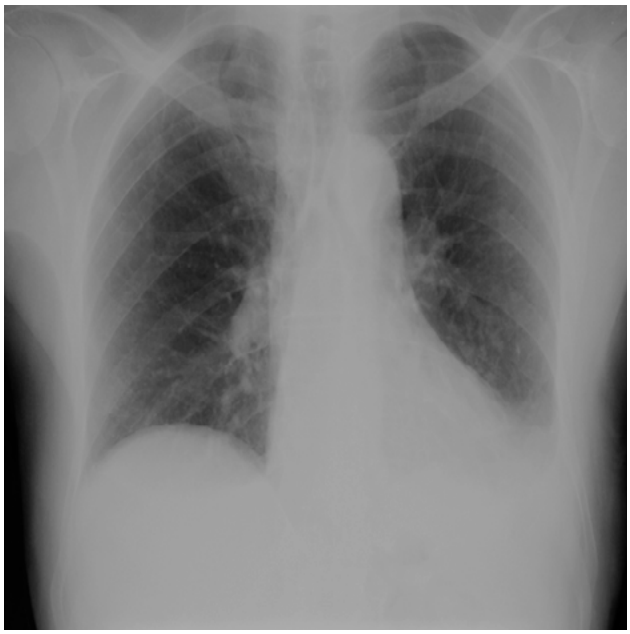
Fig. 2. Chest radiograph showing left pleural effusion



higher serum ferritin (> 4000 µg / L - five times higher than normal), increase in transaminases (146 U / L, 144 U / L), lactate dehydrogenase (437 U / L) and alkaline phosphatase (219 U / L); chest X-ray presented a small left pleural effusion (Figure 2).

Considering patient's age, a diagnostic investigation was initiated prioritizing the search for infectious and neoplastic diseases.

Infectious disease were excluded: negative blood cultures (aerobic bacteria, anaerobic bacteria and fungi), negative microbiological of pleural fluid and bronchoalveolar lavage (aerobic bacteria, anaerobic bacteria and mycobacteria), negative viral serologies and bacterial serologies (Human immunodeficiency virus 1 and 2, Hepatitis B, Hepatitis C, Syphilis, Epstein Barr virus, Cytomegalovirus, Parvovirus B19, *Borrelia burgdorferi* and *Coxiella burnetii*), Widal test, Weil-Felix, Wright and Paul-Bunnel negative, transthoracic and transoesophageal echocardiography without images suggestive of endocarditic vegetations.

Neoplastic disease was excluded: serum protein electrophoresis without peaks suggestive of monoclonality, peripheral blood immunophenotyping without suggestive alterations of lymphoproliferative disease, normal PSA, thoracoabdominal-pelvic computed tomography revealed the left pleural effusion, thyroid ultrasonography, endoscopic studies of digestive tract and bronchofibroscopy without changes, cytology of the pleural fluid and bronchoalveolar lavage negative for malignant cells, normal thyroid hormones.

Other rheumatologic diseases were excluded: increased immunoglobulin G, anti-nuclear antibodies (ANA's) 1:80, normal C3 and C4, negative ENA's, ANCA's, Anti-LKM, rheumatoid factor, anti-citrulline and HLA B27.

Despite patient advanced age, this clinical picture was very suggestive, and the hypothesis of ASD was considered (more than 5 criteria of Yamaguchi - 4 major criteria and 3 minor criteria; 4 major Fautrel criteria - figure 3).

We assumed the diagnosis of ASD and started immunosuppressive therapy with prednisolone, with rapid clinical and analytical response (normalization body temperature, improvement arthralgias and general symptoms, disappearance trunk rash and pleural effusion, normalization of leukocyte count, transaminases, ferritin and sedimentation rate) (figure 4).

Discussion

The absence of uniformly accepted diagnostic criteria contributes to the fact that it may be underdiagnosed. This pathology probably represents 5-10% of patients with fever of unknown origin [8].

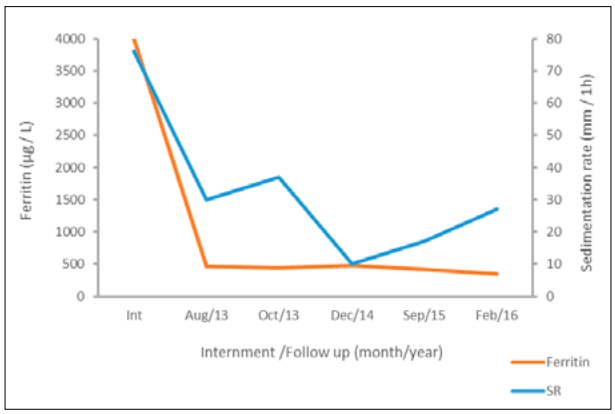
Since the main manifestations of ASD are not specific and shared by numerous pathologies, it is an exclusion diagnosis [9].

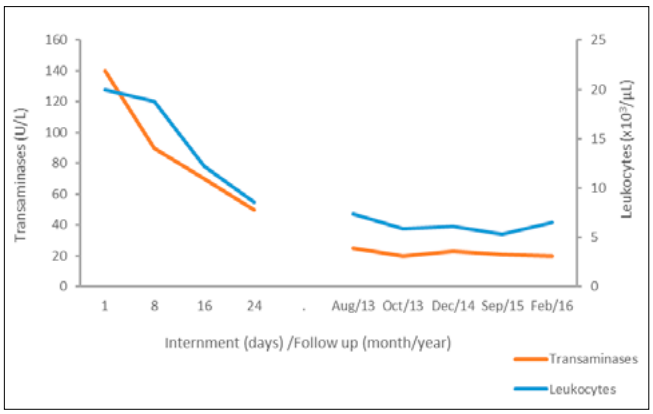
It is considered a diagnosis of the younger populations, and there are very few cases described in patients over 70 years old [10,11] Unless there is great suspicion, the diagnosis of ASD in older people is even more difficult. In older patients presenting with fever of unknown origin, the appearance of rash should make

Fig. 3. Criteria for diagnosis of ASD

	Yamaguchi Criteria (5 criteria - minimum of 2 major criteria and no exclusion criteria)	**Fautrel Criteria** (≥ 4 major criteria or 3 major criteria more 2 minor criteria)
Major Criteria	› Fever (≥ 39ºC), intermittent, lasting more than one week › Arthralgias or arthritis with > 2 weeks duration › Typical Rash › Leucocytosis (≥ 10000/mm^3), including ≥ 80% neutrophils.	› Febrile peaks ≥ 39ºC › Arthralgias › Transient erythematous Rash › Odynophagia › Polymorphonuclear ≥ 80% › Glycosylated Ferritin ≤20%
Minor Criteria	› Odynophagia › Recent lymphadenopathy › Hepatomegaly or splenomegaly › Changes in liver tests (aminotransferases and lactate dehydrogenase) › Negative Rheumatoid factor and ANA´s	› Maculopapular Rash › Leucocytosis ≥ 10000/mm^3
Exclusion Criteria	› Infections › Neoplasms › Others rheumatic diseases	

Fig. 4. Analytical evolution after immunosuppression initiation.





suspect ASD[12]. In this patient, the rash was a valuable diagnostic signal. After excluding other diseases (immunological, infectious, lymphoproliferative and neoplastic), reviewing the Yamaguchi and Fautrel diagnostic criteria (sensitivity and specificity above 92%), the diagnosis of ASD was assumed.

The prognosis of ASD depends on the clinical course of the disease: the disease can be self-limited / monophasic (with good prognosis), intermittent / polycyclic or chronic (with worse prognosis).

Due to rarity of this entity and the lack of clinical trials, there are no guidelines for the treatment of ASD. Available treatment modalities include non-steroids anti-inflammatory drugs (NSAIDs), corticosteroids, disease modifying drugs (DMDs) and biological agents. Approximately one quarter of patients respond to NSAIDs, but most patients require immunosuppression with systemic corticosteroids, DMDs, such as methotrexate, and biological agents [10,13]. In patients with resistance or dependence on corticosteroids, the choice is methotrexate.

Although there are few reported cases of methotrexate use in older patients, the treatment of these patients may be useful in disease control, avoiding relapses, and a corticosteroid sparing agent, minimizing it's adverse effects[11].

This patient appears to have a disease with a good prognosis (monophasic form), and presented clinical and analytical improvement with immunosuppressive therapy, remaining without symptoms with minimal corticosteroid doses (≤ 7,5 mg) (follow-up more than 5 years).

This case reflects a rare entity in this age, but should not be overlooked in older patients with fever of unknown origin.

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Herpes simplex type-1 pneumonia in an immunocompetent patient: a case report

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Abstract

Herpes simplex virus type-1 pneumonia is unusual and rarely described without any degree of immunosuppression. We share a case of herpes simplex virus type-1 pneumonia in an immunocompetent patient, not only by its rarity, but to call attention to the importance of thinking about this entity when respiratory symptoms persist despite various antibiotic schemes, especially in the presence of ground glass or multifocal pulmonary infiltrates, regardless of patients' immune status.

Keywords: Herpes Simplex, Viral Pneumonia, Immunity.

Introduction

Herpes simplex virus type-1 (HSV-1) infection is extremely prevalent, as more than 90% of the world's population has positive serological tests by the fourth decade of life.[1] Virus transmission occurs from person-to-person mainly by contact with infected oropharyngeal lesions, with an incubation period ranging from 1 to 26 days (median 6 to 8 days).[1, 2] Gingivostomatitis and pharyngitis are the most common manifestations of primary infection and labial herpes the most frequent form of reactivation. HSV-1 typically causes recurrent infections and similar to what happens with herpes zoster, the reactivation could occur through the lymph nodes where the virus is latent, in the absence of oropharyngeal lesions.[3]

HSV-1 pneumonia is unusual, but may arise in primary or recurrent infection. There are case reports in burned, transplanted and cancer patients, people under corticosteroids[4], mechanically ventilated, malnourished or infected with human immunodeficiency virus (HIV), but HSV-1 pneumonia is rarely described without any degree of immunosuppression.[1, 5]

Clinical case

We present a 50-year-old Caucasian non smoker man, with no relevant background. Referred to the Emergency Room with asthenia, anorexia, fever (40ºC), myalgia and productive cough with a week of evolution, already treated with levofloxacin for 3 days without improvement. He had visited a farm about 5 days before the onset of symptoms, but denied consumption of fresh farm cheese or well water. On the physical examination he had rhonchi scattered on auscultation, analytically a reactive C protein of 242,6 mg/L without leukocytosis, with normal renal function. Chest X-ray showed diffuse pulmonary infiltrates (Fig. 1) with an arterial blood gas showing hypoxemia (pO2 55 mmHg). Assuming community-acquired pneumonia, he started empirically ceftriaxone and azithromycin but had an unfavorable evolution, maintaining persistent fever and hypoxemia on the fourth day of treatment. We were unable to identify an agent in the screening initially performed (blood cultures, urinary antigen test for *Streptococcus pneumoniae* and *Legionella pneumophila*, nasopharyngeal aspirate for influenza virus, bacteriological and mycobacterial sputum culture). Because of the context for zoonotic disease and poor response to the previous antibiotherapy, treatment was changed to doxycycline but with no further response. Chest tomography showed multiple interstitial opacities scattered with ground glass areas, most exuberant in the lung bases (Fig. 2A and B), opening a range of differential diagnoses initially not placed. The immune study was nonspecific, showing only positive antinuclear antibodies (1/160 mottled pattern) with normal immunoglobulins and complement, without anti-neutrophil cytoplasmic antibodies or rheumatoid factor. The serological tests for HIV, *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, *Chlamydophila psittaci* and *Coxiella burnetii*, were negative. Chasing the diagnosis, we performed a bronchofibroscopy that showed whitish micronodules scattered through the trachea and bronchial tree, with mucosal hyperemia; bronchial and bronchoalveolar lavage were obtained and sent to microbiological and anatomopathological analysis. Cytology was negative for malignant cells. On the eighth day of hospitalization, HSV-1 was identified by polymerase chain reaction (PCR) in bronchial lavage, with negative PCR for herpes simplex type-2 and cytomegalovirus. With this information we suspended antibiotics and began intravenous acyclovir monotherapy, 10 mg/kg three times a day for 10 days, with rapid clinical and analytical improvement. Reviewing the case, the patient didn´t have oral lesions and he was never under corticosteroids. Cytology was revised with the knowledge of a positive PCR for HSV-1, but no viral inclusions were found.

Discussion

The diagnosis of HSV-1 infection was classically based on the presence of cytological abnormalities, particularly the presence of cytoplasmic inclusions and homogenization of nuclear chromatin[2, 6], but to observe these abnormalities the sample must be immediately transported to the laboratory and stored at 4°C under the penalty of not being able to observe the viral cytopathic effect due to technical reasons[1], possibly what happened in our case report. Molecular biology techniques, such as PCR, have gained increasing importance as they overcome cytology limitations. Despite its undeniable value, the relevance of a positive PCR for HSV-1 in critically ill patients, especially those with acute respiratory distress under mechanical ventilation, is unclear.[7, 8]

In the context of pneumonia refractory to standard antimicrobial therapy, the possibility of HSV pneumonia must be pursued[9],

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Fig. 1. Chest X-ray showing multifocal consolidations.

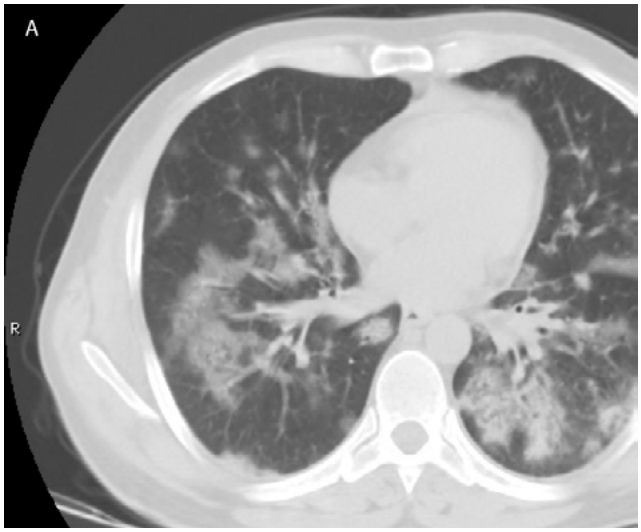
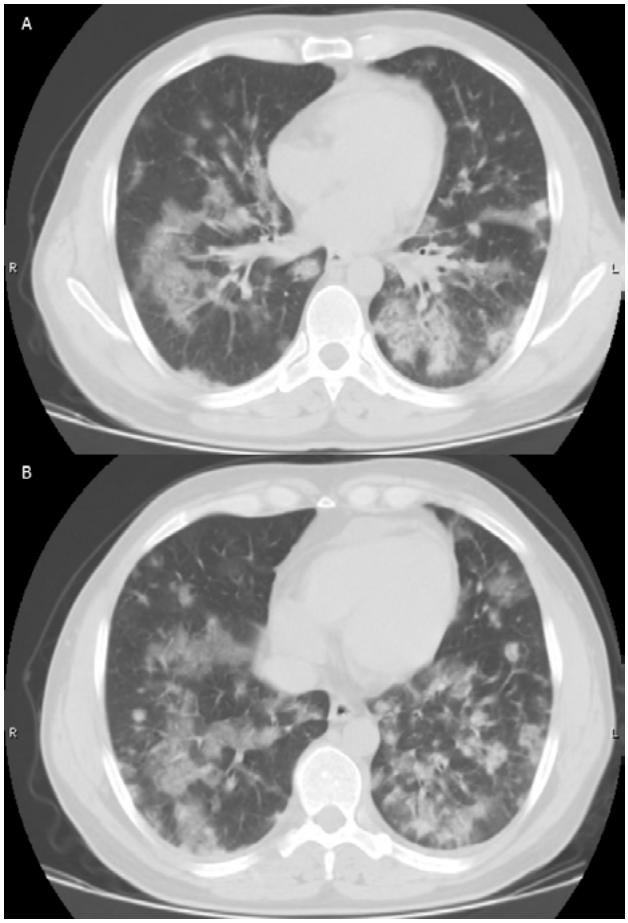


Fig. 2A and B. Chest tomography showing multifocal segmental and subsegmental consolidations with ground glass opacities and air bronchogram.



especially in the presence of ground glass or multifocal infiltrates on high-resolution tomography, without any specific difference between immunocompetent and immunocompromised patients.[3, 5]

As in other viral infections, spontaneous resolution may occur[3], but it is consensual to treat patients who present with severe infection whenever HSV-1 is isolated from the lower respiratory tract.[6] Treatment includes the use of intravenous acyclovir 5-10 mg/kg three times a day, and alternatively oral acyclovir 400-800 mg/day or valacyclovir 1000 mg three times a day.

The prognosis is related to patients' immune status, being better in the immunocompetent ones.[2] Our patient remains without any sign of immunodeficiency, cancer or autoimmune disease, after 4 years of follow up.

With this case report, we aim to call attention to the importance of thinking about HSV-1 pneumonia when respiratory symptoms persist despite various antibiotic schemes, especially in the presence of ground glass or multifocal pulmonary infiltrates whose origin is unclear, regardless of patients' immune status[2, 10]. It is an unusual diagnosis with varied severity, possibly underdiagnosed.

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Pulmonary MALT lymphoma – when an opacity persists too long: a case report and literature review

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Abstract

Mucosa-associated lymphoid tissue (MALT) lymphoma is an extranodal lymphoma arising in a number of epithelial tissues, including stomach, salivary gland and the lung. Pulmonary MALT lymphoma originates in the parenchyma and/or bronchi and is a rare disease, representing 0.5-1% of primary pulmonary malignancies. Although the time to diagnosis can be delayed because most patients are asymptomatic and investigations are usually driven by the accidental finding of abnormal lung imaging studies, it has an indolent course and a good prognosis. Here we report the case of an asymptomatic female patient with a nodular lung opacity that was found to persist five months after an episode of community-acquired pneumonia. A final diagnosis of pulmonary MALT lymphoma was obtained after an extensive diagnostic work-up.

Keywords: Lymphoma, B-Cell, Marginal Zone, Lung Neoplasms

Introduction

Pulmonary MALT (pMALT) lymphoma is a clonal lymphoproliferative disorder that arises from B lymphocytes in the epithelial lung tissue. By definition, it affects one or both lungs, without extrapulmonary involvement at diagnosis or during the subsequent three months. It usually has an indolent course and carries a good prognosis. Since it's a silent disease in over 50% of patients, the mean time to diagnosis is around 9 months[1]. Tissue biopsy is the gold standard for diagnosis[1]. There is no clear consensus on treatment: localized disease can be treated with radiotherapy, surgery or observation while diffuse lymphomas are almost always managed with chemotherapy, either with a single or multiple agents[1,2].

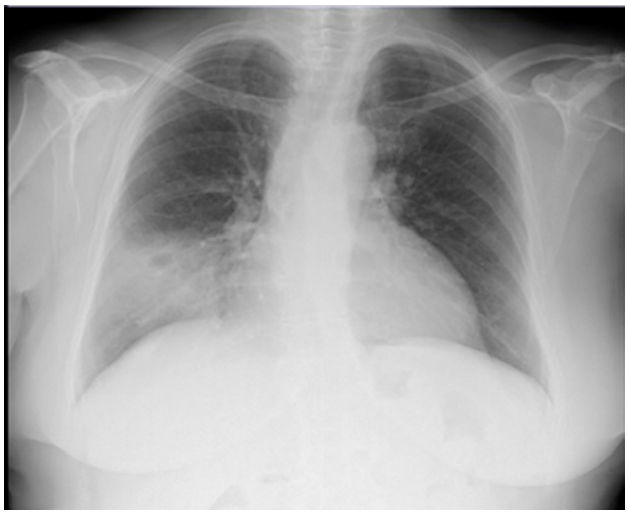
Case report

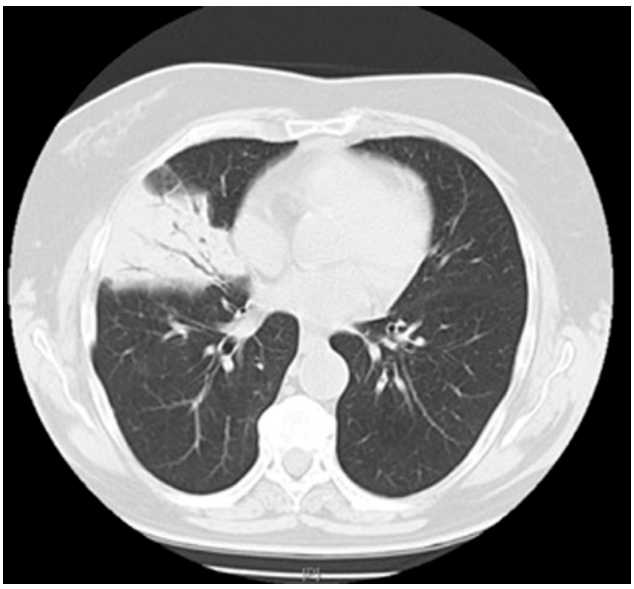
a 72-year old woman was admitted in another hospital emergency with a clinical presentation of a flu-like symptoms that started in the previous 3 days, accompanied by fever, cough and sputum. No changes on clinical examination were found; pulmonary auscultation revealed a vesicular breath sounds on both lungs. Laboratory tests revealed leukocytosis with neutrophilia, mild normocytic normochromic anemia and elevated reactive C protein. Chest x-ray showed a consolidation of the middle lobe and she was then hospitalized with the diagnosis of a community-acquired middle lobe pneumonia treated to clinical and laboratory resolution with amoxicillin-clavulanic acid and azithromycin. Her chest x-ray of discharge still showed the alveolar opacity in the middle lobe.

During follow-up, she had been always asymptomatic. However, her repeated chest x-rays remained showing the alveolar opacity in the middle lobe (Figure 1). A thorax computed tomography (CT), five months after discharge, confirmed the consolidation of the middle lobe (Figure 2) and also revealed another homolateral nodular lesion with 11,5mm and multiple hepatic hypodense nodules. At this point she was referred to our pulmonary outpatient clinic where we started her diagnostic work-up.

The patient was non smoker and denied previous history of any respiratory illness. She had a history of hypertension, type 2 diabetes, dyslipidemia and vertigo syndrome. On admission, she was asymptomatic, revealed slightly pale mucous membranes and faint symmetrical lower limb edema; otherwise normal. Laboratory tests showed mild normochromic normocytic anemia and mild leukocytosis. HIV (1 and 2) and hepatitis serologies were negative. Auto-immune serum screening was negative.

Fig. 1. A thorax computed tomography (CT), five months after discharge, confirmed the consolidation of the middle lobe (Figure 2) and also revealed another homolateral nodular lesion with 11,5mm and multiple hepatic hypodense nodules. At this point she was referred to our pulmonary outpatient clinic where we started her diagnostic work-up.





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An abdominal ultrasound favored the cystic nature of the hepatic lesions. Fiberoptic bronchoscopy revealed a diffuse mildly friable and inflamed mucosa with a slight enlargement of the right basal pyramid spurs. Bronchoalveolar lavage (BAL) was negative for malignant cells and microbiology analysis but showed marked lymphocytosis (91%) with a population of monoclonal B lymphocytes.

The patient then underwent a CT-guided needle biopsy of the middle lobe lesion that suggested MALT hyperplasia with low proliferative index (Ki67<5%). A surgical lung biopsy of both the middle lobe mass and right inferior lobe nodule confirmed the diagnosis of pMALT lymphoma, revealing enlargement of the interalveolar septa due to lymphoid cell proliferation, with positivity for CD20 and Bcl-2 and negativity for CD10, Bcl-6, cyclinD1 and MUM1 at immunohistochemical staining. Bone marrow biopsy presented no evidence of neoplastic involvement.

Subsequently, the patient was transferred to the Hematology Department where she performed a contrast enhanced CT scan of the abdomen and pelvis for staging that failed to reveal further lesions.

The diagnosis of pMALT lymphoma (Ann-Arbor satge IE) was established and the patient was started on chemotherapy with R-CVP (R – rituximab; CVP - cyclophosphamide, vincristine and prednisone).

Discussion

Pulmonary MALT lymphoma is as a subtype of non-Hodgkin lymphomas that is characterized by a low-grade extranodal marginal zone B-cell proliferation, arising in a number of epithelial tissues, including stomach, salivary gland, lung, small bowel and thyroid. It can also be described as the most common primary pulmonary lymphoma (up to 80%), nevertheless a rare disease, representing 0.5-1% of primary pulmonary malignancies[1,3].

Increasing evidence suggests that this lymphoma is related to chronic immune stimulation due to bacterial, viral or autoimmune stimuli. The most known studied association is between *Helicobacter pylory* infection and gastric MALT lymphoma, not only in the pathogenesis but also in the complete remission when the bacteria are eradicated by antibiotics[4]. However a causative agent or autoimmune process associated to pMALT hasn't been found yet. In our case we can postulate that an infection could have been the trigger since the patient was initially treated to community acquired pneumonia. Unfortunately no microbiological study was performed.

The median age at diagnosis is 50-60 years old and there is no gender predominance[3,5]. Active or former tobacco use is not higher than in general population[1]. Clinically, half of the cases are asymptomatic and features of autoimmune diseases is present in 16% of the patients at diagnosis[1,5].

Patients with pMALT lymphoma typically present as a chronic alveolar localized opacity less than 5 cm in diameter and associated with air bronchograms in nearly 50% of the cases, such as the case we present (Figure 1)[1,3,6]. The commonest CT findings are single or multiple nodules or areas of consolidation (>70%), multiple bilateral lesions (>70%) that tend to be peribronchovascular with intact bronchial lumen (Figure 4)[6]. Less commonly, hilar and/or mediastinal lymphadenopathy (30%), bronchiectasis and bronchiolitis, diffuse interstitial lung disease and pleural effusion are found[1,6]. The time between clinical or radiological findings and diagnosis varies from 15 days to 8 years, mean 9 months, which was the exact time from the pneumonia episode to the surgical biopsy result[1].

Tissue biopsy is the gold standard for diagnosis[1]. It may be obtained *via* minimally invasive procedures, including bronchoscopy or CT-guided needle biopsy, but surgical lung biopsy is sometimes necessary to clarify the diagnosis. In our patient surgical lung biopsy due to its wider representation was fundamental to clarify the diagnosis. Bronchoalveolar lavage (BAL) may aid in the differential diagnosis of chronic alveolar opacitites by revealing the absence of tumor epithelial cells and usually the presence of lymphocytic alveolitis preferably with monoclonal populations (lymphocytes > 20% of total cells)[3,7]. In our case we highlight the value of this procedure that was actually the first exam leading to the suspicion of this type of malignancy.

Initial staging includes CT scanning of the chest, abdomen, and pelvis, with contrast injection, for the Ann-Arbor staging system, as was done in our patient. Bone marrow biopsy may show involvement in 13–30% of cases[1,5].

There are many therapeutic options but no standard guidelines, owing to the rarity of this lymphoma. For localized disease, most frequently, radiotherapy is the first-line treatment[2,9]. Other options include surgical resection or "watchfull waiting" strategies for asymptomatic patients without cytopenias. In the specific case of localized pulmonary lymphoma, chemotherapy can also be considered, as happened to be the case of our patient, due to concurrent anemia[1,2,9]. In diffuse disease, chemotherapy is preferred, typically with oral alkylating agents (cyclophosphamide or chlorambucil) or purine analogues (fludarabine, 2CdA), or combination chemoterapy (CHOP – cyclophosphamide, adriamycin, vincristine and prednisone; CVP). Rituximab, anti-CD20 monoclonal antibody, has shown efficacy as an additional option to chemotherapy schemes[2].

Prognosis is good, with 5-year overall survival of over 80% and a median survival of over 10 years[1,9]. Follow-up is required for a long time as almost 50% show recurrence of the disease and histological transformation to a more aggressive lymphoma can occur in about 10% of cases[2,9].

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Acrocyanosis

Sixty-year-old woman, with a history of gastric neoplasia in remission 5 years ago, and since then have a totally iimplantable central venous catheter (TICVC) in the right subclavian vein. No other relevant history and no known allergies.

She went to the emergency department with complaints of asthenia, edema of the face and neck, dysphagia and dysphonia with two months of evolution, and edema of the right upper limb since three days.

On physical examination there was edema of the face, cervical region and the thorax that extending to the fourth rib and right upper limb, acrocyanosis; cardiopulmonary auscultation was normal; humeral, radial and ulnar pulses were present and bilaterally symmetrical.

Analytically, D-dimers of 1859 ng/dl and C-reactive protein of 32mg/L. Computed angiotomography documented: "central venous catheter terminating in the superior vena cava (SVC), thrombotic phenomena of SVC and the right jugular vein with permeability from azygous vein. Accentuated collateral circulation in the left hemithorax with densification of soft tissues in the cervical region and right anterolateral thoracic wall ".

She started anticoagulation with Low Molecular Weight Heparin, followed by warfarin; she was asymptomatic at 10 days of treatment, at which time the TICVC was removed, without intercurrences. The patient was followed up in consultation; anticoagulation was mantained for one year, with resolution of the condition.

TICVCs are devices that allow simple, safe and non-limiting vascular access in the daily activities of cancer patients.[1] Central venous thrombosis is a major late complication, whose incidence is underestimated.[2]

In the case described, the typical clinic and the imaging tests determined the diagnosis – superior vena cava syndrome of benign cause in relation to TICVC.

This syndrome is characterized by a set of signs and symptoms resulting from venous stasis caused by obstruction of the superior vena cava, either by thrombosis, extrinsic compression or direct vein invasion. The most common causes are malignant (60 to 85% of cases); the iatrogenic etiology is rare.[1,2,3] Diagnosis is usually achieved through the clinic and confirmed by imaging tests. The treatment depends on the etiology, but in all the situations the symptomatic treatment is recommended: head elevation, rest, control of the volume administered, oxygen supplementation, diuretic, corticoid if there are edema laryngeal or cerebral edema. Other treatments such as anticoagulation, surgery, endoluminal treatment, radiotherapy and chemotherapy may be considered.[2,4]

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Fig. 1. Edema of the face, cervical region and thorax, acrocyanosis.

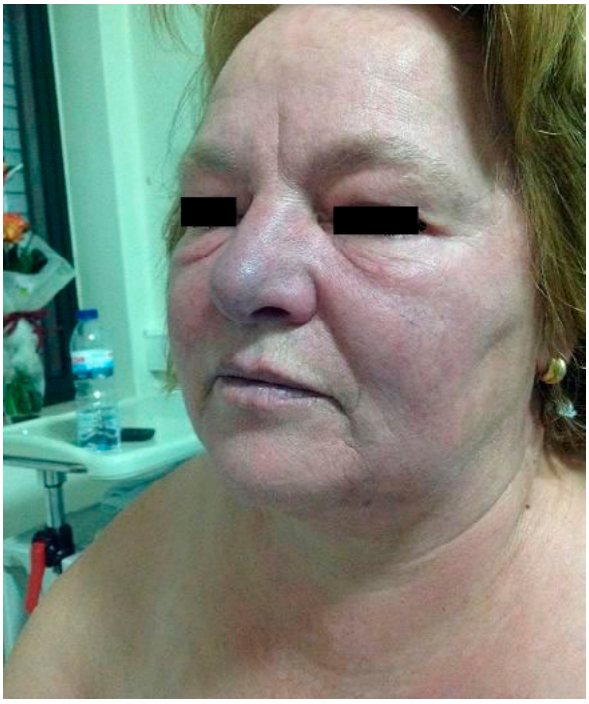
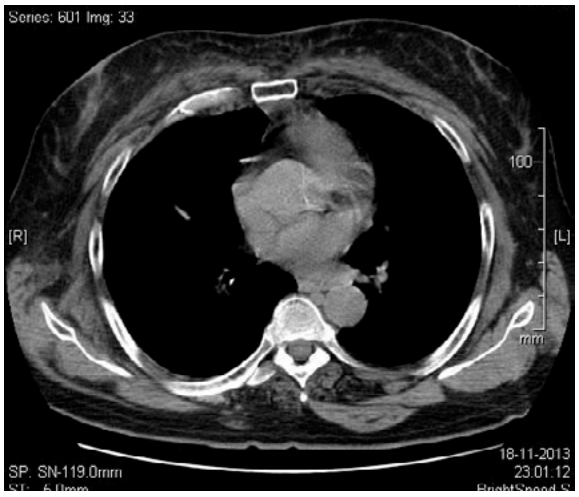


Fig. 2. TC angiograph: thrombotic phenomena of SVC and the right jugular vein with permeability from azygous vein.



Diagnóstico

Latrogenic acrocyanosis

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Paget-Schroetter Syndrome

A 34-year-old man, kickboxing, presented on emergency room with 24 hours of sudden onset right upper extremities (RUE) pain, swelling and heat after vigorous exercise with that member, without trauma. He was hemodynamically stable, with RUE edema. Color doppler showed right axillary, subclavian and jugular veins thrombosis. Computed tomographic pulmonary angiography confirmed the findings, associated to acute bilateral pulmonary embolism at the level of the segmental branches (Figure 1 and 2). Excluded secondary causes of venous thrombosis of the upper limb. As described on literature on that season, he completed 6 months with anticoagulation with symptoms resolution.

Paget-Schroetter Syndrome is an effort thrombosis more common in young athletes without significant comorbidities[1]. Symptom onset is usually acute or sub-acute and not infrequently can be nonspecific. Color doppler is the least invasive and least costly exam, with adequate sensitivity and specificity for diagnosis. Treatment include anticoagulation with or without revascularization therapy, such as thrombolysis, endovascular or surgical therapy[2,3]. Complications include pulmonary embolism, present at 3% of effort thrombosis[4]. However, irrespective of the relative risk, the risk of pulmonary embolism with effort thrombosis is real and significant[5]. It's a clinical condition with impact on the patient's quality of life.

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Diagnóstico

Paget Von Schrotter syndrome and Pulmonary thromboembolism

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Fig. 1. Computed tomographic pulmonary angiography showed showed right axillary, subclavian and jugular veins thrombosis (red arrows).

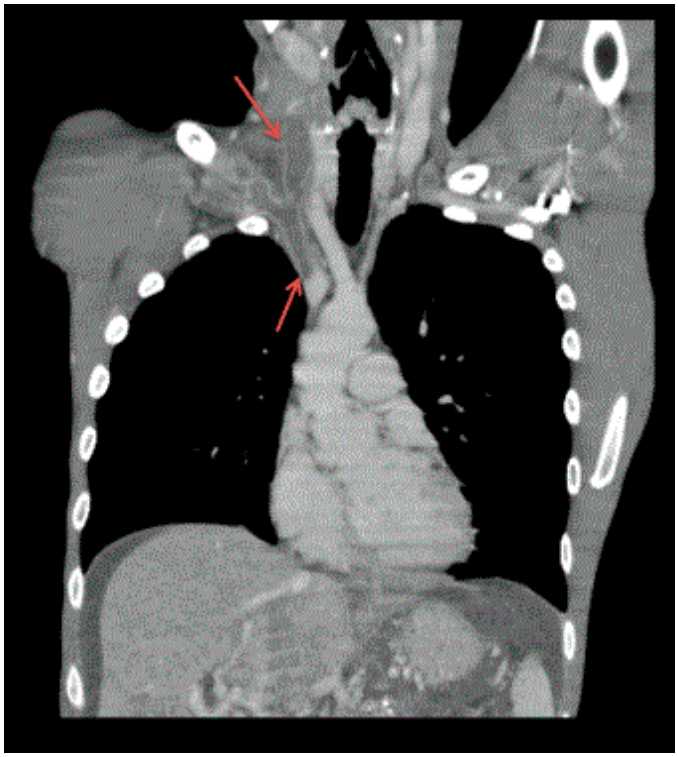
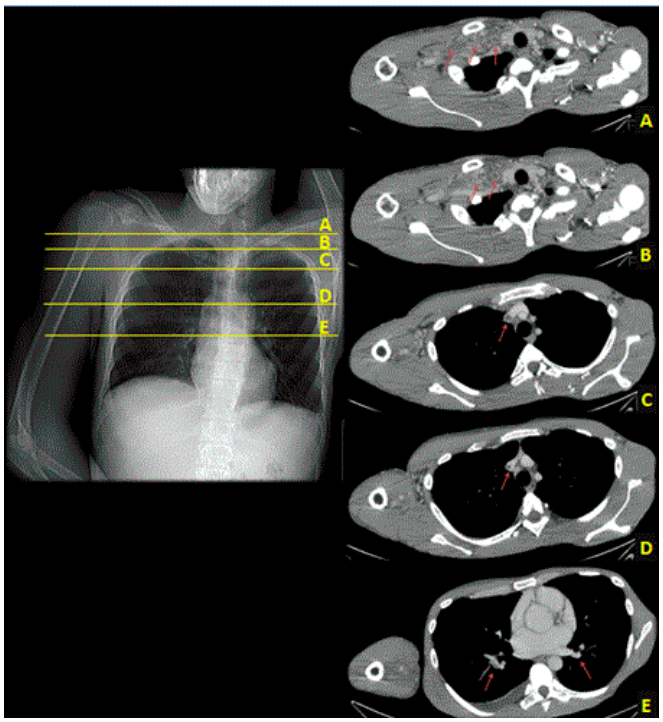


Fig. 2. Computed tomographic pulmonary angiography with different views. We can see right axillary, subclavian and jugular veins thrombosis (A, B, C), started on origin of subclavian vein (d) and pulmonary embolism (E).



Un kilo y medio más...

A kilo and a half more…

Se presenta el caso de una mujer de 25 años, con obesidad grado II, sin antecedentes patológicos relevantes y sin historia de traumatismo importante reciente. La paciente recurrió al Servicio de Urgencias por cuadro de dolor lumbar izquierdo con irradiación abdominal de moderada intensidad que empeora con los movimientos y a la palpación, con un año de evolución pero con períodos asintomáticos, y con empeoramiento significativo en los últimos dos días.

En la exploración física, a la palpación abdominal, destacó dolor leve en el flanco superior izquierdo, puño percusión lumbar izquierda negativa. En la analítica, no había alteraciones en el hemograma, bioquímica y coagulación.

La Ecografía Abdominal y posteriormente la Resonancia magnética mostraron: voluminosa formación quística intraesplénica que comprimía el cuerpo y la cola del páncreas y desviaba el estómago; extensión cráneo-caudal 15cm, laterolateral 11cm y antero-posterior 12cm. La paciente se sometió a una esplenectomía. La pieza obtenida pesaba 1498g, y estaba constituida por tejido esplénico sin revestimiento epitelial reconocible, y una cavidad con material líquido, con pared con áreas calcificadas - pseudoquiste esplénico.

El quiste esplénico es una entidad rara, con menos de 1000 casos descritos. Se clasifican, según Martin, en tipo I (primarios o verdaderos) y tipo II (secundarios o pseudoquistes). Los quistes tipo I poseen cápsula epitelial, y pueden ser de naturaleza parasitaria o no parasitaria, pudiendo ser aún de causa congénita, vascular o neoplásica. Los quistes tipo II corresponden al 75% de los quistes, no tienen revestimiento epitelial y son secundarios a trauma, infección o infarto; la mayoría son asintomáticos. Microscópicamente, los pseudoquistes se componen de tejido fibroso denso, a menudo calcificado, sin epitelio de revestimiento; pueden contener una mezcla de sangre y restos necróticos en su interior.

Las pruebas de imagen son útiles en la identificación del quiste y posibles complicaciones, pero no ayudan en la distinción del tipo de quiste, teniendo la anatomía patológica un papel fundamental en el diagnóstico final.

El tratamiento quirúrgico está recomendado en os quistes esplénicos con un diámetro superior a 4-5 cm, debido al riesgo de complicaciones. La laparotomía con esplenectomía ha sido el método de elección preferencial para el tratamiento.

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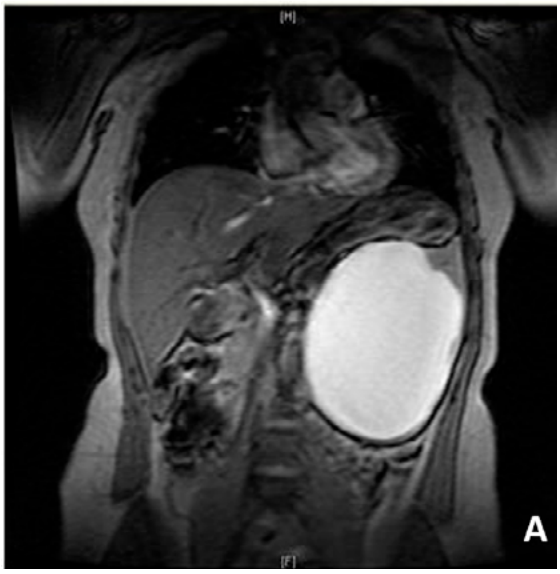
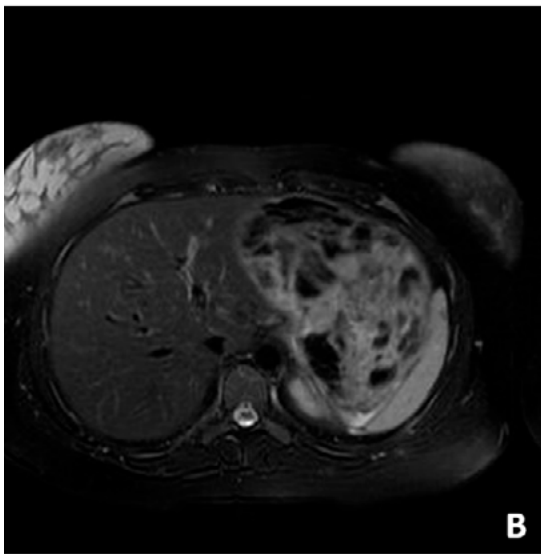
Imagen 1. Pseudoquiste esplénico con extensión cráneo-caudal 15cm, laterolateral 11cm y antero-posterior 12cm. A - corte longitudinal. B - corte axial.





Diagnóstico

Quiste esplénico

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PULSE PARA VOLVER AL ÍNDICE

Lo que puede esconder una ciatalgia

What a ciatalgia can hide

Varón de 60 años sin antecedentes de interés, que acude a urgencias en repetidas ocasiones por ciatalgia unilateral de 3 meses de evolución, que no cede a pesar de tratamiento convencional. Por dicho motivo se solicita TC de pelvis donde se evidencian una masa ligada al nervio ciático (imagen 1 y 2). En el estudio de extensión se solicita TC torácico y abdominal, donde se evidencian lesiones ocupantes de espacio en hígado y pulmón, lesiones osteoclásticas y múltiples adenopatías retroperitoneales y mediastínicas. Antes estos hallazgos se realizó una biopsia de la masa glútea, obteniendo el siguiente estudio anatomopatológico: áreas densamente celulares y áreas de matriz hialina, núcleos fusiformes y ovoides con citoplasmas eosinófilos, se tiñe con S100, enolasa neuronal y CD 57; es negativo para citoqueratinas, melanocitos, linfocitos y estirpe epitelial; todo esto compatible con tumor maligno de vaina de nervio periférico (Nervio ciático). El paciente presentaba dolor incontrolado por lo que se inició radioterapia antiálgica sobre la lesión y posteriormente tratamento quimioterápico, con mala evolución siendo éxitus.

El tumor maligno de vaina de nervio periférico es raro, solo representa un 5-10% de los sarcomas de tejidos blandos. Se puede asociar a neurofibromatosis pero hasta el 50% son esporádicos. El rango de edad suele ser de los 5 a 75 años, siendo la 5º y 6º década las de mayor prevalencia. El género más afectado suele ser el masculino. La clínica predominante la local (tumefacción) y la irritación del nervio (dolor neuropático). El diagnóstico se establece a través del estudio anatomopatológico, en el que se observa tinción frente a S100 y p53. El tratamiento suele ser cirugía radical y radioterapia, pero el pronóstico es infausto hasta para los que se extirpan con intención curativa.

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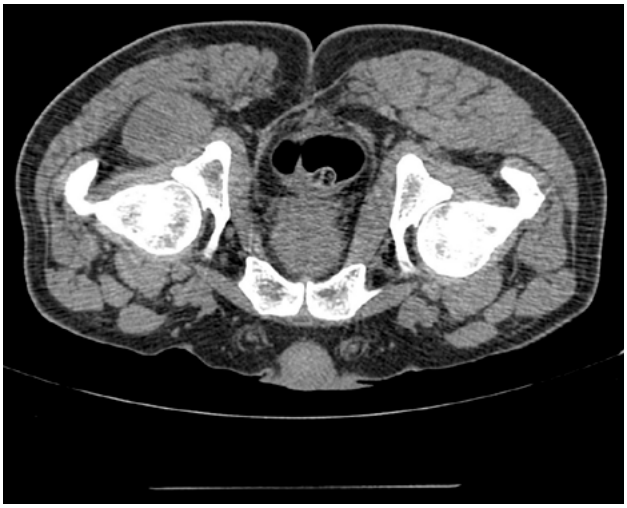
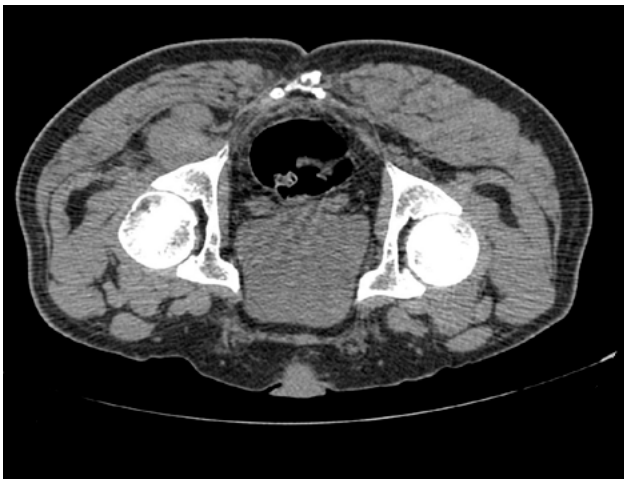
Imágenes 1 y 2. Masa ligada al nervio ciático.





Diagnóstico

Tumor maligno de vaina de nervio periférico (Nervio ciático)

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A rare cause of liver abscess in a Western Country

Klebsiella pneumoniae is a common cause of pyogenic liver abscess in patients with diabetes mellitus, preexisting hepatobiliary disease and those from Southeast Asia[1], where it accounts for 50% to 73% of cases[2]. But, in the last decades, there has been an increasing number of reports in Western countries[3].

A 61-year-old portuguese male, without significant past medical history, was admitted with right upper quadrant abdominal pain and fever. He denied any recent foreign travel. Laboratory analysis showed 6900 leucocytes, C-reactive protein 287.5 mg/L, total billirubin 1.1 mg/dL, AST 116 UI/L, ALT 88 UI/L, alkaline phosphatase 180 UI/L. Abdominal Computed Tomography (CT) scan revealed a 3.4x3.3cm complex mass in the IV hepatic segment with multiple septations. Blood cultures grew *Klebsiella pneumoniae* and, according to the antibiogram, he was placed on ceftriaxone for 21 days. The abscess was drained by interventional radiology and gram stain and cultures of pus were positive for *Klebsiella pneumoniae*. His clinical condition improved and a follow-up CT, after antibiotic therapy, showed complete resolution of liver abscess.

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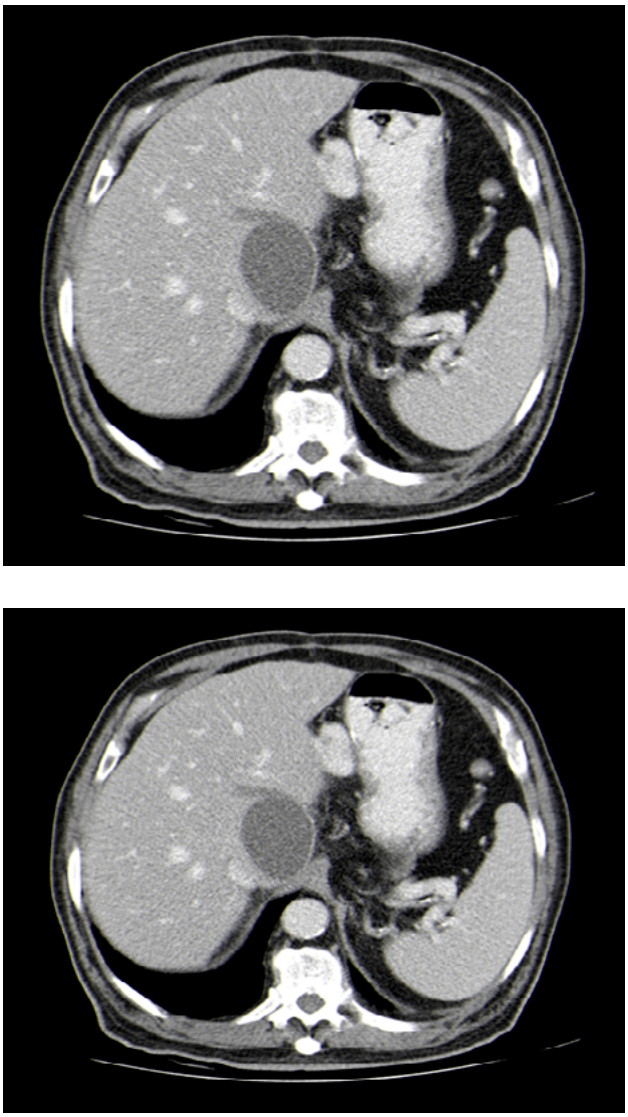
Fig. 1. Abdominal Computed Tomography with voluminous mass compatible with hepatic abscess



Diagnosis

Pyogenic liver abscess caused by Klebsiella pneumoniae

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Pruritic cutaneous lesions on the chest

We present the case of an 82 years old woman with history of Diabetes mellitus type 2 and arterial hypertension, chronically medicated with insulin glargine 20 units/day and amlodipine/valsartan 5/160 mg per day. The patient was admitted to the emergency department due to dyspnea and chest pain with 1 day evolution, reporting also a 3-months history of asthenia and pruritic cutaneous lesions on the chest. On admission the patient was pale, polypneic and tachycardic with erythematous cutaneous lesions localized on the anterior and posterior thoracic region. Blood examination showed microcytic anaemia (Haemoglobin 9.1 g/dl) and elevation of D-dimer (D-dimer 4599 ng/ml). Arterial blood gas revealed hypoxemia and hypocapnia. Chest angiotomography exhibited pulmonary thromboembolism and enoxaparin was instituted. Thoraco-abdomino-pelvic computed tomography (CT) exhibited a gastric neoplasm with invasion of hepatic left lobe and local adenomegalies. The gastric biopsy confirmed the diagnosis of gastric adenocarcinoma. Skin lesions biopsy revealed histological findings compatible with bullous pemphigoid (BP) and treatment with prednisolone 40 mg/day was started. In cancer multidisciplinary consultation it was decided to initiate palliative chemotherapy. The patient died 6 months after the diagnosis.

BP is the most common autoimmune blistering disease, occurs mostly in elderly patients and it is characterized by autoantibodies deposition at the epithelial basement membrane zone.[1-4] Classically presents with an intensely pruritic eruption with tense subepithelial blisters filled with a serous or haemorrhagic content.[1-4] Bullous phase may be preceded by a prodromal phase lasting weeks to months and characterized by pruritic eczematous, papular or urticarial-like lesions. [1-4] In the presented case, the patient was in the nonbullous phase which made the diagnosis difficult.[4] The association between malignant neoplasms and BP has been emphasized, but it is unclear whether coexisting malignancy and BP patients are pathogenically connected or whether the association is merely linked to aging.[4,5] Corticosteroids are the first line of treatment, but other immunomodulatory therapies are often necessary.[2] This case alerts to a less obvious presentation of the PB and its association with a neoplasm.

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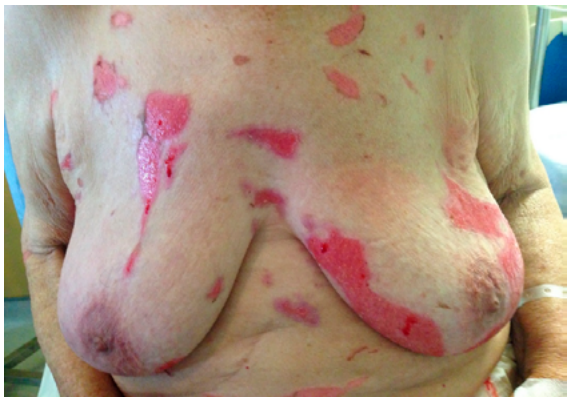
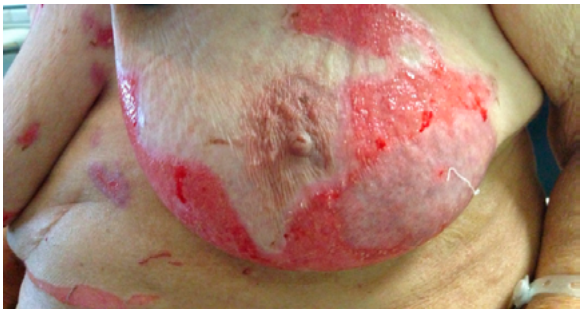
Fig. 1-2. Pruritic cutaneous lesions on the chest





Diagnosis

Cutaneous Manifestations of Gastric Neoplasm

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